د بیړنیو پیښو د درملنې لارښود

(په انګلیسی ژبه)

پوهنوال ډاکتر ایمل شیرزی

AFGHANIC

ننګرهار طب پوهنځی

Pashto PDF
2013

Funded by
Kinderhilfe-Afghanistan

Guideline For Treatment of Emergency Cases

(In English)

Prof. Dr. Aimal Sherzay

Download: www.ecampus-afghanistan.org

ننگرهار طب پوهنځی

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Nangarhar Medical Faculty

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With CD

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بسم الله الرحمن الرحيم

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د بیړنیو پیښو د درملنې لارښود

(په انګلیسی ژبه)

پوهنوال ډاکتر ایمل شیرزی

د لوړو زده کړو وزارت

پیغام

د بشر د تاریخ په مختلفو دورو کې کتاب د علم او پوهې په لاسته راوړلو کې ډیر مهم رول لوبولی دی او د درسي نصاب اساسي برخه جوړوي چې د زده کړې د کیفیت په لوړولو کې مهم ارزښت لري. له همدې امله د نړیوالو پیژندل شویو ستندردونو، معیارونو او د ټولنې د اړتیاوو په نظر کې نیولو سره باید نوي درسي مواد او کتابونه د محصلینو لپاره برابر او چاپ شي.

د لوړو زده کړو د مؤسسو د ښاغلو استادانو څخه د زړه له کومې مننه کوم چې ډېر زیار یې ایستلی او د کلونو په اوږدو کې یې په خپلو اړوندو څانګو کې درسی کتابونه تألیف او ژباړلي دي. له نورو ښاغلو استادانو او پوهانو څخه هم په درنښت غوښتنه کوم تر څو په خپلو اړوندو برخو کې نوي درسي کتابونه او نور درسي مواد برابر کړی څو تر چاپ وروسته د ګرانو محصلینو په واک کې ورکړل شي.

د لوړو زده کړو وزارت دا خپله دنده بولي چې د ګرانو محصلینو د علمي سطحې د لوړولو لپاره معیاري او نوي درسی مواد برابر کړي.

په پای کی د افغان ماشومانو لپاره د جرمنی کمیټی او ټولو هغو اړوندو ادارو او کسانو څخه مننه کوم چې د طبي کتابونو د چاپ په برخه کې یې هر اړخیزه همکاري کړې ده.

هیله مند یم چی نوموړې پروسه دوام وکړي او د نورو برخو اړوند کتابونه هم چاپ شي.

په درنښت

پوهاند ډاکتر عبیدالله عبید

د لوړو زده کړو وزیر

کابل، ۱۳۹۲

د درسي کتابونو د چاپ پروسه

قدرمنو استادانو او ګرانو محصلینو!

د افغانستان په پوهنتونونو کې د درسي کتابونو کموالی او نشتوالی له لویو ستونزو څخه ګڼل کېږي. یو زیات شمیر استادان او محصلین نوي معلوماتو ته لاس رسی نه لري، په زاړه میتود تدریس کوی او له هغو کتابونو او چپترونو څخه ګټه اخلی چې زاړه دي او په بازار کې په ټیټ کیفیت فوتوکاپي کېږي.

د دې ستونزو د هوارولو لپاره په تېرو دوو کلونو کې مونږ د طب پوهنځیو د درسي کتابونو د چاپ لړۍ پیل او تر اوسه مو ۱۱۶ عنوانه طبي درسي کتابونه چاپ او د افغانستان ټولو طب پوهنځیو ته استولي دي.

دا کړنې په داسی حال کې تر سره کېږي چې د افغانستان د لوړو زده کړو وزارت د (۲۰۱۰ـ۲۰۱۴) کلونو په ملي ستراتیژیک پلان کې راغلي دي چې:

"د لوړو زده کړو او د ښوونې د ښه کیفیت او زده کوونکو ته د نویو، کره او علمي معلوماتو د برابرولو لپاره اړینه ده چې په دري او پښتو ژبو د درسي کتابونو د لیکلو فرصت برابر شي د تعلیمی نصاب د ریفورم لپاره له انګریزی ژبې څخه دري او پښتو ژبو ته د کتابونو او درسی موادو ژباړل اړین دي، له دې امکاناتو څخه پرته د پوهنتونونو محصلین او استادان نشي کولای عصري، نویو، تازه او کره معلوماتو ته لاس رسی پیدا کړي".

د افغانستان د طب پوهنځیو محصلین او استادان له ډېرو ستونزو سره مخامخ دي. نویو درسي موادو او معلوماتو ته نه لاس رسی، او له هغو کتابونو او چپترونو څخه کار اخیستل چې په بازار کې په ډېر ټیټ کیفیت پیداکېږي، د دې برخې له ځانګړو ستونزو څخه ګڼل کېږي. له همدې کبله هغه کتابونه چې د استادانو له خوا لیکل شوي دی باید راټول او چاپ کړل شي. د هیواد د اوسنی حالت په نظر کې نیولو سره مونږ لایقو ډاکترانو ته اړتیا لرو، ترڅو وکولای شي په هیواد کې د طبي زده کړو په ښه والي او پرمختګ کې فعاله ونډه واخلي. له همدې کبله باید د طب پوهنځیو ته زیاته پاملرنه وشي.

برابر شی خو بیا هم کیدای شی د کتاب په محتوی کی ځینی تیروتنی او ستونزی وجود ولری ، نو له دی امله له درنو لوستونکو څخه هیله مند یو تر څو خپل نظریات او نیوکی د مولف او یا زمونږ په پته په لیکلی بڼه را ولیږی، تر څو په راتلونکی چاپ کی اصلاح شی.

د افغان ماشومانو لپاره د جرمنی کمیټی او دهغی له مشر ډاکتر ایروس څخه ډېره مننه کوو چی ددغه کتاب د چاپ لګښت یی ورګړی دی. دوی په تیرو کلونو کی هم د ننګرهار د طب پوهنځی د ۲۰ عنوانه طبی کتابونو د چاپ لګښت پر غاړه درلود.

په ځانګړي توګه د جی آی زیت (GIZ) له دفتر او CIM (Center for International Migration and Development) یا د نړیوالی پناه غوښتنی او پرمختیا مرکز چې زما لپاره یې په تېرو دریو کلونو کې په افغانستان کې د کار امکانات برابر کړی دي هم مننه کوم.

د لوړو زده کړوله محترم وزیرښاغلي پوهاند ډاکترعبیدالله عبید ، علمی معین ښاغلي پوهنوال محمد عثمان بابری، مالي او ادري معین ښاغلی پوهنوال ډاکتر ګل حسن ولیزي، د ننګرهار پوهنتون رییس ښاغلی ډاکتر محمد صابر، د پوهنتونواو پوهنځیو له ښاغلو رییسانو او استادانو څخه هم مننه کوم چې د کتابونو د چاپ لړۍ يي هڅولی او مرسته يي ورسره کړی ده.

همدارنګه د دفتر له ښاغلو همکارانو څخه هم مننه کوم چې د کتابونو د چاپ په برخه کې يې نه ستړی کیدونکی هلی ځلی کړی دي.

ډاکتر یحیی وردګ، د لوړو زده کړو وزارت
کابل، مارچ ۲۰۱۳
د دفتر ټیلیفون: ۰۷۵۶۰۱۴۶۴۰
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تراوسه پوري مونږ د ننګرهار، خوست، کندهار، هرات، بلخ او کاپیسا د طب پوهنځیو او کابل طبی پوهنتون لپاره ۱۱۶عنوانه مختلف طبي تدریسي کتابونه چاپ کړي دي. د ننګرهار طب پوهنځی لپاره د ۲۰ نورو طبي کتابونو د چاپ چارې روانې دي. د یادونې وړ ده چې نوموړي چاپ شوي کتابونه د هیواد ټولو طب پوهنځیو ته په وړیا توګه ویشل شوي دي.

ټول چاپ شوی طبی کتابونه کولای شی د www.ecampus-afghanistan.org ویب پاڼی څخه ډاونلوډ کړی.

کوم کتاب چې ستاسی په لاس کې دی زمونږ د فعالیتونویوه بېلګه ده. مونږ غواړو چې دې پروسې ته دوام ورکړو تر څو وکولای شو د درسي کتابونو په برابرولو سره د هیواد له پوهنتونو سره مرسته وکړو او د چپټر او لکچر نوټ دوران ته د پای ټکی کېږدو. د دې لپاره دا اړینه ده چې د لوړو زده کړود موسساتو لپاره هر کال څه نا څه ۱۰۰ عنوانه درسی کتابونه چاپ کړل شي.

د لوړو زده کړو د وزارت، پوهنتونونو، استادانو او محصلینو د غوښتنې په اساس په راتلونکی کی غواړو چې دا پروګرام غیر طبي برخو لکه ساینس، انجنیري، کرهنې، اجتماعی علومو او نورو پوهنځیو ته هم پراخ کړو او د مختلفو پوهنتونونو او پوهنځیو د اړتیا وړ کتابونه چاپ کړو.

له ټولو محترمو استادانو څخه هیله کوو، چې په خپلو مسلکي برخو کې نوي کتابونه ولیکي، وژباړي او یا هم خپل پخواني لیکل شوي کتابونه، لکچر نوټونه او چپټرونه اپډېټ او د چاپ لپاره تیار کړي. زمونږ په واک کې یی راکړي، چې په ښه کیفیت چاپ او وروسته یې د اړوندې پوهنځی، استادانو او محصلینو په واک کې ورکړو. همدارنګه د یادو شویو ټکو په اړوند خپل وړاندیزونه او نظریات زمونږ په پته له مونږ سره شریک کړي، تر څو په ګډه پدې برخه کې اغیزمن ګامونه پورته کړو.

له ګرانو محصلینو څخه هم هیله کوو چې په یادو چارو کې له مونږ او ښاغلو استادانو سره مرسته وکړي.

د یادونی وړ ده چی د مولفینو او خپروونکو له خوا پوره زیار ایستل شوی دی، ترڅو د کتابونو محتویات د نړیوالو علمی معیارونو په اساس

سریزه

د ټولو څخه د مخه د پاک الله (ج) شکر پر ځای کوم چه ددی توان ئی را په برخه کړه چه د داخلی د بیړنیو پیښو د درملنی لارښود تر عنوان لاندی کتاب تالیف ئی را په برخه کړه.

په نوموړی کتاب کی د داخلی عاجلی پیښی چه OPD او ICU ته راځی د هغی د بیړنی درملنی لارښود شویدی. په ډیر لنډه ډول باندی تر څو محترم ډاکتر صاحبان په آسانه طریقه و کولای شی د ناروغ درملنه وکړی.

د کتاب د لیکلو په وخت د نړی د منل شویو Text books ، انټرنټ د داخلی څانګی د محترمو استادانو د علمی اثارو، نشر شوی لیکنو څخه استفاده شویدی.

ددی کتاب د لیکلو موخه دادی تر څو د عاجلو پیښو لپاره یوه ځانګړی Guidline څخه کار واخیستل شی تر څو د درملنی په وخت کی دوګونی ناروغانو ژوند وژغورل شی.

په پایله د محترم ډاکتر یحیی وردګ او د هغی د مکمل ټیم څخه د زړه له کومی مننه کوم چه ددی کتاپ په چاپولو کی هر نوعه هڅه او هاند ئی کړیدی تر څو نوموړی کتاب چاپ شی او د ګرانو لوستونکو لپاره ورکړل شی.

پوهنوال ډاکتر ایمل "شیرزی"

د ننګرهار د طب پوهنځی د داخلی څانګی استاد

ورسوله، چې وروسته يې د پوهنياري علمي رتبې ته ارتقا وکړه.

په ۱۳۸۰ هـ شمسي د کورپولمونل (CorPulmonal) په ناروغانو کې د توبرکلوز د پېښو څېړنو تر عنوان لاندې علمي تحقيق يې ترسره کړ، چې پوهنملۍ رتبې ته يې ارتقاء وکړه.

په ۱۳۸۴ هـ شمسي د زړه په احتشاء کې د چپ بطين د عدم کفايې د پېښو د څېړنې تر عنوان لاندې علمي تحقيق څخه وروسته پوهندوی علمي رتبې ته ارتقاء وکړه.

استاد سربېره پردې تدريسي کتاب، چې د پوهنوالۍ علمی رتبې د ارتقاء لپاره يې تأليف کړی، نور ګڼ شمېر علمي اثار هم ليکلي، چې په معتبرو علمي خپرونو کې نشر شوی دی.

په **۱۳۹۲** هـ ش کال کی ئی د بېړنی ناروغيو د درملنی لارښود تر سر ليک لاندی نوی کتاب تاليف کړيدی.

د مولف لنډه پېژندنه

پوهندوی دوکتور ايمل شيرزی د حاجي شير ولي زوی، په ۱۳۴۹ هـ شمسي کال کې د ننګرهار ولايت په جلال آباد ښار کې زېږېدلی دی.

په ۱۳۵۵ هـ شمسي کال کې د عبدالوکيل په منځنۍ ښوونځي کې شامل شوی، منځنۍ زده کړې يې د ننګرهار په عالي لېسه کې ترسره کړې، په ۱۳۶۶ هـ شمسی کال کې د کانکور ازموينې له لارې د ننګرهار طب پوهنځي کې شامل او په ۱۳۷۳ هـ شمسی کال کې د نوموړي پوهنځي څخه د فراغت ډيپلوم ترلاسه کړی. په ۱۳۷۴ هـ شمسي د ننګرهار د طب پوهنځي د فزيالوژي ډيپارتمنت کې په دنده وګمارل شو او په ۱۳۷۶ هـ شمسي کال کې د کانکور ازموينې څخه وروسته د ننګرهار طب پوهنځي د داخلي ځانګې کدر ته د نامزد پوهنيار په توګه جذب شو.

د نامزدۍ يوه کلنه دوره يې په بريالیتوب سره پای ته

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ACUTE SEVER ASTHMA IN ADULTS

RECOGNITION AND ASSESSMENT

Features of acute sever asthma.

1. Can't complete sentence in one breath.
2. Respiratory rate more than 30 breaths/min.
3. Pulse > 110 beats/min.
4. Use of accessory muscles.
5. Peak expiratory flow (PEF)< 50% of predicted.

Life threatening features.

1. PEF < 200l/min or < 33% of predicted.
2. Silent chest, cyanosis or feeble respiratory effort.
3. Bradycardia or hypotension.

A patient with sever or life threatening attacks may not be distressed may no have all these abnormalities. The presence of any one of these should alert the doctor.

4. Exhaustion, confusion or coma.

Investigations

1. Peak Expiratory Flow.
2. Oximetry.

If SaO2 < 92% or a patient has any life threatening features or not responding to the treatment, measure arterial blood gases.

Blood gas markers of a life-threatening attack.

1. Normal (38-45mmHg) or high PaCO2.
2. Sever hypoxia: PaO2 < 60mmHg irrespective of

treatment with oxygen.

3. Low pH (or high H+)

No other investigation are needed for immediate management.

Immediate treatment

1. Oxygen – high flow (40-60%) (CO2 retention is not usually aggravated by oxygen therapy in asthma)
2. **Sulbutamon 5mg** via an air or oxygen-driven nebulizer.
3. **Prednisolone** tablets 30mg (if on maintenance prednisolone, increase daily dose by 30mg) or hydrocortisone 250mg IV bolus, or both if very ill.
4. No sedative of any kind.
5. Chest X-Ray to exclude pneumonthorax.
6. If patient has coincident chronic bronchitis (regularly produces sputum), consider antibiotic treatment.
7. Chest physiotherapy is not indicated.

Patients with life threatening features.

1. Add ipratropium 500 mcg to the nebulised beta-agonist.
2. Give aminophyline 250 mg IV over 20 min (See prescribing regimens)
3. Do not give bolus amionophyline to patients already taking oral theophylines.
4. Transfer patient to ICU under the care of a

pulmonologist; the patient should preferably be accompanied by a doctor

5. Deteriorating PEF, worsening or persisting hypoxia, or hypercapnia
6. Exhaustion, feeble respirations, confusion, or drowsiness
7. Coma or respiratory arrest.

SUBSEQUENT MANAGEMENT

If patient is improving continue.

1. 40-60% oxygen.
2. Prednisolone at dose of 30mg daily.
3. Nebulised salbutamol 2.5-5mg 4 hourly.
4. Change to discharge medication (Check inhaler technique) 24hrs before discharge

If patient is not improving after 15 to 30 min.

1. Continue oxygen and steroids.
2. Give nebulised salbutamol more frequently, up to every 15-30 min.
3. Add ipratropium 500mg to nebulizer and repeat 6 hourly until patient is improving.

If patient is still not improving.

- Give aminophylline or salbutamol by infusin (see prescribing Regimens for doses) **If any life-threatening features (See above) present, transfer**

to ICU and refer to pulmonologist.

MONITORING TRETMETN

1. Repeat measurement of PEF 15- 30 min after starting treatment.
2. Oximetry: maintain SaO2 > 92%.
3. Repeat Blood Gas measurements within 2 hours of starting treatment if:
   - Initial Pa O2 60 mmHg unless subsequent SaO2 > 92%.
   - Initial PaCO2 normal or raised.
   - Patient deteriorates.
4. Chart PEF before and after giving nebulised or inhaled salbutamol and change to morning and evening before salbutamol dose.

DISCHARGE POLICY.

1. **When discharged from hospital patients should have:**
   - Been on discharge medication for 24 hours and have had inhaler technique checked and recorded by the PFT technician or medical / nursing staff.
   - PEF > 75% of predicted or his best and PEF diurnal variability < 25%.
2. Out patient follow up within 4 weeks.

Follow-up clinic.

1. **Check patient has:**
   - Minimal symptoms including nocturnal.
   - Minimal need for relieving bronchodilators.
   - Minimal limitation of activities.
   - Home PEF circadian variation < 20%.
   - PEF 80% or better, of predicted or best.
   - Minimal side effects from medicine.

EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE.

RECOGNITION AND ASSESSMENT.

Symptoms/Signs.

1. Worsening of cough.
2. Worsening of dyspnoea.
3. Wheezing.
4. Increase in sputum volume, tenacity (Difficult expectoration) and purulence.
5. Feverishness
6. Pyrexia (often)
7. Tachypnoea.
8. Tachycardia.
9. Indrawing of intercostals spaces (especially lower chest).
10. Prominent abdominal movement.
11. Pursed lip breathing tracheal tug, prolonged expiration
12. Predominant use of accessory muscles
13. Inspiratory or expiratory wheezes
14. Look for signs of corpulmonale (Peripheral oedema, raised JVP. Hepatomegaly)
15. Look for signs of type 2 respiratory failure (drowsiness, confusion, cyanosis, flapping tremor, papilloedema)

Investigations.

1. Arterial blood gases when breathing ari.
2. Chest X-ray.
3. ECG.
4. Sputum (Inspect for purulence and viscosity, and send for culture)
5. Blood complete
6. Blood cultures (If pyrexia)
7. Urea & elecrrolytes.

Differential diagnosis.

1. Exacerbation of asthma: if in doubt treat as such (See acute severe asthma in Adults)
2. Pnemothorax: even a small one can be dangerous (Mortality in advanced COPD complicated by pneumothorax is 5%)
3. Left ventricular failure (See cardiac Failure)
4. Pulmonary embolism (see Pulmonary embolism)
5. Review medication for sedative and beta blockers.

IMMEDIATE TREATMETN.

1. **Oxygen:** Start with 24% by ventimask or 1-2/min via nasal prongs and watch for Co2narcosis. See Respiratory Failure.

   High percentage (> 24%) 02 must NOT be given unless arterial blood gases confirms o CO2 retention.

Antibiotics.

2. Usual organisms: Strep, Pneumoniae and H.influenzae. consider staphy aureus, if influenza prevalent.
3. Co-Amoxiclave 625% orally 8 hourly.
4. Only if severely ill, give ciprofloxacin 1-2gm dialy for 48 hours followed by co amoxiclav 625mg orally 8 hourly.
5. Quinolones like ciprafloxcin, levofloxacin, Moxifloxacin or Gemifloxacin orally are alternate options.
6. If stuph aurieus suspected, add flucloxacillin 500mg orally IV 6 hourly.

Bronchodilators.

7. Salbutamol (2.5-5mg) via air-drien nebulizer 4-6 hourly.
8. Consider adding ipratropium bromide (500mcg) via nebulizer 6 hourly.
9. If not improving after 4 hours, add aminophyline infusion (See prescribing Regiments)

Corticosteroid

10. Prednisolone tablets 30 mg daily.
11. If already on maintenance (long term) dose of prednisolone, increase the daily dose by 30mg.
12. If severly ill give hydrocortisone 100mg iv bolus 6 hrly

13. Correct dehydration.

Physiotherapy.

14. Only aids the clearance of sputum.

Mechanical ventilation.

15. See Respiratory Failure.

SUBSEQUENT MANAGEMENT.

After 48 hours if improving:

1. Continue with oral antibiotics until sputum mucoid.
2. Continue nebulised bronchodilators if already using at home OR
3. Check inhaler technique and substitute appropriate inhaler device for nebulised bronchodilators.
4. Continue prednisolone at same dose for 7-10 days before stopping (No need to taper withdrawal)
5. Stop oxygen if PaO2 55mmHg but watch for deterioration.

If not improving:

1. Consider resistant organisms. Change antibiotic based on sputum culture result, where known.
2. Consider underlying disease (e.g. bronchogenic carcinoma, bronchieactasis etc)
3. Consider assessment for non invasive ventilation (See table and type II Respiratory Failure.

Monitoring TREATMENT

1. PEF measurement aiming to attain patient's best PEF rate when well (if known)
2. Arterial blood gases: See respiratory failure.
3. Sputum volume and conversion from mucopurulent/ purulent to mucoid.
4. Objective improvement as reflected by increased exercise tolerance.

DISCHARGE POLICY.

1. Check inhaler technique when changing from nebulizer therapy to metered dose inhaler or space devices.
2. Advice to stop smoking.
3. Advise to consult the doctor whenever sputum becomes purulent
4. Arrange prophylactic influenza vaccination.
5. If hypoxic, PaO2< 55mmHg (PaO2< 7.3kPa) after recovery consider referral to pulmonologist for domiciliary oxygen assessment.

COMMUNITY ACQUIRED PNEUMONIA

RECOGNITION AND ASSESSMENT.

Treat as pneumonia if symptoms are signs below plus new unexplained chest x-Ray shadowing and the illness is the primary clinical problem.

Symptoms.

1. Malaise, fever, rigors.
2. Vomiting, diarrhea.
3. Confusion (especially in the elderly)
4. Dyspnoea, cough
5. Sputum (May be bloodstained, viscid and difficult to expectorate).
6. Pleuritic pain.

Signs.

1. High grade fever (Often absent in the elderly).
2. Tachycardia.
3. Tachyponoea.
4. Localized crackles.
5. Bronchial breathing (In about one third of hospital Admissions)
6. Chest signs may be absent or masked by other respiratory signs (e.g. COPD, CCF)

Enquire about pet birds (Psittacosis, chlamydia) and recent hotel residence away from home (Legionellosis)

Investigations.

1. Chest x-ray

2. Arterial blood gases
3. Blood complet, biochemical screen. CRP if available.
4. Microbiology:
   - Sputum inspection microscopy, culture and sensitivity.
   - Blood-cultures; in the seriously ill, serology for atypical organisms (influenza A and B, Coxiella burmetti, Chlamydia psittaci, Mycoplasma, pneumonia, Legionella, pneumophilia)
   - Urine-In the seriously ill for Legionella antigen.

Indicators for severity:

Mental confusion, multi lobar involvbement, respiratory failure, respiratory rate > 30/min, diastolic blood pressure < 60mm Hg, WBC Low (<4 X 10^9 /l) or very high **(> 20 X 10^9** /l**), serum urea > 50mg/l, serum albumin < 35 g/l.**

Differential diagnosis.

1. Pulmonary thromboembolism.
2. Lung cancer.
3. Left ventricular failure.

IMMEDIATE TREATMETN.

Supportive.

1. Oxygen to maintain PaO2> 60mmHg (8kPa)
   - In patients with COPD, start with 24% by ventimask or 1L/min via nasal prongs and

watch for signs or CO2 narcosis. See respiratory failure.

2. Maintain fluid balance.
3. Adequate analgesia for pleuritic pain: Diclofenac sodium 25-50 mg orally 8 hourly.
4. Treat any accompanying airflow obstruction or cardiac failure.
5. Physiotherapy only in patients with copious secretions.

Antibiotic Therapy.

6. Start as soon as diagnosis is made; therapy should always cover streptococcus pneumonia.
7. Route of administration depends on the severity of illness and likely pathogens.
    - Hospital treated not severe oral: co amoxiclav 625mg 8 hourly plus Clarithromycin 500mg 12 hourly or erythromycin 500mg 6 hourl.
    - If I/V treatemtn is needed: Co amoxiclave 1.2gm I.V 8 hourly plus Clarithromycin 500mg 12 hourly I.V or Erythromcin 500mg I/V 6 hourly.
    - Alternative for hospital treated not severe: Fluroquinolones with enhanced pneumococcal activity e.g. Levofloxacin

500mg daily, Moxifloxacin 400 mg daily or Gemifloxacin 320 mg daily.

- Hospital Treated Severe Co Amoxiclave 1.2gm IV 8 hourly or Ceftriaxone 2mg IV dialy Cefotaxime 1gm I.V 12 hourly plus Clarithromycin 500mg I.V 12 Hourly or Erythromycin 500mg IV 6 hourly.
- Alternative for Hospital treated: Severe; Fluroquinolones with enhanced Pneumococal activity e.g Levofloxacin 500mg IV daily or Moxifloxacin 400mg IV dialy or Gemifloxacin 320 mg daily, plus Benzypencillin 1.2g IV 6 hourly.

8. Staphylococcal pneumonia (Consider if influenza suspected) Co-amoxiclav 1.2 g IV 8 hourly plus Flucloxacilli 2g IV 6 hourly.
9. Atypical Pneumonia.
    - Legionella sp./ Mycoplasma Erythromycin 500 mg or 1g orally IV by infusion 6 hourly or Clarithromycin IV 500mg orally IV 12 hourly, or Alternately Fluoroquinolones e.g Levofloxacin, Moxifloxacin and Gemifloxacin.
    - Confirmed Q fever (Coxiella burneti) , psittacosis (Chlamydia psittaci) or suspected infection with Chlamydia sp or Mycoplasma: Oxytetracycline 500mg orally 6 hourly/

tetracycline 500mg IV by infusion 12 hourly depending on severity, OR erythromycin 500mg or 1g orally iv by infusion 6 hourly depending on severity.

10. Aspiration Pneumonia.

Co-amoxiclave 1.2 g IV 8 hourly and Metronidazole 500mg IV 8 hourly OR Ceftriaxonne 2gm IV daily.

11. Cavitating Pneumonia. Metronidazole (dose as for aspiration pneumonia) + Ceftriaxone (Doses as for severe pneumonia)

Assessment of requirement for intensive care.

1. **For intensive Care.**
   - Sever pneumonia.
   - Arterial PaO2 < 60mmHg (8 kPa) with inspired oxygen > 60%
   - Severe acidosis: PH< 7.25
   - Exhausted, drowsy or unconscious patient.
   - Respiratory or cardiac arrest.
   - Shock.

MONITORING TREATMENT.

2. In hypoxaemic patients, repeat arterial blood gases 1 hourl after a change of inspired oxygen concentration; continous assessment with a pulse oximeter is ideal.
3. Pulse, BP, temperature and arespiratory rate 4

hourly until patient is stable.

4. Biochemical screen: repeat every 24 to 48 hr while significant abnormalities persist.
5. Chest x-ray; repeat if patient not improving after 72 hr despite adequate therapy.

SUBSEQUENT MANAGEMENT.

Duration of antibiotics.

1. If IV route used on admission, change to oral when patient is a febrile for 24 hours and clinical parameters stable. Use oral antibiotic to which microbe is sensitive.
2. In uncomplicated pneumonia 5 to 7 days treatment.
3. In patient with severe pneumonia, staphylococcal pneumonia, or Legionella pneumonia, antibiotics should be continued for 2 weeks or longer.

Failure to respond to therapy.

1. Incorrect diagnosis (e.g pulmonary embolism, pulmonary edema, pulmonary eosinophilia, wagener's granulomatosis)
2. Resistant organism (e.g ampicillin-resistant haemophilus, mycoplasma, psittacosis. Q fever or staphylococcal pneumonia.
3. Unrecognized pulmonary tuberculosis.
4. Unrecognized immunodeficiency (eg HIV infection leading to pneumocystis pneumonia)

Complications.

- Parapneumonic effusion or empyema-aspirate for culture and drain.
- Lung abscess.
- Bronchial obstruction-refer to pulmonologist.
- Pulmonary embolism-see Pulmonary embolism.
- Fever related to drug therapy omit therapy for 48 hr.

DISCHARGE POLICY.

Follow up with chest x-ray about 6 weeks after discharge to ensure that resolution of radiological shadowing is occurring.

HOSPITAL ACQUIRED PNEUMONIA

RECOGNITION AND ASSESSMENT.

Definition.

Pneumonia occurring not less than 48 hr after hospitalization and excluding any infection that is incubating at the time of admission.

Symptoms.

1. Fever, rigors.
2. Confusion.
3. Cough, dyspnoea.
4. Purulent sputum.
5. Pleuritic chest pain.

Signs.

1. Fever
2. Tachycardia.
3. Tachypnoea.
4. Crackles
5. Bronchial breathing.
6. Effusion.

Investigations.

1. Chest x-ray
2. Sputum- inspection, microbiology, culture and sensitivity.

False positive results occur often due to contaminants in these patients.

3. Two sets of blood cultures from separate sites.

4. Arterial Blood Gases.
5. Blood complete, biochemical screen.
6. Diagnostic aspiration if patient has parapneumonic effusion.
7. Consider urinary Legionella antigen.

Differential Diagnosis.

1. Congestive heart failure.
2. Pulmonary thrombembolism.
3. Drug reactions.
4. Pulmonary haemorrhage.
5. Adult respiratory distress syndrome.

IMMEDIATE TREATMENT

Contact ICU early if deteriorating.

Supportive.

1. Oxygen to maintain arterial PaO2 60mmHg (8 kPa).
2. Maintain fluid balance.
3. Adequate analgesia for pleuritic chest pain: Diclofenac 25-50mg orally 8 hourly.
4. Physiotherapy in patients with copious secretions.

Antibiotic Therapy.

1. Start antibiotic treatment as soon as clinical criteria for diagnosis are met, do not wait for microbiological confirmation.
2. Initial therapy may be modified once results of respiratory tract secretions or blood cultures

become available.

3. Route of administration depends on severity of illness.

Antibiotic Regimens

Non-severe suitable for oral drugs:

- Co-Amoxiclave 625mg 8 hourly.

Severe requiring parenteral treatment:

- Ceftazidime 2g 12 hourly or Ceftriaxone 2gm daily.
- If anaerobic cover required add Metranidazde 500mg 8 hourly.
- Patient who have had previous antibiotics, prolonged hospital stay, previous ICU stay, used steroids or immunosuppressed will require cover for pseudomonas and MRSA (methicillin resistant staph aureus)
- Consider Ciprofloxacin 500mg 12 hourly + Gentamycin 8 hourly or Ceftazidime 2gm 12 hourly for pseudomonas.
- Vancomycin 1gm 12 hourly or Linezolid 600mg 12 hourly should be considered for MRSA.

Sever hospital acquired pneumonia.

The presence of any of the following indicates a severe illness.

1. Respiratory failure with PaO2 < 60 mmHg (8kPa) and

or paCO2 > 50 mmHg (6.7kPa).

2. Respiratory rate > 25/min.
3. Rapid radiographic progression, multilobar pneumonia, or cavitation of lung infiltrate.
4. Diastolic BP< 60 mmHg.
5. WBC low (< $4X10^9$) or very high (> $20X\ 10^9$ /l)
6. Poor urine output or rising cratinine.
7. Metabolic acidosis.

SUBSEQUENT MANAGEMENT.

Duration on antibiotic.

Usually for seven days, but in severe cases continue treatment for at least two weeks.

Failure to respond to treatment.

1. Incorrect diagnosis (See differential diagnosis)
2. Re-evaluate and consider bronchoscopy to obtain protected specimens brushing and/or Bronchoaleveolar lavage specimens for quantitative cultures refer to pulmonologist.
3. Complications: empyema, lung abscess refer to pulmonologist.

MONITORING TREATMENT.

1. In hypoxaemic patients repeat ABG 1 hour after change of inspired oxygen, continue assessment with pulse oximeter.
2. Pulse, BP, and temperature 4 hourly until patient is stable.

3. Repeat chest x-ray after 72 hour if patient not improving despite the therapy.
4. Repeat biochemical screen every 24 to 48 hours while significant abnormalities persist.
5. Refer to ICU if no improvement.

DISCHARGE POLICY.

Follow up with chest x-ray about 6 weeks after discharge to ensure that resolution of radiological shadowing is occurring.

PULMONARY EMBOLISM

Pulmonary thromboembolism (PTE) is often missed clinically and the diagnosis should be suspected in patients with vague symptoms in those not responding to initial therapy or when there has been an unexplained deterioration. PTE is rare in patients under age 40 in the absence of risk factors. Most episodes follow popliteal or illiofemoral DVT; Calf DVT rarely leads to PTE although it can propagate to become popliteal DVT with attendant risks. Small emboli present with dysponea moderate size emboli present with signs of infarction and pleuritic pain.

RECONGNITION AND ASSESSMENT.

Symptoms.

1. Dyspnoea (Present in 90% of cases) –may be of sudden onset.
2. Pleuritic chest pain.
3. Haemoptysis.
4. Syncope.

Signs

1. May be absent
2. Tachypnoea (>20/min)
3. Fever.
4. Pleural rub
5. Tachycardia.

Massive pulmonary embolism.

1. Severe dyspnoea.
2. Dull central chest pain

3. Elevated venous pressure
4. Hypotension and shock
5. Right heart failure.

Risk Factors.

1. DVT (Present in 50% of patients)
2. Major trauma.
3. Recent surgery (Especially abdominal/pelvic)
4. Obesity
5. Immobility
6. Age (Increasing)
7. Malignancy.
8. Oral contraceptive pill
9. Late pregnancy
10. Puerperium
11. Hyperviscosity syndromes
12. Nephrotic syndrome
13. Defective fibrinolyssi
14. Antithroombin iii deficiency
15. Lupus anticoagulant
16. Protein C and S defeciencies.

Investigations.

1. **ECG.**
   - These are often normal and should not be used to confirm or refute the diagnosis, but are useful for identifying other diseases and

to explain symptoms.

- ECG may show sinus tachycardia, an SI, Q3 and T3 pattern, right bundle branch block, p pulmonale or right axis deviaton.

2. Chest x ray.
    - Chest x-ray may show non-specific shadows or a raised hemidiaphragm, pulmonary oligaemia, linear atelectasis or small pleural effusion.
3. Blood gases may show low PaCO2 and or PaO2 (breathing room air) with 18hr of symptoms.
4. Computed tomographic pulmonary angiography is the recommended initial lung imaging modality for non massive P.E. Patients with negative CTPA do not require further imaging for P.E arrange CT PA if abnormal CXR or cardio respiratory diseases.
5. D-Dimer should be considered only after assessment of clinical probability.
    - A negative D-Dimer reliably excludes PE.
    - In patients with low probability of P.E, it is not a routine screening test and should ot be performed if the clinical probability is high.
6. Baseline INR and APTT.
7. Ventilation-perfusion (V/Q) scan. Arrange as soon as possible. A normal perusion scan virtually excludes pulmonary embolism. The report will usually be in

terms of low, intermediate and high probabilities.

8. Also investigate for cause of PTE e.g Doppler US of legs, US of pelvis (occult malignancy)

Confirming the diagnosis

1. Management is determined by the perceived clinical probability (judged from clinical assessment and results of investigation) and the rest of the V/Q scan or CTPA
2. Clinical probability is considered high if other diagnoses are thought unlikely on clinical grounds and in light of the white cell count, chest x-ray and ECG, or if the patient has any of the following major risk factors:
    - Recent immobilization/major surgery.
    - Recent trauma and or surgry to lower limbs
    - Clinical DVT
    - Previous proven DVT or PTE
    - Pregnancy/Post partum
    - Major medical illness

Differential diagnosis.

1. Pneumonia.
2. Myocardial infarction.
3. Exacerbations of dyspnoea in conditions such asthma and chronic obstructive airways disease, dyspnoea may be due to pulmonary embolism.

IMMEDIATE TREATMETN

General.

1. Oxygen 35-50% (higher is shocked), mechanical ventilation if patient is tiring.
2. Adequate analgesia for pleuritic pain; Diclofenac 25-25 mg orally 8 hourly if contraindicate or ineffective. Diamorphine 5-10mg SC 4 hourly with Metoclopramide 10mg IV.
3. Allow right atrial pressure (i.e. JVP) to remain high if elevated then give (Crystalloids/colloids)
4. **AVOID** diuretics.

Specific

- Heparin. Loading dose of 5000 iu Iv followed by 1400 iu/hr IV on suspicion of diagnosis. Check APTT 4 to 6 hr and adjust the infusion rate according to APTT ration (see prescribing regiments) if infusion pump not available then loading dose of 15000 unit s/c followed by 15000 s/c B.D, aiming for a ration of 1.5 to 2.5 time the control value. Heparin has a short half life (40-90 mins), so infusion must not be nterrupted.

Thrombolysis.

1. If life-threatening features present (right heart failure, shock), give streptokinase by peripheral vein (see prescribing regimens). Monitor with daily thrombin time, aiming for va value 2-4 tiems the

normal. Failure of response to streptokinase is an indication for emergency direct thrombolysis, catheter thromboembolectomy or pulmonary embolectomy.

2. Do not start heparin or warfarin until 4 hr after thrombolytic therapy has ceased.

SUBSEQUENT MANAGEMENT.

1. Daily clinical examination for signs of further embolisation, right heart failure and secondary infection of a pulmonary infarct.
2. Warfarin should be started as soon as the diagnosis is confirmed (see prescribing regiments.
3. Heparin may be discontinued when the INR has, for two consecutive days, been within the therapeutic range: 2 to 3 (3 to 4 for recurrent PE)

RESPIRATORY FAILURE

RECONGNITONA DN ASSESMENT.

Respiratory failure is present when the lungs are unable to maintin normal gas exchange at rest, so that arterial PaO2 is below 60 mmHg (8 kPa) and or arterial PaCO2 is above 50mmHg (6.7kPa) it has many causes (see below) and these must be indentified and treated as part of the oerall management.

Symptoms and signs

1. Central cyanosis (difficult to detect in anaemic patients).
2. Drowsiness.
3. Warm peripheries, bounding pulse, tachycardia, flapping tremor
4. Papilloedema (In patients with hypercapneoa).

Investigations

1. Arterial blood gases on air.
2. Chest x-ray.
3. Blood complete
4. Urea and electrolytes.
5. ECG

Consider whether:

1. Type 1 (oxygenation failure) Low PaO2 normal PaCO2 owing to asthma, pneumonia, pneumothorax, pulmonary edema,pulmonary embolism, epigottitis and pulmonary fibrosis.
2. Type 2 (Ventilatory failure) Low PaO2 high PaCO2

owing to exacerbation of COPD, neuromuscular disorders, encephalitis, muscular dystrophies, or use of respiratory depressant drugs.

IMMEDIATE TREATMETN.

Treat underlying cause.

Oxygen.

1. **Type 1**
   - High concentration (40-60%)
   - Measure arterial blood gases after 30-60 min increasing inspired oxygen appropriately.
   - Aim to raise PaO2 above 50mm Hg (6.7kpa)
2. **Type 2**
   - Low concentration start with 24% ventimask or 1 l/min via nasal prongs.
   - Measure arterial blood gases after 30-60 min.
   - Aim to raise PaO2 above 50mm Hg (6.7 kPa)
   - Keep PaCO2 below 60 mm Hg (8 kPa) or confine its rise to less than 7.5 mm Hg (1 kPa)
   - If 24% O2 well tolerated increase to 28% ventimask or 2 l/min nasal prongs these deliver an inspired oxygen concentration (Fi O2 of about 25-30%)
   - Repeat afterial bloog gases again after 30-60 min.

It may be necessary to accept only a modest increase in PaO2 most patients will survive if PaO2 is above 50 mg (6.7 kPa)

SUBSEQUENT MANAGEMENT.

If improving:

1. Continue oxygen, adjusting the inspired oxygen concentration to achieve PaO2 55-60mm Hg (7.3-8.0 kPa).
2. Treat underlying disease.

If not improving:

If not improving consider first non-invasive ventilation (NIV)

1. **Selection criteria for NIV:** (at least 2 should be present).
    - Moderate to severe dyspnoea with use of accessory muscls and paradoxical abdominal motion.
    - Moderate to severe acidosis (pH< 7.30-7.35) and hypercapnia PaCO2 > 45-60 mm Hg (6.0-8.0 kPa)
    - Respiratory frequency > 25 breaths per minute.
2. **Contra selection (any may be present)**
    - Respiratory arrest.
    - Cardiovascular instability (Hypotension, arrhythmias, myocardial infarction)
    - Somnolence, impaired mental status,

uncooperative patient.

- High aspiration risk; viscous or copious secretions.
- Recent facial or gastroesophageal surgery.
- Caraniofacial trauma. Fixed nasopharyngeal abnormalities.
- If NIV contraindiacated or si not effective, consider mechanical ventilation.

3. **Indications for invasive mechanical ventilation.**

Severe dyspnoea with use of accessory muscles and paradoxical abdominal movements.

- Respiratory frequency > 35 breaths per minute.
- Life threatening hypoxemia PaO2 <40mm Hg (5.3 kPa) or PaO2/FiO2 < 200 mmHg>
- Severe acidosis (pH <7.25) and hypercapnoea PaCO2 > 60 mm Hg (8.0 kPa)
- Respiratory arrest.
- Somnolence, impaired mental status.
- Cardiovascular complications (Hypotension, shock, heart failure
- Other complications (metabolic abnormalities, sepsis, pneumonia, pulmonary embolism, barotraumas, massive pleural effusion)
- NIPPV Failure.

Mechanical ventilation.

1. **Type 1.**
   - If PaO2 cannot be maintained above 50 mm Hg (6.7 kPa) despite hight concentration oxygen therapy, especially in acute severe asthma with life-threatening features (see acute severe asthma in adults), contct ICU and request transfer.
2. **Type 2.**
   - Important to consider overall outlook before embarking on this. In general, ventilator support is appropriate in a previously y active patient with a good quality of life over the previous 6 months, or where the history is unclear. There is no point in embarking on mechanical ventilation when the patient has end stage chronic respiratory failure and there is no cure for underlying diseases.

MONITORING TREATMENT

1. **Type 1.**
   - For patients with type 1 respiratory failure secondary to asthma, see acute severe asthma in adults.
   - Regular 6 hourly (at least) arterial blood gases until patient stabilizes.
2. Ensure that infection is adequately treated, that a

collapsed lung in pneumothorax is completely expanded (See spontaneous Pneumothorax), or that anticouagulation is stabilized following a pulmonary embolism (see pulmonary embolism)

DISCHARGE POLICY.

1. Follow up at the discretion of the supervising physician.
2. Advice on life-style appropriate to the underlying disease that precipitated the admission.
3. Consider referral to a pulmonologist for assessment for long term domiciliary oxygen therapy or home non invasive ventilation.

ACUTE MYOCARDIAL INFARCTION (MI)

RECOGNITION AND ASSESSMENT

Symptoms.

1. Severe, persistent chest pain, epigastria pain.
2. Dyspnoea.
3. Fear.

Signs.

1. Pallor.
2. Sweating
3. Anxiety.
4. Peripheral vasoconstriction.
5. Shock.

Investigations.

1. ECG (see below).
2. The troponin level peaks at about 12 hrs from the onset of myocardial damage and remains elevated for 5 days. The specimen for troponin should be taken 12 hourly after the onset of chest pain to confirm or exclude myocardial damage.
3. CK-MB will only be used for suspected re-infarction (because the troponin level remains elevated for several days after a myocardial infarction).
4. AST and LDH are no longer being used for the diagnosis of myocardial ischemia
5. Plasma cholesterol (with 12 hr of onset of symptoms; otherwise eave for at least 6 weeks)

6. Venous blood glucose.

IMMEDIATE TREATMENT.

1. **Aspirin** 300 mg (Chew, macerate and awallow).
2. **Morphine** 2-8mg iv until pain relieved (to maximum 10 mg).
3. **Metoclopramide** 10 mg iv over 1-2 min or Diphenhydramine 25mg iv if vomiting.
4. **Nitrates** (Needs caution in inferior MI with RV infarction).
5. Consider thrombolytic therapy (see below).
6. **Oxygen** high flow 40-60% (24% in patients with co-existent COPD)
7. **Bisoprolol** 2.5 mg orally or **Metoprolol** 50mg orally (Unless contraindicated;.
8. **Ace Inhibitors:** all patients with ST elevated MI (STEMI) Or LBBB with infarction should receive ACE inhibitors with first 24 hours..
9. **Clopidogrel** 300mg orally.

All patients with suspected acute myocardial infarction should be treated and admitted to the CCU under the are of cardiologist.

Thrombolytic Therapy.

Start treatment as soon as diagnosis is made and contra-indications excluded. The interval between patients arrival and commencement of thrombolytic therapy (door to needle time) should be less than 30 min. Time delay means

muscle lost.

Indications.

1. Presentation within 12 hr of onset of symptoms.
2. Typical cardiac chest pain persisting for more than 30 min.
3. More than 1mm ST segment elevation in 2 or more precordial leads or, 2 or more contiguous limb leads or > 1mm ST segment depression in leads V1 to V3 (Suggesting acute posterior infarction. Can be confirmed on doing leads V7-9)
4. LBBB with any of the following in leads V1-V3:
    a. > 1mm ST segment depression.
    b. > 1m St segment elevation where QRS omplex is positive.
    c. > 5mm ST segment elevation where QRS complex negative

Contraindications

Absolute:

1. Active bleeding.
2. Suspected aortic dissection.
3. Recent head trauma or intracranial tumor.
4. Previous hemorrhagic stroke at any time
5. Previous ischemic stroke within past 1 year.
6. Previous allergic reaction to fibrinolytic agent.
7. Trauma and/or surgery with in past 2 weeks at risk of bleed.

Relative:

1. Major trauma/major surgery within previous 2 weeks.
2. Non haemorrhagic stroke over 1 year ago.
3. Prolonged cardiac massage or intracardiac injection.
4. Known bleeding disorder or on warfarin with INR of more than 2.
5. Active dyspepsia or history og GI hemorrhages.
6. Uncontrolled sustained systolic BP > 180 mmHg.
7. Proliferative retinopathy.
8. Recent head injury
9. Pericarditis.
10. Lumbar puncture within past one month.
11. Non compressible arterial puncture eg. Subclavian
12. Pregnancy or postpartum.
13. Menstrual bleed or lactation.
14. Prior exposure to streptokinase (especially previous 6-9 months.)

Cardiogenic Shock and ventricular arrhythmias are not contraindications. There is no upper age limit for this treatment.

Choice of agent.

1. The standard agent is streptokinase (SK) 1.5 million units in 100ml of 0.9 sodium chloride by IV infusion over 1 hr (no advantage of routine heparin after SK) Streptokinase can be re-administered within 3 days

of first administration, but after 3 days, the likely presence of streptokinase antibodies precludes its further use for at least 24 months.

2. Alteplase (rtPA) can be used instead of streptokinase, For patients > 65 kg 15 mg iv bolus followed by 50 mg in 30 minutes and 35 mg in one hour followed by heparin infusion. Heparin 1000 iu/hr via infusion pump for 48 hr, adjusting the dose to maintain activated partial thromboplastin time (APTT) ration twice normal.

Complications:

1. Hypotension if this occurs de novo, stop IV infusion and recommence at a slower rate after BP has recovered.
2. Bradycardia usually responds to atropine 300 micro g IV.
3. Ventricular tachycardia or idioventricular rhythm usually self limiting and requires no therapy. If sustained, see Cardiac arrhythmias.
4. Arterial punctures, central venous cannulation and IM injections should be avoided in patients receiving thrombolytic therapy, unless essential to patient care.

SPECIFIC SITUATIONS.

Right ventricular infarct.

1. Aim to maintain high preload; initially give 1-2 litres

0.9% saline rapidly, if BP is less than 90 mmHg. (Monitor PCWP not > 18mmHg) avoid nitrates, diuretics.

2. Reduce afterload; especially if concomittent LV dysfunction, may need intra aortic balloon pump. Avoid inotropes and only use if all other measures fail to restore haemodynamics. Reperfusion of RCA may help.

Diabetic patients and patients with blood glucose > 280 mg/dl.

1. If the patient is diabetic with blood glucose < 280 mg/dl; continue the patients usual insulin regimen or oral hypoglycemic (Sulphonylureas or acarbose)
2. Metformin should always be discontinued.
3. Blood glucose > 280mg/dl, stop the patient's usual diabetic treatment and commence insulin glucose infusion (IGI)
4. Check plasma potassium.
5. Check baseline blood glucose
6. Insulin glucose infusion – dextrose 5% 500ml 12 hrly.
7. Soluble/Regular insulin 50 units in 50 ml sodium chloride 0.9% (1 unit/ml) with an initial infusion rate of 4 units/hour. Dextrose and insulin infusions should be given simultaneously via 3-way cannula.
8. Check blood glucose 1 hour after starting IGI. Use

table to adjust insulin infusion Rate.

Capillary glucose (mg/100ml)	Insulin infusion rate (units/hr=ml/hr)
> 300	Give 8 units of insuin as an intravenous bolus injection and increase infusion rate by 1 unit/hr.
200-300	Increase infusion rate by 0.5 unit/hr
130-200	Leave infusion rate unchanged
120-130	Decrease infusion rate by 1 unit/hr.

SUBSEQUENT MANAGEMENT.

1. **Aspirin** 75-150mg oral daily (to be continued indefinitely)
2. **Bisoprolol** 2.5 mg orally daily or Metroprolol 50 mg orally twice daily (unless contra-indicated)
3. **Clopidogrel** 75mg oral daily.
4. If pain persistent, consider nitrate infusion, or further dose of Metroprolol 5mg IV if heart rate > 70/min and systolic BP > 100Hg.
5. Early return to physical activity unless complications ensure:
   - Day 1 bed rest.
   - Day 2-3 sit out of bed.

   - Day 3-4 walk round and to toilet
   - Day 4-5 extend walking distance
   - Day 5-6 try stairs
6. All patients should receive appropriate lifestyle and dietary advice.
7. Refer all patients who have been treated with glucose or insulin infusion to diabetologist/endocrinologist.

MONITORING TREATMENT

1. Continue ECG monitoring for 24-48 hrs (longer if continuing instability or arrhythmia)
2. Measure blood pressure 4 hrly for 24 hrs, then twice dialy.
3. Daily 12 lead ECG.
4. Observe for specific complications.

Arrhythmias
See cardiac arrhythmias.
Cardiac failure.
Management of acute cardiac failure after myocardial infarction.

1. Patients with significant left ventricular failure should have an angiotensin convertyin enzyme inhibitor induced as soon as the is practical.
2. Arrange an echocardiogram in patients with significant LVF and or anterior Q wave infarct, to document LV function and exclude LV aneurysm

and/ or thrombus.

Pericarditis.

1. More likely after large infarcts
2. Pain with persistent / intermittent pericardial rub 2-5 days after infarction
3. Adequate alalgesia especially high dose of aspirin 600 mg 6 hrly with proton pump inhibitors…. Avoid NSAIDS if possible,; it increases risk of cardiac rupture.

RECURRENT ISCHEMIC PAIN.

1. Isosorbide mononitrate orally 20mg bid or (nitrate infusion if necessary)
2. If persistent rest pain occurs, consider coronary angiography and possible angioplasty.
3. If re-infarction occurs during admission, contact consultant cardiologist immediately.

DISCHARGE POLICY

1. Check risk factors for recurrent MI (e.g smoking, hyper lipidaemia, hypertension, obesity) and advise or treat accordingly (mortality in first 2 yers is doubled in those who continue to smoke and is 3.5 times greater if total cholesterol > 6.5 mmol/l)
2. Explain graded return to full activity: Day 6-8 walk about home and garden, thereafter gradually increase activity for example, extending walks to 30

min by 2 weeks after discharge. Many patients can return to usual activity levels 4-6 weeks after discharge.

3. Advise not to drive for 4 weeks.
4. Warm about post infarct angina.
5. If no complications, discharge home on day 5-7.
6. Follow up in outpatient clinic after 4-6 weeks. Look for evidence of inducible ischemia.
7. Check that follow up has been arranged in diabetic clinic for all patients treated with glucose and insulin infusion.

UNSTABLE ANGINA

Definition

1. Sudden onset of frequent attacks of angina for the first time
2. Or sudden worsening of previously stable angina without change in medical treatment.
3. Or recurrent angina at rest.
4. An attack of angina that lasts for more then 20 minutes or keeps recurring despite use of glyceryl trintrate is an indications for hospitalization

Symptoms and signs.

1. Central chest pain/ tightness or discomfort (pain may also occur in the arm, shoulder, throat, jaw, teeth, back or upper abdomen)
2. Breathlessness.

Investigations

1. ECG on admission, during pain and at 24 hrs, if ST depression only during pain shows ischemia, (Consider posterior MI if in V1 and V2 and slow to resolve. Do leads v7-9)
2. ST elevation shows prinzemetal angina or acute MI.
3. ST elevation that persists after S/L Nitrates shows MI
4. CK MB and troponins at 12 and 24 hrs after onset of symptoms.
5. Blood complete

6. U & E
7. Random cholesterol

Differential diagnosis.

1. Pulmonary embolism.
2. Esophageal spasm
3. Musculoskeletal pain
4. Biliary colic
5. Peptic ulcer
6. Aortic valve disease
7. Mitral valve proplapse.
8. Hypertrophic cardiomyopathy.

IMMEDIATE TREATMETN.

1. **Asprin** 300 orally (chew, macerate and swallow)
2. Sublingual Nitroglycerine (0.3 – 0.6 mg) tablets 4 hrly, increase the dose if needed to relieve symptoms.
3. Atenolol 50 mg orally; or diltiazem 60mg orally if beta blocker contra indicated
4. Heparin 5000 iu IV followed by 1000 iu/hr by IV infusion pump for at least 24 hrs, continued until 24 hrs after pain is relieved.
5. Low molecular weight heparin as alternative to heparin.
6. Clopidogrel 300 mg stat, 75 mg per day.
7. Consult cardiology department.

SUBSEQUENT MANAGEMENT.

1. Aspirin 150 mg orally.
2. Altenolol 50 – 100 mg orally or
3. Diltiazem 60mg TDS if beta-blocker not indicated
4. If responding add isosorbide monoitrate 20-40 mg 12 hrly or isosorbide dinitrate 5-80 mg 8- 12 hrly
5. If NOT responding add nitrate (Isoket) infusion and consider of angiography / angioplasty and surgery
6. Morphine 2-4 mg IV
7. Metoclopromide 10mg IV
8. Simvastation 20-40mg or Altovstatin 10-80 mg daily before evening meals.
9. Hrly pulse and blood pressure monitoring unitl stable and then 4 hrly.
10. Repeat ECG and cardiac enzymes after 24 hrs.
11. Monitoring APTT to adjust heparin to APTT twice the control.

DISCHARGE POLICY.

1. If patient stable, no ECG and enzyme changes, can be discharged after 48 hrs.
2. Continue aspirin, nitrate, and beta-blockers.
3. Givve dietary advice, and modify risk factors (Smoking, hypertension, hyperlipidaemia, diabetes, obesity) and advise
4. ETT and further investigation and procedures if needed.

5. Risk stratification. If high TIMI risk score then early intervention is required.

CARDIAC FAILURE

RECOGNITION AND ASSESSMENT

Symptoms.

1. Fatigue
2. Dyspnoea of effort and when severe at rest
3. Orthopnea
4. Paroxysmal nocturnal dyspnoea
5. Swelling of feet and ankles
6. Cough
7. Wheeze

Signs

1. Tachycardia
2. Raised jugular venous pressure (JVP)
3. Hypotenison
4. Enlarged heart
5. Third/furth heart sounds
6. Crackles
7. Enlarged, tender liver, jaundice
8. Peripheral edema, ascites.

Investigations.

1. Blood complete
2. U & E, cardiac enzymes.
3. Echocardiography.
4. Arterial blood gases (if patient has dyspnoea at rest or severe pulmonary edema.)

Causes

Pump failure.

1. Acute myocardial infarction/ chronic myocardial ischemia.
2. Hypertensive heart disease
3. Cardiomyopathy / myocarditis
4. Drugs and toxins (Including alcohol)

Mechanical abnormality.

1. Valvular heart disease (Including infective endocarditis, post infarct mitral regurgitation).
2. Ventricular aneurysm, post-infarct septal defect.
3. Congenital heart disease
4. Pericardial constriction / tamponade

Excessive demand

1. Anemia
2. Thyrotoxisosi
3. Systemic infection

Arrhythmia.

1. Tachycardia, atrial fibrillation.
2. Bradycardia, heart block.

Differential diagnosis.

1. Dependent edema due to immobility.
2. Chronic obstructive pulmonary disease
3. Aute severe asthma
4. Pneumonia

5. Pulmonary embolism
6. Interstitial lung disease
7. Renal failure / nephritic syndrome
8. Cirrhosis
9. Constrictive pericarditis /cardiac tamponade.

IMMEDIATE TREATMENT

Acute pulmonary edema.

1. Nurse patient in sitting position in bed/chair
2. Oxygen high flow 40 to 60% (24% in patients with co-existent COPD)
3. Frusemide 40 mg by slow IV injection.
4. Morphine 2-5 mg by slow IV injection (1mg/ min) : 2.5 mg iv in elderly or frail patients.
5. Treatment hypertension (see Accelerated hypertension) or cardiac arrhythmias)
6. Additional therapy in severe cases: If systolic BP> 100mmHg consider Nitrate (Isoket) infusion. If systolic BP< 100mmHg, consider doubutamine (see Prescribing regiments)

SUBSEQUENT MANAGEMENT.

If responding:

1. Reduce salt intake (no added salt, avoid salty food)
2. Advise against excessive fluid intake.
3. Frusemide 40 mg orally daily (or an extra 40 mg daily if already talking frusemide. If peripheral edema gross (above knees), give frusemide 80 mg

daily by IV infusion, titrating dose upwards to achieve dialy weight loss of up to 0.5 kg. Continue until edema cleared before changing to maintenance oral dose, to maintain stable “dry” weight. Divide daily dose only if nocturnal dysponea troublesome (doses at 7am and 2 pm)

4. Identify causes(s) trigger factors and treat as appropriate.
   - If no cuases apparent and heart size and ECG are normal, discuss further investigation and anagement with consultant cardiologist.
   - Arrange echocardiogram.
5. Introduce ACE Inhibitor in all cases, unless clinical suspicion of significant mitral/ aortic stenosis or hypertorophic cardiomyopathy, or erenal function seriously impaired (plasma creatinine 3.5 mg/dL)
6. If ACE inhibitor coontraindiated or not tolerated, add spironolactone 25 mg orally daily to conserve potassium if plasma creatine < 1.7 mg/dl

If not responding:

If edema persists despite IV diuretics and optimal dose of ACE inhibitors, consider addition of

1. **Digoxin (** even if hert is in sinus rhythm).
2. **Add Mtolazone** orally in single dose 2.5-5 mg once only (if available).

If resistant to treatemtn despite these additional measures,

seek advice on further management from senior members of cardiology unit.

MONITORING TREATMENT.

1. Arterial blood gases: repeat 2 hr after starting oxygen.
2. Pulse, BP and respiratory rate 4 hrly until no longer dyspnoeic at rest.
3. Weight and fluid balance daily
4. U & E alternate days.
5. Chest x-ray repeat after 3 or 4 days to assess response.

DISCHARGE POLICY.

1. Mobilize once dysponea at rest subsides. Prolonged bed rest is counterproductive.
2. Discharge once;
   - Mobile around ward without dyspnoea.
   - Free of peripheral edema
   - Weight stable
   - Renal function stable
3. Arrange eCHO as outpatient
4. Encourage patient to exercise as much as possible.

Follow up clinic visits.

1. Check weight and compare with previous weight.
2. Review cardiac function.
3. Check U & E.
4. If echocardiogram confirms left ventricular

dysfunction and reveals no significant mitral/aortic stenosis, and renal function is not seriousl impaired (Plasma creatinine 3.5mg/dL) continue treatment with ACE inhibitor.

AORTIC DISSECTION

Symptoms.

1. Sudden chest pain, severe, tearing, may radiate retrosternallly, to neck, arms, interscapular area, or abdomen.
2. Paralysis due to involvement of erebral or spinal arteries.
3. Loss of consciousness.
4. Dyspnoea

Signs.

1. BP elevated, normal or low.
2. BP discrepancy between limbs.
3. Pulse deficit.
4. Aortic regurgitation murmur
5. Neurological signs
6. Evidence of limb or abdominal ischemia due to involvement of major aortic branches.
7. Cardiac tamponade or evidence of myocardial infarction if dissection has extended into aortic root.

Suspect diagnosis particularly if patient is known to have hypertension or connective issue disorder.

Investiations.

1. CXR.
2. Trans-oesophageal/trans-thoracic Echo, whichever available.
3. ECG.

4. Cross match blood for transfusion.
5. U & E.
6. Blood complete
7. CT/CT angiography.
8. MRI

IMMEDIATE MANAGEMENT.

1. Control pain with morphine and metoclopromide.
2. Control BP with isoket infusion
3. Beta blockers to control the BP and heart rate. Lebetalol 50 mg over 1 min then 1-2 mg/min or esmolol 50-200 mcg/kg/min.
4. Sodium Nitroprusside 0.5 mcg/kg/min.
5. If dissection involve ascending aorta urgent cardiac surgery, if dissection involve descending aorta then control B.P and pain and involve vascular surgeon if branches of abdominal aorta are involved.
6. Subsequent management is control of BP and pain and referral to surgeon if needed.
7. Check BP, pulse, temp and urine output hrly to monitor treatment.
8. Discharge medically treated patient within pain and BP controlled and arrange follow up with cardiologist.
9. Discharge surgically treated patient when stable and pain, BP controlled.

CARDIAC TEMPONADE

Symptom and signs

1. Dyspnoea
2. Decrease conscious level
3. Right heart failure
4. Hypotension
5. Pulsus paradoxus
6. Raised JVP
7. Raised JVP on inspiration
8. Soft heart sounds
9. Heart rate >80 bpm
10. Oliguria or anuria

Investigations

1. Echocardiography.
2. CXR.
3. ECG
4. U & E.

Life threatening features.

1. Severe symptoms
2. Signs of shock
3. Large effusion on CXR or Echo with right ventricular diastolic collapse on echocardiography.

IMMEDIATE MANAGEMENT.

1. Confirm diagnosis by echo, with immediate aspiration by cardiology team.

2. Start iv normal saline as you aspirate
3. Aspirton can be both diagnositic and therapeutic, send fluid for examination, drain can be left in-situ for many days if effusion large or pyogenic.
4. SUBSEQUENTLY, Identify and treat cause if possible, monitor BP pulse temp. Urine output when in hospital.
5. Discharge when stable, effusion resolved, needle removed.
6. Further follow up depends on causes.

DEEP VEIN THROMBOSIS

Symptoms and signs

1. Swelling in the calf or leg and pain or stiffness in affected limb.
2. Pitting edema.
3. Increased skin temperature
4. Erythema
5. Tenderness
6. Mild fever.

Differential diagnosis.

1. Ruptured baker's cyst.
2. Torn calf muscle.
3. Cellulitis.

Investigations.

1. Doppler US leg, if positive start treatemtn
2. If negative ask for D-Dimmer provided no other causes for raised D-Dimer present
3. If D-dimer netgative re-consider diagnosis
4. If D-Dimer positive but clinical suspicion low don't start anti-cougulants. Repeat Doppler US after 4-7 days. Negative D-dimer rules out possibility of DVT but positive does not confirme diagnosis unless dopller us positive
5. CT-angiography.

MANAGEMENT IMMEDIATE

1. Rest and elevation of leg.
2. Simple analgesia
3. Anti coagulants preferably low molecular weight heparin plus warfarin together. Stop heparin once INR of 2-3 achieved. Continue warfarin for 3 months if no pulmonary embolism, 6 months if pulmonary embolism. Life long if recurs on stopping anti coagulants, surgical intervention if recurs despite on anti-coagulants. Monitor by PT/INR for warfarin and APTT for heparin.

SUBSEQUENTLY

4. Mobilize after 36 hrs if well anti-cougulated and pain, edema, settled.
5. Apply grade 3 compression hose to minimize post-phlebitis syndrome
6. If after 72 hrs no sign of resolution, reconsider diagnosis.

DISCHARGE

1. On warfarin with INR in the range 2-3.
2. Patient sent home on warfarin should be warned about drug interactions.
3. Monitor INR weekly initially then monthly for duration of treatment.

CARDIAC ARREST

RECONITONA ND ASSESSMENT

Signs

1. Unconscious
2. Not breathing for 10 seconds
3. No pulse in carotid or femoral artery for 10 seconds

IMMEDIATE TREATMENT

BASIC LIFE SUPPORT

1. Call for help and for a defibilator.
2. In no evidence of trauma, turn patient on to his back.
3. Open airway by tilting head and lifting chin. Clear airway of foreign boides, bomitus, secretions etc and use appropriate airway adjunct if available.

START CARDIO PULMONARY RESUCITATION (CPR)

1. Perform expired air ventilation:
2. Mouth to mouth breathing: Maintain patient airway (head tilt/chin lift), take a breath, place your lips around patient's, pinch patients nostrils, blow steadily into mouth.
3. Pocket face mask: maintain patient airway (triple airway maneuver/jaw thrust), position pocket facemask, and use both hands to maintain adequate seal. Take a breath, place your lips around one-way valve, and blow steadily into his mouth
4. Supplement oxygen (15 l/min) can be delivered into

pocket face mask simultaneous to mouth to mask technique

5. Deliver 2 effective breaths using if necessary up to 5 attempts
6. To minimize gastric inflation:
   - Each breath to take 5 sec inflation 2 sec, expiration 2-3 sec.
   - Limit inflation volume; adequate tidal volume is indicated by rising of the chest.

START CHEST COMPRESSION

1. Place heel of one hand over lower half of sternum 2 finger breadths up from the xiphisternum and cover with other hand, interlocking fingers so that pressure is not applied over ribs.
2. Lean well over patient. With your arms straight, press down vertically on sternum to depress it by about 4-5 cm
3. Release the pressure, then repeat at a rate of approximately 100 compressions per min
4. Compression and relaxation should be of equal duration

COMBINED VENTILATION AND COMPRESSION:

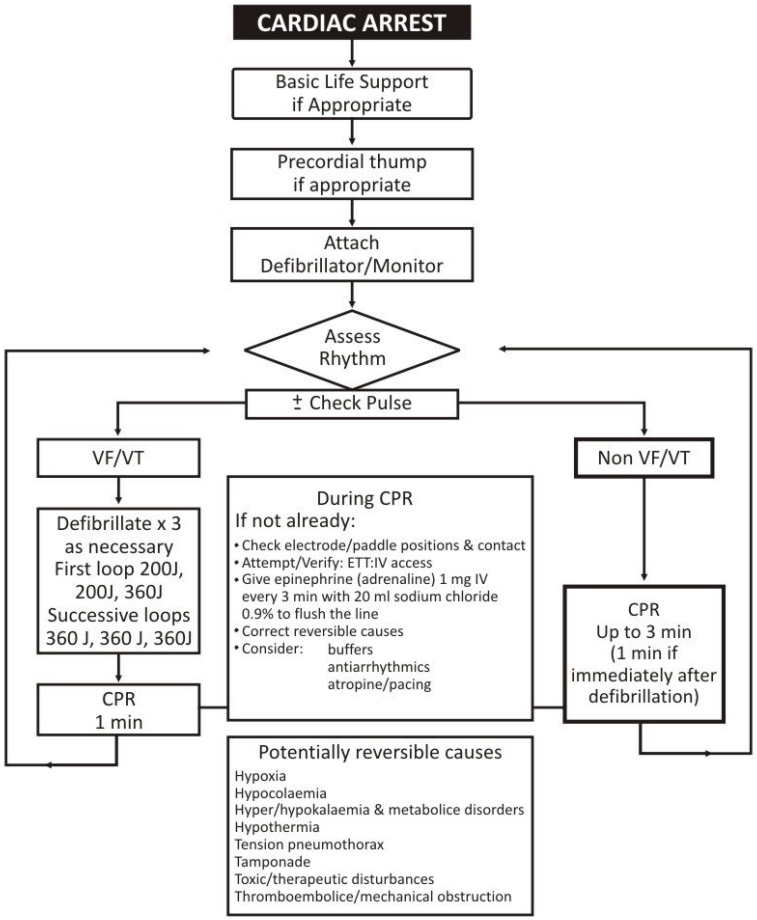
1. Single person BLS provides 15 compressions 2 ventilations
2. If a laryngeal mask airway inserted, attempt to

perform continuous chest compressions (100/min uninterrupted during ventilation 12 breaths per minute. Chest compressions interrupted for defibrillation or pulse check when indicated. If excessive gas leakage results in inadequate ventilation of the patient's lungs, chest compressions will have to be interrupted to allow for ventilation. Use a 15:2 compression: ventilation ration.

3. Patient's trachea intubated. Chest compressions at a rate of 100/min should continue uninterrupted except for defibrillation or pulse check when indicated and ventilation continued at a rate of approximately 12 breaths per minute
4. Two persons BLS: provide 5 compression to 1 ventilation, interrupting compression to ventilate lungs.
5. Continue resuscitation until:
   - Victim shows signs of life in which case reassess airway, breathing, circulation
   - Qualified help arrives
   - You become exhausted
6. Monitor ECG as soon as possible and treat arrhythmias (see cardiac arrhythmias)

THE ALS ALGORITHM FOR THE MANAGEMENT OF CARDIAC ARREST IN ADULTS

Note that each successive step Is based on the assumption that the one before has been unsuccessful.



1. **PRECORDIAL THUMP.** Precordial thump is indicated for cases of witnessed / monitored cardiac arrest.
2. **DEFIBRILLATION.** For defibrillation, the standard anterior-anteriour paddle position is as follows. One paddle to the right of the sternum below the right clavicle and the other around the postion of the cariac apex with the center of the electrode in thi mid axillary line. When using handl held defibrillator paddles always press firmly to ensure good contact with conductive gel pads. During treatment of VF, pulse checks are necessary only if the post shock ECG rhythm is potentially perfusing type.
   WARNING Most defibrillators should not have 'Live' paddles placed on them, Read instructions about dumping charge for defibrillator in use.

AT THE APPROPRIATE STAGE DURING CPR:

1. Check electrode paddle positons and contact
2. Attempt verify endotracheal intubation and iv access
3. Verify adequate BLS and ventilate using highest possible inspired oxygen concentration.
4. Give epinephrine adrenaline 1 mg 0.01 mg/kg every 3 minutes if no rhythm
5. Correct reversible causes such as
   - Hypoxia / hypovolaemaia

   - Hyper / hypokalaemia and metabolic disorders
   - Hypothermia
   - Tension penumothrax
   - Cardiac tamponade
   - Toxic therapeutic disturbances
   - Thromboembolic mechanical obstruction
6. Consider buffers, antiarrhythmics, atropine/ pacing
7. Perform CPR at all times for pulseless patients with the obvious exception of during rhythm analysis and defibrillation shocks. Do not interrupt for more than 30 seconds.
8. Adrenaline increases the efficacy of basic life support but doers not affect the outcome of defibrillation. Intubation, gaining of IV access or the administration of drugs should not be allowed to causes undue delay in the continuation of BLS or giving further shocks
9. if peripheral venous access is used during resuscitation, IV medications should be administered rapidly by bolus injection and followed by a 20 ml bolus of IV fluid and elevation of the extremity.
10. Endotracheal ET Drug administration
11. Epinephrine (Adrenaline) Lignocaine, and atropine can be administered by the ET tube at dose of 2-3

times the recommended IV dose diluted in 10ml of sodium dhloride 0.9% or sterile water

12. To administer, withhold chest compression, spray durg quickly down a long catheter placed within fube, follow with 5 insufflations, and recommence chest compressions
13. The greatest chances of survival result when the interval between the start of VF and the delivery of defibrillation si as briedf as possible. Defibillate VF/ pulse less VT until VF/ pulse less VT is no longer present.
14. VF is so readily treatable that defibrillation should always be tried if there is any doubt that the rhythm is asystole
15. Shock refractory cardiac arrest due to VF or pulse less VT. Amiodarone can be considered as early as before delivery of the fourth shock provided if does not delay delivery of this shock. Amiodarone 300 mg made up to 20 ml with dextrose or from a prefilled syringe may be administered into a large peripheral vein or central venous line. A further dose of 150 mg may be given for recurrent or refractory VT/VF, followed by an infusion of 1mg/ min 6 hrs and then 0.5 mg/min to a maximum daily dose of 2g.
16. Magnesium 8 mmol should be given for refractory VF if there is any suspicion of hypomagnesaemia eg

patients on potassium losing diuretics or long Qt.

Special situations:

1. Atropine will counter any excess vagal tone although it bring no proven benefit in clinical practice. During the treatment of asystole IV atropine 3 mg once daily is recommended.
2. Transcultaneous or transvenous pacing should be considered if electriacal activity p waves or occasional QRS complexes has recently been present
3. Calcium chloride 10% should be considered during the treatment of specific toxic/therapeutic disturbances following discussion with senior team member
4. The buffer sodium bicarbonate 1meq/kg may be considered in cases of suspected or known acidosis. Discuss with senior team member.
5. Pulseless electrical activity. Epinephrine 1mg IV is administered every three minutes. If PEA is associated with a bradycardia <60/min atropine 3 mg IV, or 6mg via the tracheal tube should be given.

SUBSEQUENT MANAGEMENT

The immediate goals of post resuscitation care are:

1. Provide cardio respiratory support to optimize tissue perfusion especially to the brian

2. Trasnport the patient to an appropriately equipped cornonary care unit
3. Attempt to identify the precipationtincauses of the arrest
4. Institute measures such as anti-arrhythmic therapy to prevent recurrence
5. If cardiac arrest follows ventricular tachycardia fibrillation, consider giving lignocaine 100mg IV slowly, followed by IV infusion to prevent further ventricular arrhythmia: 4mg/min for 30 min, then 2mg/min for 2 hr, then 1mg/min for 24 hrs. Reduce concentration further if continued beyond 24hrs.

Establish causes of cardiac arrest and treat underlying diagnoss.

Investigation

1. Blood gases and acid-base
2. U & E, glucose
3. Chest X-Ray
4. Cardiac enzymes
5. Diagnostic ECG

DISCHARGE POLICY

Dependent upon underlying cause

CARDIAC ARRYTHMIAS

RECOGNITIONA ND ASSESSMENT

The mode of presentation dictates the urgency of assessment and treatemtn. Accurate diagnosis is rarely possible without an ECG.

Symptoms.

1. Palpitation.
2. Dyspnoea.
3. Chest pain
4. Dizziness
5. Syncope
6. Cardiac arrest

Signs

1. Heart rate < 60, or > 100/min
2. Hypotension
3. Hypoperfusio
4. JVP elevated
5. Cannon waves or flutter waves in internal jugular vein
6. Variable intensity of first heart sound
7. Signs of heart failure

Investigations

12 lead ECG during attack, unless patient unconscious with no pulse, when resuscitation takes priority (See cardiac arrest.)A single lead rhythm strip is an inferior alternative, but better than no ECG at all.

Differential diagnosis.

No-cardiac causes of syncope (Neurological, metabolic)

IMMEDIATE TREATMENT. The effects of all the following treatments should be monitored by continuous ECG recording.

BRADYCARDIAS.

1. Sinus bradycardia may need no treatment. If symptomatic, give atropine 600micro gram IV, and repead once after 5 min if necessary atropine available as 1mg/vial.
2. Sinus pauses and sino atrial block. If episodes prolonged and symptomatic, consider pacing
3. Sinoatrial disease manifest as tachycardia or bradycardia. Seek advice of cardiology unit.
4. Atrioventiruclar AV conduction block
   - First degree: no treatment necessary
   - Second degree: Mobitz type 1 – rarely requires pacing unless clearly symptomatic. Mobitz type 2-usually requires pacing.
   - Third degree complete requires pacing, even if congenital.
5. Intraventricular conduction block/bundle branch block. Pacing unnecessary except as follows:
   - New appearance of bifascicular block (right bundle branch block and left axis deviation or alternating left and right bundle branch block after myocardial infarction.
   - Bifascicular block with otherwise

unexplained syncope

- Trifascicular block (long PR interval and left bundle branch block, not drug induced.)

TACHYCARDIAS

Clinical significance depends upon site of origin. Accurate diagnosis requires 12 lead ECG (paper speed 25mm/sec, 40 msec = 1 small sqare)

Narrow (< 110 msec) QRS complexes originate from sinus node, atrium or AV junction (See below)

Broad (> 110 msec) QRS complexes Should be considered ventricular in origin unless or until proven otherwise.

If diagnosis in doubt, try carotid massage first, if unsuccessful, give adenosine 3 mg IV over 2 sec. If no respopnce after 1-2 mg IV over 2 sec. Adenosine causes toxicity in paitents taking dipyridamole reduce initial dose to 0.5-1mg. Atrial tachycardias should be revealed, junctional re-entrant tachycardias terminated, and ventricular tachycardias will be unaffected, though retrograde conduction will be blocked. Adenosine should not be given if there're is a history of wheezing.

Verapamil 5-10mg IV over 3-5 min is commonly used in this country. Dose can be repeated in 2o min if no effect. Do not give Verapamil for wise QRS complex tachycardias or if the paitent is on beta blocker. Alternatively metaprolol IV 5mg over 5 min can e given. If can be repeated twice at 5 min interval.

If tachycardia associated with hypotension, shock, or cardiac failure, consider DC cardioversion (or overdrive pacing for selected tachycardias)

If patient with a pathologic tachycardia is haemodynamically stable with no overt heart failure or impaired ventricular function, an anti-arrhythmic drug may be given by slow IV injection provide that full resuscitation facilities are available, preferably on CCU. Use either lignocaine or amiodarone and then only in patients with no adverse signs and no history or cardiac disease.

1. Sinus tachycardia si usually physiological. Identify and treat the cause e.g blood loss, heart failure, thyrotoxicosis, anaemia. If no obious underlying causes, cardiac function adequate, and tachycardia inappropriate and distressing consider starting oral beta blockers (atenolol 50mg daily)
2. Atrial tachycardia arises from atrial myocardium. Amiodarone is the drug of choice. Dose 5mg/kg over 20-120 minutes with ECG monitoring. Maximum daily dose is 1.2 grams.Flecainide, sotalol are preferred drugs but not freely available in local market. Dose of flecainide 1-2mg/kg over 10 min then 0.20 mg/kg/hr. oral dose 100-400 mg/day in 2 divided doses. Sotalol 160-480 my/day in 2 divided doses.
3. Atrial fibrillation/ flutter. If present for less than 24 hrs. aim to restore sinus rhythm immediately using anti arrhythmic drugs or DC cardioversion, unless there is a persistent underlying cause e.g thyrotoxicosis, mitral valve disease, pneumonia. If

unable to cardiovert immediately give heparin by IV infusion 1000 iu/hr and cardiovert next working day. If present for more than 24 hrs give digoxin and / or diltiazem., verapamil or a beta-blockers unless wolff Parkinson white syndrome suspected. Consider anticoagulation with a view to elective cardioversion.

4. Wofl Parkinson white syndrome. May present as atrail fibrillation or flutter. The QRS complexes will be pre excited i.e wide and bizarre and the ventricular response every fast with a tendency to degenerate to ventricular flutter and fibrillation. Never give digoxin, verapamil or adenosine but seek advice of cardiology team with a view to restoring sinus rhythm urgently with DC cardioversion.
5. Junctional re-entry tachycardia usually involves the AV node in the re-entry circuit and is liely to be terminated by aV nodal blockade. Give adenosine as above; or verapamil 5mg IV over 2 min, repeated if necessary at 5-10 min intervals to total 15mg.
    - Ibutide infusion or oral Dofetide are new anti arrhythmic drugs used in SVT.
6. Ventricular tachycardia areises from ventricular myocardium. Haemodynamic consequences are related to the ventricular rate and underlying left ventricular function. Give lignocaine 100mg IV over

2 min, repeated once if necessary at 10 min. If ineffective seek help from cardioilogy unit (if not in cardiology unity already with a view to urgent DC cardioversion under sedation general anesthesia.

7. Ventricular flutter and fibrillation, if sustained, lead to cardiac arrest and must be treated by immediate electrical defibrillation when the patient is unconscious.

SUBSEQUENT MANAGEMENT

General

After any emergency treatment to revert or stabilize a patient's heart rhythm further assessment should include:

1. Accurate identification of arrhythmia a 12 lead ECG during the arrhythmia will give the diagnosis in most cases, sometimes with the addition of specific maneuvers, such as carotid massge / adenosine, or comparison with ECG in sinus rhythm. Electrophysiologic testing may be required where there is doubt.
2. Diagnosis of the cause- ECGs in sinus rhythm, cardiac enzymes, thyroid function tests, chest x-ray.
3. Definition of underlying heart disease echocardiography, cardiac catheterization where appropriate.
4. Identification of precipitaitn contributing factors electrolytes (Ca, Mg,), ECG Monitoring.

5. Provocation testing where necessary e.g. exercise testing, tilt testing, carotid sinus pressure, drug challenge, invasive electrophysiologic testing.

Amiodaron should always be given either in a large peripheral vein or via central venous line. Extra vascular leakage can cause seneve necrosis & at times gangrence.

Specific

Atrial fibrillation/ Flutter.

1. If reversion to sinus rhythm occurs, consider long term prophylactic therapy with propafenone, flecainide, sotalol subject to availability or standard beta blocker in some cases eg. Hypertensive disease. Pacing may be necessary for prevention of atrial fibrillation in the bradycardia/ tachycardia form of sino atrial disease.
2. Avoid comninations of anti-arrhythmic drugs (Including beta-blockers, diltiazem and verapamil) unless on specific cariological advice.
3. Patients with Wold Parkinson white syndrome should undergo electrophysiological assessment with a view to ablation of accessory pathway.
4. IF DC carioversion is unsuccessful, consider log term control of the ventricular response with digoxin and or diltiazem, verapamil or a beta blocker. Choice depends on clinical picture, eg hypertension, heart failure , angina or thyrotoxicosis.

5. Consider oral anticoagulation in patients with sustained or paroxysmal atrail fibrillation in following categories':
   - Awaiting elective cardioversion.
   - Co-existent valvular heart disease or valve prosthesis of any age
   - Prvious stroke, TIA or systemic thromboembolism
   - Cardiac failure of LV dysfunction
   - Controlled ypertension
   - Diabetes mellitus
   - Thyrotoxic
   - Aged 65 years or over
6. Consider aspirin in all other patients and if oral anticoagulation contraindicated.

Ventricular flutter/ Fibrillation

If the arrhythmia fails to terminate or recurs, consider and deal with possible trigger factors:

1. Electrolyte abnormalities hypokalaemia, hypocalcaemia, hypomagnesaemia
2. Anti arrhythmic or anti psychotropic drug toxicity
3. Underlying relative bradycardia temporary pacing will be necessary
4. Acute myocardial infarction- urgent revascularization by angioplasty / stent may be appropriate

5. For recurrent episodes try lignocaine with ECG monitoring by IV infusion 4mg/min for 30 min, then 2mg/min for 2 hr, then 1mg/min for 24 hrs. Reduce concentration further if continued beyond 24 hrs.
6. Patients with ventricular tachycardia or ventricular flutter/ fibrillation occurring 48 hrs after acute myocardial infarction (AMI) or with no obious reversible factors and low EF should be considered for implantation of an ICD (Implantable cardiac defibrillator)

DISCHARGE POLICY

1. Patients with recurrent arrhythmias that require prophylactic anti arrhythmic treatment should be followed up in a cardiology clinic.
2. Appropriate arrangements should be made for following up patients with atrial fibrillation flutter who are anticoagulated.

ACUTE UPPER GASTRO-INTESTINAL HAEMORRHAGE

RECOGNITIONA ND ASSESSMENT

Symptoms

1. Coffee-ground vomit
2. Haematemesis (Bright red blood in vomit)
3. Melaena (digested blood in stool). Rarely, dark altered blood per rectum may indicated a severe upper gastro-intestinal hemorrhage with no other features to suggest upper gastro intestinal pathology.

Bright red rectal bleeding in the absence of hypotension is likely to arise from the lower gastro intestinal tract.

Previous history

1. **Enquire aboutu**
   - Peptic ulcer.
   - Previous bleeds
   - Liver disease
   - Family history of bleeding
   - Ulcerogenic medication/ anticoagulants
   - Alcohol
   - Weight loss

Signs

1. Evidence of severe bleeding-defined as the presence of shock tachycardia, hypotension, clammy skin or orthostatic hypotension in patient who is not

clinically shocked.

2. Evidence of anaemia
3. Features of precipitating disease, jaundice, stigmata of liver disease
4. Features of bleeding disorder (petechiae)
5. Buccal or facial telangiectasis
6. Corrosive at the mouth.

Investigations

Non-severe bleeding

1. Blood complete send with 10ml clotted blood for cross match (non-urgent)
2. All other investigations can wait until normal working laboratory hrs.

Severe Bleeding

1. Blood complete
2. U & E
3. INR
4. Cross match (4 units) notify blood bank of clinical problem and degree of urgency.

IMMEDIATE TREATMETN

Non severe bleeding.

(patient < 60, previous fit, No hypovolaemai, haemoglobin > 10g/dl.)

1. Baseline observation.
2. Oral nutrition.

3. Oesophago gastro edudenoscopy next routine list
4. Oral tranexamic acid 1gm 8 hrly contraindication thromboembolic disease
5. Oral proton pump inhibitors e.g omeprazol, lansoprazole, pantoprazole , essomeprazole
6. In case of suspected case of variceal bleeding, injection terlipressin 2 mg IV state, followed by 1 mg IV QID fro 2 days or octerotide injection, 25-50 mcg by IV infusion for 2 days.

Severe bleeding.

1. Consider ICU.
2. Establish intravenous access and once sample obtained for cross matching, restore blood volume rapidly with plasma expanders (Haemacce, Gelofusin)
3. Give blood as soon as available, after flushing infusion line with saline/dextrose.
4. Consider CVP line
5. Discuss surgical involvement with gastroenterologist
6. If possible transfer to ICU
7. Mangy patients with severe bleding have oesophageal varices. These patients may be identified by clinical history, previous hospital notes or by obiosu clinical signs jaundice, ascites, spider naevi, etc. See oesophageal varices for management.

8. Oral intake hips only until endoscopy
9. Give injection tranexamic acid 1gm 8 hrly
10. Omerprazole infusion 12 hrly
11. In suspected cases of variceal bleeding: injection terlipressin 2mg IV stat, followed by 1 mg IV 6 hrly for 2 days or octreotide infusion 50mcg/h by IV infusion for 2 days
12. In case of suspected case of variceal bleeding, antibiotics must be given even in the absence of evidence of any injection: inj.ceftriaxone 2g IV daily or infusion Ciprofloxacin 200mg 12 hrly for 2-7 days, may be changed to oral if patient becomes stable
13. In case of suspected case of variceal bleeding, protein restriction, Domperidone and lactulose should be given to prevent development of hepatic encephalopathy.

SUBSEQUET MANAGEMENT.

Young patient with minor bleeding and obvious precipitating cause, eg vomiting following excess alcohol, can be managed by a general medical team.

Non-severe bleeding.

1. Continue observations until outcome of upper gastro intestinal endoscopy known. Subsequent management will depend on the condition identified eg oral H2 receptor antagonist or regimen for eradicatin of H.Pylori taking into account any

advice appearing on the endoscopy report.

Preferred eradication regimen is

2. PPi 12 hrly
3. Plus 2 antibiotics with include:
   - Clarithromycin 500mg 12 hrly.
   - Amoxicllin 1g 12 hrly
   - Metronidazole 400mg 8 hrly
   - Duration of therapy: 7-14 days.
   - As resistance is increasing, avoid clarithromycin or metronidazole if used in past year for others infections.
   - Oxytetracycline 500mg 6 hrly may be substituted in treatment failure.
4. Treatment of active peptic ulcer should also include a week course of a proton pump inhibitor or H2 blocker
5. If neoplasm is identified consider surgical referral

Patients who rebleed.

1. Evidence of rebleeding in an otherwise stable patient justifies a requet for urgent endoscopy if not previousl performed if the patient is potentially referable for surgery.
2. Even when rebleeding occurs, there is no indication for ulcer healing treatment until a firm diagnosis has been established endoscopically.

3. Rebleeding after admission should prompt a gastroenterology and surgical opinion if not previously obtained.

Indication for surgical intervention:

1. If patient aged > 60 yr requires more than 4 units of whole blood to restore or maintain blood volume over 24 hrs, or continuous to bleed, or rebleeds, operation is generally advised.
2. If patient aged < 60 yr requires more than 8 units of whole blood to restorer or maintain blood volume over 24 hrs, or more than 12 units over 48 hrs, or shows evidence of 2 episodes of rebleeding, operation is generally advised.

Patients with oesophaegeal varices.

1. **IV terlipressin** 2mg STAT and then 1 mg 6 hrly for 24 hrs, or 25 – 50 mcg by IV infusion per hour for 2 days followed by sclerotherapy or banding.
2. **Ciprofloxacin.** 500mg 12 hrly orally, contact senior doctor on call.

MONITORING TREATMENT

All patients:

1. 4 Hrly pulse and blood pressure.
2. Observe vomit for blood content
3. Repeat haemoglobin dialy until it is stable (not falling)

DISCHARGE POLICY

1. This will depend on age and cause of gastro-intestinal haemorrhage but most patients will be discharged within 5 days.
2. Young patients with non-severe bleeding and ulcer-related disease may usually be discharged promptly after endoscopy.
3. Older patients with similar pathology may be discharged when their condition is stable.
4. Start ferrous sulphate 200mg orally 8 hrly if Hb has fallen below 10g/dl.
5. Patients with neoplasia may need further investigation and treatment.
6. Patients with severe bleeding should be followed up as outpatients
7. Patients with H.pylori positive ulceration must be followed up in outpatients until H.Pylori has proven to be eradicated.
8. No follow up needed for patients with non severe bleeding and or transient pathology eg Mallory weiss tear acute erosion.

ACUTE LIVER FAILURE WITH ENCEPHALOPATHY.

RECOGNITION AND ASSESSMENT

Liver failure should be considered in all patients with abnormal liver function tests where deterioration in conscious level has been identified.

Symptoms/Signs

1. Jaundice
2. Malaise, nausea, vomiting
3. Altered conscious level (hepatic encephalopathy, see below)
4. Evidence of infection (e.g fever)
5. Evidence of coagulopathy eg bruising, petechiae
6. Hepatic Flap

Grade of encephalopathy.

Grade 1: Confused. Altered mood or behavior.
Grade 2: Drowsy with inappropriate behavior.
Grade 3: Stupor with inarticulate speech. Arousable and can obey simple commands.
Grade 4: Coma, Unarousable.

Investigations.

1. Blood complete
2. INR
3. Urea and creatinine
4. Serum electrolytes
5. Blood glucose
6. LFT
7. Toxicology screen e.g paracetamol

8. Hepatitis serology
9. Blood and urine cultures
10. Culture and white cell content of ascetic fluid
11. Chest x-ray
12. Arterial blood gases on air

Look for evidence of multiple organ failure.

1. Oliguria/ anuria
2. Hypotension (mean arterial pressure < 80mmHg) despite initial fluid administration. Inotropes dependency
3. Impaired gas transfer: hypoxaemai PaO2 < 75mmHg PaO2 < 10kPa despite 40% oxygen.
4. Metabolic acidosis
5. Radiological pulmonary shadowing/ edema
6. Patients looks severely ill/ exhausted / obtuned
7. Spontaneous bruising and or mucosal bleeding

IMMEDIATE TREATMENT

1. Consider admission to ICU
2. Inform a senior member of the on call team registrar or above.
3. In consultation, consider contacting a gastroenterologist for urgent assistance.

Indications for transfer to ICU

1. Multiple organ failure in patients with acute liver failure who exhibit Grade 3 or Grade 4

encephalopathy. Contact on duty anaesthetist.
2. Cerebral edema.

Uncomplicated liver failure.

1. Avoid:
   - Intravenous or oral sodium supplementation.
   - Unnecessary intravenous or invasive monitoring risk of infection
   - Proton Pump inhibitors unless positive indication.
2. Establish IV access
3. Correct hypoxia
4. Correct hypokalaemia and hypoglycaemai, and maintain blood glucose with continuous infusion of 5%-10% glucose. Correct hypotension with crystalloid or colloid infusions.
5. Give Vitamin k, 10mg IV daily by slow IV infusion in 55ml glucose 5%. Do not give fresh frozen plasma unless there is clinical evidence of bleeding.
6. Give 30ml Lactulose orally or via NG tube initially 2-hrly till purgation occurs, and then three tiems daily or phosphate enema. Adjust dosage to produce 2-3 soft stools daily. It is not necessary to produce diarrhea.
7. Give broad spectrum prophylactic Antbiotics:
   - Cirprofloxacin 200mg IV 12 hrly or inj

Ceftriaxone 2gm IV daily

- Convert to oral after 48 hrs if possible, discontinue after 5 days if culture negative.

8. Treat suspected infection with
   - Ciprofloxacin 200mg IV 12 hrly or inj. Ceftriaxone 2g daily state and then 1g 12 hrly PLUS.
   - Metronidazole infusion 100ml 8 hrly for at least 7-10 days.
   - Modify therapy on basis of cultures
9. Diet. Aim for 30 kcal/kg with hight protein and low salt.
10. Avoid sedatves benzodiazepines, phenothiazines, opioids

SUBSEQUENT MANAGEMENT

1. Give prophylactic antibiotics Ciprofloxacin 250mg orally 12 hrly.
2. In patients who deteriorate from Grade 1 or Grade 2 encephalopathy to grade 3 or Grade 4 encephalopathhy, consider referall to ICU for more invasive monitoring and treatemtent.

Additional factors to consider

1. **Varices**. Evidence of upper GI haemorrhage should prompt a referral for endoscopy and possible variceal sclerotherapy or band ligation.

2. **Ascites**. This need not be treated urgently unless it is causing symptoms. If it is, give spironolactone 100mg orally daily, increasing by 100mg every 2-3 days if necessary max 400mg to achive weight reduction of 0.5-1kg/day. Frusemide orally max 80mg 12 hrly may be added if spironolactone not effective. If drainage is though necessary, replace fluid volume drained with salt free albumin 6-8g of albumin for each litere drained. Haemaccel may be used n place of albuminin a dose of 1000ml for 2-3 liter ascities drained. Stop diurectics and restrict fluid and salf if patient develops hyponatremai.
3. **Spontaneouus bacterial peritonitis.** This carries a high mortality and must be excluded if there is deterioration or evidence of sepsis. Arrange urgent ascetic tap for microscopy, C/S.
4. **Renal failure.** If greater than 50% reise in creatine in 24 hrs and oliguria less than 400ml in 24 hours, discontinue diuretics and reanal toxic drugs. Ensure adequate fluid replacement with CVP monitoring inform or nephrology unit.
5. **Crebral edema.** Distub as little as possible. Give IV infusion of 20% **manitol** 0.25 – 0.5g/kg over 15-30 min and repeat every 4-8 hr if necessary, monitoring urine output hrly and viatla signs. Consider transfer to ICU.

Complications

1. Coagulopathy.
2. Electrolytes distubbance
3. Gastrointestinal haemorrhage
4. Hypoglycaema
5. Hypotension
6. Intercurrent infection
7. Oliguric renal failure
8. Respiratory failure

MONITORING TREATEMTN

In-day:

1. Pulse oximetry (Continuous)
2. Urine output hrly
3. Blood glucose 2 hrly
4. Blood pressure. 4 hrly
5. Pulse 4 hrly
6. Temperature 4 hrly
7. Conscious level 4 hrly
8. Fundoscopy seeking papiloedema twice daily.

Daily:

1. Blood complete, INR
2. U & E
3. LFt
4. Weight and fluid balance

Alternative days.

1. Culture-blood, urine, ascites.

DISCHARGE POLICY

1. Arrange outpatient follow up with gastroenterology unit.

ACUTE ULCERTIVE COLITIS AND CROHN'S DISEASE.

RECOGNITION AND ASSESSMENT

Symptoms/Signs.

1. Severe diarrhea, tenesmus
2. Abdominal pain
3. Anorexia, Weight loss
4. Malaise
5. Variable amount of blood in stool
6. Dehydration
7. Tachycardia
8. Fever
9. Anaemia

Life-threatening features:

a. **Sepsis syndrome/septic shock**
b. **Toxic dilation of colon**
c. **Free perforation of colon**
d. **Profound electrolyte disturbance**
e. **Massive haemorrhage**
f. **Obvious weight loss**
g. **Secondary multi organ failure.**

Investigations

1. Blood complete
2. Biochemical screen, blood glucose
3. Abdominal X-ray
4. Erect chest x-ray looking for gas under the

diaphragm

5. Stool culture (Salmonella. Shigella, campylobacter), diffcile toxin
6. Cross match
7. Arterial blood gases

Differential diagnosis.

1. Bacterial and amoebic colitis history of travel
2. Pseudomembranous colitis-history of antibiotic use
3. Deverticular disease
4. Ischaemic colitis
5. Bowel cancer
6. Abdominal lymphoma
7. Radiation colitis
8. Ileocaecal TB

IMMEDIATE TREATMENT

1. Establish IV line. Correct dehydration/ electrolyte disturbance
2. Give blood transfusion if Hb< 80g/dl 4 units plus an extra unit for each g/dl below
3. Hydrocortisone 200mg IV 8 hrly
4. Metronidazole 500mg IV 8 hrly
5. In patients with life threatening features inform duty surgical team
6. Barrier Nurse – inflammatory bowel disease can be indistinguishable from infective diarrhea at first.

DO NOT GIVE

Anti-diarrhoeal drugs in acute phase they increase the risk of toxic dilation.

DO NOT PERFORM. Barium enema or colonscopy in acute phase there is high risk perforamtion of colon.

SUBSEQUENT MANAGEMENT

1. Once infective element has been excluded relax barrier nurshing restrictions.

If improving.

1. Substitute metronidazole 400mg orally 8 hrly and prednisolone (not enteric coated 60mg orally dialy in place of hydrocortisone
2. Start restricted oral feeding. Seek dietetic opinion
3. Give mesalazine 800mg orally 8 hrly
4. For distal disease consider prednisolone enema one daily
5. If extent and severity of inflammation not apparent from supine plain abdominal x-ray, plan colonoscopy of barium enema in convalescent phase n consultation with consultant gastroenterologist.

If not improving

1. Consider surgery if not improvement has been achieved by day 5.

MONITORING TREATMENT

Two hrly

1. Temperature
2. Pulse
3. Blood pressure
4. Respiration

Twice daily.

1. Abdominal examination look fro local peritonism and check bowel sounds
2. Measure abdominal girth

Daily

1. Blood complete, U & E, stool culture
2. Abdominal x-ray- look for free abdominal gas or colonic dilation > 6cm.
3. Count stools and inspect for blood.

Alternative days.

1. Erect chest x-ray: looking for gas under the diaphragm.

DISCHARGE POLICY

1. Plan home treatment regimen
    a. **Prednisolone-** reduce by 5mg weekly
    b. **Prednisolone** enema once daily.
    c. **Mesalazine** 800mg orally 8 hrly
    d. Nutritional support, as advised by dietician
2. Arrange outpatient colonoscopy or barium enema, if

not already performed, in consultation with gastroenterologist

3. Arrange follow-up in Gastroenterology outpatient clinic after 4 weeks.

ACUTE RENAL FAILURE

RECOGNITONA AND ASSESSMETN

Acute renal failure is a rapid decline in renal excretory functions, acid/base balance and removal of solutes and water, occurring over hrs to days.

Recognition.

1. Oliguria < 30ml urine /hr or anuria and or
2. Rising plasma creatinine: daily rise> 1.5mg/dl or in the presence of infection the daily rise in creatinine may be > 3mg/dl.

Causes

1. **Pre renal**
   - Volume depletion
   - Hypotension, pump failure
   - Sepsis
2. **Renal**
   - Established acute tubular necrosis
   - Glomerulonephritis / vasculitis
   - Nephrotoxins
3. **Obstructive: Hospital acquired renal failure is often multifactorial, with contributions from hypotension, sepsis and drugs.**

Assessment

1. Take a detailed clinical history; examine the patient with particular reference to features associated with

volume depletion, sepsis or multisystem disorder.

2. Record drug history
3. Obtain previous record for evidence of pre-existing renal dysfunction.
4. Look for signs of urinary tract obstruction, i.e. palpable bladder

Look for evidence of multiple organ failure.

1. Hypotension mean arterial pressure < 80mmHg, despite initial fluid administration. Inotrope dependency
2. Impaired gas transfer : hypoxemia PaO2 < 75mmHg < 10kPa despite 40% oxygen
3. Metabolic acidosis-compensated as well as non-compensated
4. Pulmonary shadowing / edema on chest x-ray
5. Patient looks severely ill/ exhausted / obtunded

Patient with developing or established multiple organ failure should be identified early and referred to intensive care for for further investigations and management.

Investigations

1. Urgent urine R/E, presence of haematuria or proteinuria may indicate acute glomerulonephritis/ vasculitis etc
2. Blood complete
3. Biochemical screen
4. Blood gases to assess acidosis, hypoxia.

5. Urgent renal ultrasound to assess renal size / exclude obstruction.
6. Chest x-ray looking for signs of fluid overload, infection.
7. Plasma proteins and urine electrophoresis
8. Immunology scree: (ANA/ANCA/C3/C4/ Anti-GBM) required only in patients with single organ failure and with active urinary sediment in urine.

Referral to Nephrology Unit.

1. Contact the nephrology unit
2. Discuss with renal unit any patient with:
   - Creatinine > 2.5mg/dl
   - ARF without any obvious cause (eg volume depletion, sepsis)
   - ARF with haematuria/proteinuria
   - ARF in the setting of multisystem disorder

IMMEDIATE TREATMETN.

1. Accurate charting of fluid input and urine output (urinary catheter may be required)
2. If dehydrated, correct with IV crystalloid. CVP line if necessary to maintain pressure 10-14cm H20.
3. Once rehydrated, continue IV crystalloid to matchurine output + 30ml/hr.
4. If hypotensive , despite rehydration consider colloid gelofusin, blood and inotropes.

NOTE: **AVOID DOPAMINE**

5. If fluid overlaod, give frusemide 120-200mgIV over 1 hr. If no response, contact nephrology team urgently.
6. Keep strict intake output chart and observed aily chemistry to assess changes in reanl functions.
7. Discountineu or avoid nephrotoxic drugs eg NSAIDs

Patient whose renal function continues to decline even if creatine < 2.5mg/dl despite initial resuscitationshould be referred to nephrologist within 48hrs of diagnosing ARF.

SUBSEQUENT MANAGEMENT

Often undertaken by nephrology unit.

MONITORING TREATMENT.

1. Daily weight and fluid balance
2. Daily biochemical screen
3. Monitoring of underlying cause

DISCHARGE POLLICY

Arrange outpatient review with nephrologist if renal function remains abnormal despite treatment.

ACCELERATED (MALIGNATN) HYPERTENSION

RECOGNITION AND ASSESSMENT.

Accelerated hypertension is a specific form of severe hypertension diastolic pressure usually > 120mmHg with evidence of arteriolar damage, evident from retinopathy Grade iii/iv. It may occur in patients with essential or secondary hypertension. Of greatest importance are the absolute height of the blood pressure and the rate of its rise.

Symptoms

1. Blurring of vision, mental impairment
2. Headache
3. Dyspnoea (left ventricular failure)

Signs

1. Haematuria (usually microscopic)
2. Cotton wool spots and hemorrhages with or without papiloedema on fundoscopy. The fundi must be properly examined in patients suspected of having accelerated hypertension . Other target organ damage includes strokes, encephalopathy, left ventricular failure/ enlargement, and renal failure with red cell, cast and protein in the urine.

IMMEDIATE INVESTIGATIONS

1. Blood complete, U & E, creatinine, electrolytes.
2. Urine microscopy
3. ECG, Chest X-Ray

IMMEDIATE TREATEMTN

1. Diastolic BP > 120mmHg and Grade iii or grade iv retinopathy requires urgent assessment, but are not in themselves indications for iv treatment. Most patients can be treated safely with an oral hyptensive drug. Sustained high BP alters cerebral auto regulation; sudden reduction of blood pressure will reduce cerebral perfusion and can be dangerous.
2. **Bisoprolol** 10mg per day with **Nifidipine** long acting 30 mg once or twice a day is sufficient in most cases of severe hypertension, some may need the addition of lisinopril 20mg per day or perindopril 8mg per day or candesrtan 16-32 mg/day or captopril 25mg every 6-8 hrly. Avoid beta blockers if contraindicated. Do Not use sublingual nifidipine.

Note: in the fist instance diastolic BP should not be reduced below 110-115mmHg.

3. Parenteral therapy is indicated only if accelerated hypertension is accompanied by one or more of the following.
    - Hypertensive encephalopathy.
    - Aortic dissection
    - Intracranial hemorrhage
    - Phaeochromocytoma crisis
    - Acute pulmonary edema

- Acute renal insufficiency
- Unstable angina

If parenteral therapy is indicated

4. If there is any doubt about ther need for treatment, seek advice from a nephrology team.
5. If there is any doubt about the need for treatment, seek advice from a nephrology team.

SUBSEQUENT MANAGEMENT

IF IMPROVVING:

1. In patients treated with IV infusion, start oral treatment, before IV agents is withdrawn.
2. Continue maintenance oral treatment with atenolol 100mg/day, Bisoprolol 5-10mg/day, or nifedipine LA 30 mg/day, captopril 6.25-50mg every 6-8 hrly.
3. Add a diuretic after 2-3 days if necessary. Assess renal function in more detail clearance, ultrasound scan.
4. Aim to reduce BP to 160/90mmHg or less over 7-10 day.
5. If not improving, seek advice from nephrology unit.

MONITORING TREATMENT

1. After oral dose, measure BP every 30 min
2. During parenteral therapy, measure BP every 15min continuous intra-arterrial BP monitoring is ideal, if available.

3. On maintenance therapy, measure BP 4 hrly
4. Monitor urine output and plasma urea, creatinine and electrolytes daily.

DISCHARGE POLICY

1. Address other risk factors for cardiovascular disease smoking, hypercholestrolaemia, obesity and advise accordingly.
2. Discharge home when BP < 160/90mmHg and condition is stable

STATUS EPILEPTICUS AND FREQUENT SEIZURES

RECOGNITION AND ASSESSMENT:

Symptoms and Signs

Status epilepticus is defined as a state of seizure activity such that between seizures there is no return to consciousness lasting longer than 30 minutes.

Ask about:

2. Previus diagnosis of epilepsy.
3. Previous history of status epilepticus.
4. Recents withdrawal of anti-epileptic drugs (AED) / missed medication.
5. Vomiting / diarrhea.
6. ing longer than 30 minutes.

Investigations:

1. Capillary blood glucose using glucose **stix.**
2. Venous blood glucose, calcium sodium.
3. Serum anticonvulsant drug concentration if patient has a history of seizures and is taking carbamazepine or phenytoin.
4. CT scan if new onset epilepsy, to exclude space occupying lesion, basal ganglia calcification (hypoparathyroidism)

Differential Diagnosis:

Non-epileptic attack disorders (pseudoseizures)

Important Underlying Causes:

1. **Infection:** Meningitis, Encephalitis, Abscess.
2. **Acute head injury.**
3. Cerebral tumours.
4. Metabolic disorders (renal failure, hypoglycaemia, and hypocalcaemia)
5. Druge over dosage (tricylics, phenothiazines, theophylline, isoniazid, cocaine)
6. Acute cerebral infarction
7. Alcohol intoxication / withdrawal
8. Anoxic encephalopathy
9. Non-epileptic attack
10. Hypoparathyroidism
11. Eclampsia

IMMEDIATE TREATMENT

1. Do not attempt to put anything into the mouth during a seizure even if the tongue is injured.
2. Intubation, if necessary, requires special care.
3. Avoid rolling the patient during a seizure unless absolutely necessary as this can caouse injury to shoulder/hip joints.
4. Generalized tonic-clonic status is potentially life-threatening and should be treated without delay.
5. Consider admission to icu.
6. Once seizure activity has cased, place patient in the recovery position.
7. Protect airway and support respiration if necessary,

If there is any period of relaxation try carefully to insert an airway.

8. **Oxygen** (high flow mack) 10/min.
9. **Diazepam** 10-20mg IV at 5 mg/min, repeated if necessary after a further 10 min and again after a further 20 min OR **lorazepam** 4 mg IV \9diluted 1:1 with sodium chloride 0.9%) as a single bolus injection over 2 min into a large forearm vein. Watch carefully for evidence of respiratory depression by monitoring oxygen saturation.
10. If seizures continue after 20 min, give **phenytoin** with cardiac monitoring.
11. Contact on-call neurology team, if not already informed.
12. Consider secondary metabolic factors (hypoglycaemia, electrolyte imbalance, lactic acidosis, dehydration, and hyperpyrexia)
13. Check blood gases.
14. If, at any stage, respiratory depression or cardiac disorder is apparent, or pH<7, contact ICU.
15. If satisfactory control still not established after 60 min, and neurology junior staff are in attendance, contact registrar/consultant for advice and to arrange transfer to ICU.
16. If patient transferred to ICU arrange EEG as soon as possible after intubation. This may have to be

repeated at regular intervals (eg 24 hrly) during sedation.

Reasons for failure to respond:

1. Incorrect diagnosis.
2. Underlying cause, eg metabolic abnormalities, not recognized and treated.
3. Delay intubation and anaesthesia.
4. Inappropriate use of drugs/dosage.
5. Delay in initiating maintenance anticonvulsant therapy.

SUBSEQUENT MANAGEMENT

If Improving:

Start oral anti-epileptic treatment

In Not Improving:

Reconsider underlying causes.

DISCHARGE POLICY

1. Discharge when patient has been seizure-free for 48 hr, antiepileptic treatment is established, and is fit to leave hospital.
2. EEG and CT scan as outpatient (new cases)
3. Review existing follow-up arrangements for known epileptics, refer new cases to a consultant neurologis.
4. All patients presenting with status epilepticus should have maintenance therapy reviwed within 4 weeks of discharge.

STROKE

RECOGNITION AND ASSESSMENT

Definition

Stroke is:

- A neurological deficit of sudden onset.
- With focal rather than global dysfuntion.
- With symptoms lasting more than 24 hrs or resulting in death before 24 hrs.
- In which, after adequate investigation, syptoms are presumed to be of a non-traumatic vascular origin.

Minor srtoke (reversible ischaemic neurological deficit, RIND) implies recovery without significant and intraparenchymal hemorrhage, which cannot be differentiated clinically but only by neuroimaging (CT of MRI)

Transient ischaemic attack (TIA) is a stroke-like event that resolves within 24 hrs, most resolve within 30 min.

Crescendo TIAs are TIAs of increasing frequency and duration, several episodes occurring within a few days.

Differential Diagnosis:

1. Subarachnoid hemorrhage, extramural subdural hemorrhage.
2. Space-occupying lesion.
3. Arterial dissection (especially with whiplash injury, neck trauma).
4. Meningitis/encephalitis (HIV).
5. Seizures.
6. Hypertensive encephalopathy (diastolic BP> 120

mm Hg, depressed consciousness, papilloedema)
7. Metabolic e.g. hypoglycemia, hyponatraemia.
8. Hepatic encephalopathy
9. Toxic, e.g. overdose
10. Anoxic encephalopathy eg shock, arrhythmia
11. Trauma
12. The distinction between cerebral infarction, embolism, and hemorrhage is not urgent if the patient is conscious and stable, unless the patient is already anticoagulated, has a coagulation disorder or anticoagulation is being urgently considered.

Urgent investigations:

1. Glucose, U & E, Blood complete, INR
2. Save serum for lipids
3. Chest x-ray
4. ECG
5. CT of MR if:
   - Patient anticoagulated
   - Bleeding disorder (platelets <100,000 INR> 1.2)
   - Head injury
   - Neck stiffness / photophobia
   - Crescendo TIAs
   - The diagnosis is in doubt because of unusual features
   - Coma or deteriorating stroke (unless surgery contraindicated)

IMMEDIATE TREATMENT

1. Position to minimize risk of pressure sores and turn 2 hrly. Consider a pressure relieving mattressin obese or frail hemi paretic patients.
2. If consciousnesss impaired of swallowing doubtrul, nil by mouth (give fluid by IV route), consider passing N/G tube.
3. Give **normal saline** (**not** glucose) IV for the first 24 hrs for all patients who are nil by mouth, dehydrated or at risk of dehydration, thereafter the standard regimens for maintaining hydration can be followed.
4. TED stockings for all non-ambulant patients unless contra-indicated.
5. Catheterization (condom) for incontinence in conscious patients, and Foley's catheter if in retension.
6. Antibiotics for suspected infection (temperature >99 F)
7. Treat pyrexia (temperature >99 F) with paracetamol 1 g orally 6 hrly
8. If blood glucose >200 mgl/l give insulin to maintain it below this level
9. Correct hypotension
10. **Do no** lower BP unless>220/120 mm Hg **and** there is other evidence of hypertensive encephalopathy

[**see Accelerated (Malignant) Hypertension**]

11. Reverse anticoagulation urgently, aiming for an INR of 1.0, in warfarinised patients with intracerebral hemorrhage. Consider fresh frozen plasma or factor concentrate to correct INR quickly
12. Ask for senior/consultant advice about:
    - Intracerebral hemorrhage in anticoagulated patients.
    - Crescendo TIAs whose Ct or MR reveals no hemorrhage
    - In patients where an unusual cause for stroke is suspected
    - Cerebellar haematoma in patients with impaired Consciousness (neurosurgical team)
    - Hydrocephalus (neurosurgical team)

SUBSEQUENT MANAGEMENT

1. Sit up as tolerated (bed/chair) as soon as possible
2. Mobilize conscious patients from day 1.
3. If no hemorrhage on CT, give **aspirin** 300 mg orally immediatedly and then 150 mg daily unless contra-indicated.
4. **Avoid sedatives**

Further Investigations

1. ESR
2. Fasting glucose if random glucose >130 mg l/l

3. Lipid status in patients <75 year (<38 hr after stroke or after 6 weeks)
4. Serological tests for syphilis, TPHA and VDRL.
5. CT scan (unless moribund)
6. Carotid ultrasound in patients with a carotid territory ischaemic event, good recovery (TIAs or minor infarct) and a surgical candidate.
7. Echocardiography: patients <50 Years, consider also in patients with TIA, mumurs and/or history of rheumatic fever
8. In patiens <50 years screen for arthritis (CRP, ANA, ANCA, Rh Factor) and thrombotic disorders (coagulation screen, lupus anticoagulant, protein C, protein S, antithrombin III)
9. Young patients with intracerebral bleed may have operable vascular abnormality (request neurosurgical assessment)

Fluid and nutrition management:

1. Assess swallowing at bedside Check that patient is:
    - Alert and cooperative
    - Able to sit up for feeding
    - Able to cough on demand
    - Not drooling excessively
2. Sit patient up, listen to voice and give 5 ml of water on a spoon

- Watch and feel swallow with fingers on larynx.

Observe for 2 min, looking for:

- Choking or impaired breathing
- Delayed swallow
- Cough
- Change of voice

3. If 5 ml swallowed without difficulty give 50 ml of water before giving soft diet.
4. If there is any doubt about swallowing, prescribe'nil by mout, give fluid (2 Lit/24hrs) IV/SX and ask speech therapist to assess swallowing.
5. If nasogastric tube not tolerated or still required after 2 weeks, consider percutaneous endoscopic gastrostomy (PEG)
6. Patients having IV fluids only will require nutritional support within 5 days. Involve nutrition team if available.

Complications:

1. Chest infection after starting oral fluids.
   - Reassess swallowing, treat as aspiration pneumonia
2. Pressure sores
   - Prevent / treat
3. DVT/PE
   - Prevention: full length TED stockings until

patient is fully mobile.

- Treatment, conventional after CT head scan excludes haemorrhage.

4. Depression
   - High suspicion but consider emotional incontinence, lability, dementia, communication difficulty.
5. Seizures
   - Treat conventionally
6. Shoulder pain
   - Prevent shoulder pain by always supporting the weight of the area and by not pulling on the affected arm.
   - Maintain correct position and adequate support, consultant physiotherapis, consider paracetamol
   - If pain persists consider the addition of NSAIDs, or intraarticular steroids

Causes of Deterioration

1. Complications (see above)
2. Coincident medical condition (eg hypoxia, hypoglycemia, hyperglycemia, pyrexia, infection, heart failure, fluid/electrolyte disturbance)
3. Brain oedema (especially in large parietal strokes)
4. Further strokes, cerebral emboli, or vasculitis
5. Hydrocephalus (especially in cerebellar strokes)

6. Hemorhagic conversion (especially in large infarcts)
7. Review differential diagnosis
8. Consider, repeat CT, EEG (for possible encephalitis or epilepsy), LP

Secondary Prevention
Patients with new onset TIAs or minor strokes are at high risk of stroke or early recurrence. Initiate secondary prevention as soon as possible.

1. **Aspirin**: 150 mg daily if haemorrhage excluded by CT or unless contraindicated
2. **Warfarin**: for all patients with AF who have no contraindications. Start 2 weeks after stroke with 3 mg daily (no need to achieve rapid anticoagulation), check INR frequently (patients at higher risk of bleeds). Stop aspirin
3. Give advice to stop smoking.
4. Indentify and treat hypertension, diabetes, hyperlipidaemia
5. Refer for carotid endarterectomy if Doppler shows > 70% stenosis in appropriate carotid artery and patient fit for surgery

DISCHARGE POLICY

1. Make sure all secondary prevention measures are in place and follow-up arranged

SUBARACHNOID HAEMORRHAGE

RECOGNITION AND ASSESSMENT

Symptoms and Signs

1. Severe headache of sudden onset (becoming severe within seconds) implies subarachnoid haemorrhage until proved otherwise. It may be associatd with vomiting and loss of consciousness, with subsequent photophobia and neck stiffness.
2. The symptoms can sometimes resolve within a few hrs but should still be investigated with CT head scan. 30% of patients with SAH may have minor 'leaks' hrs or days prior to often misdiagnosed as simple headaches or migraine.

Investigations

CT head scan. If negative' equivocal, examine CSF for blood/xanthochromia.

Differential Diagnosis

1. Meningitis
2. Encephalitis
3. Migraine

IMMEDIATE TREATMENT

1. If consciousness impaired, check airway and maintain it.
2. **Dihydrocodeine** 30 mg orally (or 50 mg IM) 4 hrly as

required or alternate analgesia
3. Observe respiratory effort and monitor ECG
4. Call neurology unit

SUBSEQUENT MANAGEMENT
(BY NEUROLOGIST)

1. **Nimodipine** 60 mg orally 4 hrly including throughout night. If unconscious srush tablets and give via NG tube.
2. Laxatives

If improving:
Consider angiography
If Not improving:
Request a further CT head scan

MONITORING TREATMENT

1. Until headache has subsided and the patient is stable, monitor BP 4 hirly
2. When stable, monitor BP twice daily in patients taking nimodipine
3. Prevent constipation, vomiting, cough

DISCHARGE POLICY

1. As a rule, angiography is carried is carried out with a view to operative treatment. If no operative inter vention planned, continue **oral nimodipine** for a

total of 21 days. Discharge after 2-4 weeks and review in an outpatient clinic
2. If patient is hypertensive, treat blood pressure aggressively at this stage.

ACUTE SPINAL CORD COMPRESSION

RECOGNITION AND ASSESSMENT

Symptoms and Signs

1. Weakness of arms or legs or both
2. Sensory level
3. Hyperreflexia and extensor plantar responses (not that because of spinal shock these may not be present at the outset)
4. Bowel/bladder/sexual dysfunction
5. Local spinal pain and/or tenderness

Investigations

1. An MRI scan likely to be required but referral for an urgent spinal opinion should not be delayed by the performance of an MRI scan. An MRI of the whole neural axis may well be required
2. Blood complete, U & E, LFTs
3. Chest x-ray

Differential Diagnosis

1. Transerve myelitis
2. Cord ischaemia
3. Guillain-Barre syndrome

IMMEDIATE TREATMENT

1. **Dexamethasone phosphate**

4mg IV 6 hrly

2. Immediate referral to the on-call Orthopaedic spinal team or Neurosurgeon is necessary

Acute spinal cord compression is true emergency and such patients should be immediately referred to Neurosurgeon. It is better if the emergency MRI scan is organized by the Neurosurgeon rather than the referral being delayed for this purpose.

MONITORING TREATMENT

Until headache has subsided and the patient is stable, monitor BP 4 hrly

SUBSEQUENT MANAGEMENT & DISCHARGE POLICY

This will be decided by the Neurosurgeon

DIABETIC KETOACIDOSIS (DKA) AND NON-KETOTIC HYPEROSMOLAR COMA

RECOGNITION AND ASSESSMENT

Symptoms

1. Thirst
2. Weight loss
3. Polyuria

Signs

1. Flushed appearance
2. Sighing respiration (Kussmaul breathing)
3. Odour of ketones
4. Dehydration
5. Drowsiness
6. Coma

Investigation

1. Blood glucose (capillary)
2. Test for ketones in plasma and or urine
3. Electrolytes
4. Blood gases
5. Blood glucose (venous)
6. MSU
7. ECG
8. Chest x-ray

History and initial examination should be brief but must include an assessment of precipitating cause of DKA. There should be a thorough search for sepsis (including blood cultures if pyrexial), and the cardiovascular system should be assessed looking for signs of shock or evidence of recent myocardial infarction.

IMMEDIATE TREATMENT

1. Blood glucose > 360 mg/dl in the presence of ketones or metabolic acidosis should be managed vigorously
2. IV fluid; **Sodium chloride** 0.9% in following regimen -1L over ½ hr; 1L over 1 hr; 1L over 2 hr; 1L over 4 hr. Further replacement dictated by the patient's condition, usually 4-6 L over next 24 hr.
   Non-ketotic hyperosmolar coma should be treated in the same way; sodium chloride 0.45% should be used only if plasma Na> 150 mmol/I, either initially or after startin treatment with sodium chlorie 0.9%
3. **Insulin infusion** 1 unit/ml in sodium chloride 0.9%: via IV syringe pump at 6 units/hr; if no change in glucose after 2 hr (very unusual – check pump and patency on IV cannula) double the dose and continue doubling at hrly intervals until response occurs
4. If IV infusion pump is not available, bolus doses of insulin 6-10 units IM hrly can be given depending on blood glucose.
5. **Potassium** need depends on initial plasma concentration: if K>5.5 add nil; if K 3.5 5.5 infuse at

20 mmolhhr; if K<3.5 infuse at 40 mmol;hr. When potassium is infused, the heart rate and rhythm should be monitored by ECG.

6. **Bicarbonate;** Use only with extreme caution and **after discussion with a consultant.** As a guide - if after 2-3 L of sodium chloride 0.9%, pH>6.9, give nil sodium bicarbonate; if pH 6.8-6.9 give 50 mmol sodium
7. General: A nasogastic tube should normally be introduced if the patient is unconscious, and the stomach aspirated
8. Give a broad-spectrum antibiotic after blood cultures taken (eg **co-amoxiclv** 8 hrly) if patient is febrile and no obvious cause can be found
9. Introduce a urethral catheter to monitor urine volume if patient is hypotensive or comatose or fails to pass urine within 3 hr of starting IV fluids
10. Although patient with hyperosmolar coma have an increased risk of venous thromboembolism, prophylactic heparin increases the risk of Bl bleeding, so treat only **proven** venous thromboembolism

SUBSEQUENT MANAGEMENT

1. When blood glucose has fallen below 270 mg/1 change infusion to glucose *5%* at a rate of 1L 8 hrly and reduce **insulin** infusion rate to 3u/hr
2. If blood glucose falls below 90 mg/dl change IV fluid to 10% glucose
3. Add potassium according to plasma concentration

4. If hypotension persists beyond 12 hr, look again for evidence of sepsis, myocardial infarction or pancreatitis. Discuss further management with consultant Consider transfer to ICU
5. As a rule subcutaneous insulin can be started within hr of admission, by which time the patient should be normoglycaemic and eating normally. As a guide, the total dose for the first 24 hrs of **SC insulin** will be 2/3 of the previous 24 hrs requirement for IV insulin; give as short-acting insulin in divided dises 8 hrly. Continue IV insulin for 1 hr after first dose of SX insulin
6. Regular capillary glucose be measured hrly to adjust dose of insulin

MONITORING TREATMENT

1. Monitor patient for complications of over-rapid treatment.
2. Cerebral oedema (decreased conscious level)
3. ARDS (Adult Respiratory Distress Syndrome; hypoxia resistant to high Fio_2). Consider transfer to ICU.

DISCHARGE POLICY

Follow for diabetic control mandatory preferably in diabetic clinic.

ACUTE HYPOGLYCAEMIA

Remember hypoglycaemia begets hypoglycaemia. In cases of severe hypoglycaemia be watchful for another episode in next 24 hrs.

RECOGNITION AND ASSESSMENT

Symptoms/Signs

1. Skin cold, clammy
2. Tachycardia
3. Restlessness
4. Confusion
5. Coma

Consider

Hypoglycaemia in any patient with acute agitation, abnormal behavior or impaired consciousness. These signs do not usually occur unless blood glucose falls below 45 mg/dl but can occur at higher concentrations in insulin-dependent diabetics whose day-today blood glucose is above normal.

Investigations:

1. Finger-prick blood glucose strip (if not available, treat after taking venous sample)
2. Venous sample for blood glucose (if venous access not possible, give glucose immediately)
3. If recurrent hypoglycaemia, consider
4. LFTs
5. U&Es
6. Short synacthen test (? Addision's)
7. TSH/Free T4 (? Hypothyroid)

IMMEDIATE TREATMENT

If semi-conscious:

1. **Glucose 25 g** (or 3-4 lumps of sugar) in 150 ml of water orally. Repeat as necessary after 10-15 min.

If unconscious:

2. **Glucose 20%** 100 ml IV over 15 min. Repeat if still unconscious after 15 min
3. Once conscious, give oral glucose or further carbohydrate intake
4. If hypoglycaemia induced by excess oral agents or overdose of insulin, consider maintenance IV infusion of **glucose 5% or 10%**
5. All patients with severe hypoglycaemia, especially if caused by oral agents, should be admitted for observation and monitoring.

SUBSEQUENT MANAGEMENT

1. Review maintenance treatment for diabetes.
2. Seek cause of hypoglycaemia (eg poor control, too much insulin)
3. If hypoglycaemia prolonged, continue IV **glucose** infusion (hypoglycaemia can persist for several days in patients taking sulfonyleurease)

MONITORING TREATMENT

Blood glucose (finger-prick) four times daily before meals.

DISCHARGE POLICY

Ensure diabetic control stable

ACUTE ADRENAL INSUFFICIENCY

RECOGNITION AND ASSESSMENT

The commonest cause is secondary adrenal failure, where mineralocorticoid production is generally preserved.

Symptoms

1. Lethargy
2. Nausea
3. Weight loss

Signs

Primary adrenal failure

a. Hypotension (postural/sustained)
b. Pigmentation (palmar/buccal/scars/pressure areas)
c. Vitiligo

Secondary adrenal failure

a. Pallor

Risk Facotrs

Primary adrenal failure

a. Auto-immune disease (diabetes/ hypothyroidism/ pernicious anaemia)
b. TB
c. Metastases especially from ca lung

Secondary adrenal failure

a. Withdrawal of oral (or potent topical or inhaled) steroids
b. Pituitary surgery/ radiotherapy

Investigations

1. Blood Complete
2. U&E
3. Blood glucose
4. Random serum cortisol
5. Plasma ACTH (EDTA sample-contact laboratory and ask for plasma to be separated on arrival)
6. Unless severely ill, perform Short Synacthen Test (serum cortisol before, then 30 min and 60 min after tetracosactide 250 mcg IM)
7. Primary adrenal failure confirmed by serum cortisol<150 nmol/ 1 when ACTH>80 pg/ml; excluded by basal or peak serum cortisol> 550 nmol/1 during Short Synacthen Test

Indicators of severty

1. Hyponatraemia
2. Hypoglycaemia in primary adrenal failure only:
3. Hyperkalaemia
4. Raised urea

IMMEDIATE TREATMENT

Blood sample for serum cortisol and plasma ACTH must be obtained before hydrocortisone is given but treatment must not await the result.

If severely ill

1. **Hydrocortisone** 100 mg as IV bolus, followed by 100 mg IV 6 hrly
2. **Sodium chloride** 0.9% 1 L by IV infusion over 30-60 min, followed by 3-4 L IV over next 24 hrs

3. If hypoglycaemic, give simultaneous infusion of
4. **Glucose 20%** 100 ml by IV infusion over 30 min, followed by glucose 10% 1 L by IV infusion over 12 hr. Monitor blood glucose and change to glucose 20% if 10% inadequate.
5. Glucagon is **unhelpful in** this situation

SUBSEQUENT MANAGEMENT

When improving and tolerating oral fluid

1. **Hydrocortisone** 20 mg orally 8 hrly
2. Refer to endocrinology unit for advice on maintenance dosage
3. If diagnosis in doubt, substitute **dexamethasone** 1 mg orally 8 hrly for hydrocortisone and perform Short Synacthen Test within 3 days.

In primary adrenal failure

1. Add **fludrocortisone** 50-100 mcg orally daily
2. Request adrenal autoantibodies
3. Arrange chest and abdominal x-rays
4. If TB suspected, request CT scan of adrenals

If secondary adrenal failure suspected, refer to endocrinology unit.

MONITORING

1. U&E dialy
2. Lying and standing BP twice daily

DISCHARGE POLICY

1. Patients must understand need for life long **hydrocortisone:**

- Doubling the daily dose for the duration of any intercurrent illness
- Parenteral hydrocortisone if vomiting (supply with ampoule of hydrocortisone 100 mg to keep in fridge)

2. Arrange steroid for patients.
3. Asked patients/ relatives to keep hydrocortisone injection at home for use in emergency
4. Arrange follow-up with endocrinology unit

HYPERKALAEMIA

Conversion factor from SI unit (mmoles) to old units (mg) for calcium is 4.008 & for Sodium & Potassium is 1.

RECOGNITION AND ASSESSMENT

Plasma Potassium > 6 mmol/I

Common Causes

1. **Increased Intake**
   Food containing potassium and I/V fluids with high consentration of Potassium.
2. **Tissue breakdown**
   Bleeding into GI tract and cavities, haemolysis, rhabdomyolysis
3. **Shift of K out side Cells**
   Tissue damage (shock, ischemia), acidosis, insulin deficiency, aldosterone deficiency & beta blockers.
4. **Impaired Excretion**
   ARF, CRF, potassium sparing diuretics
5. **Pseudo Hyperkalaemia**
   Release of potassium from cells in vitro due to mishandling of blood specimen.

Symptoms and Signs

1. Frequently none, or non-specific
2. Neuromuscular symptoms
3. Cardiac arrest without warning

Investigations

1. Repeat potassium, urea & other electrolytes (U & E) on **plasma** sample (lithium heparin), as potassium rreleased from cells during clotting in serum may give an artificially high level.
2. Blood Complete
3. HCO_3 in venous blood (or from blood gases, if indicated for other reasons)
4. Monitor urine output
5. Serum CPK (rhabdomyolysis)
6. ECG changes: peaked, Tall T waves and PR and QRS complexes are lengthened.
7. If cause not obvious take blood for cortisol

Management

Management should depend on **plasma potassium (K) level**

1. Identify and treat the underlying cause
2. Inject 10-20ml of 10% **Calcium gluconate** I/V over 10 minutes in sever cases. It has membrane stabilizing effect & prevent arrhythmias. Continue ECG monitoring and repeat as necessary while awaiting correction of potassium.
3. Give 10 units of **regular insulin** IV and 50 ml of 50% glucose I/V over 10 minutes into a large vein or, if access poor, give 500 ml glucose, 10% IV by infusion over 30 minutes with 10 units of insulin.
4. Alternatively, **salbutamol** 0.5 mg I/V in 5% dextrose over 15 mins.
5. **Calcium resonium** 15 to 30 gm orally
6. In case of metabolic acidosis **sodium bicarbonate** (NaHCO3) 1.26% 500 ml 6-8 hrly

HYPOKALAEMIA

RECOGNITION AND ASSESSMENT

Plasma potassium < 3.5 mmol/l

Common Causes

1. Secondary to GI fluid loss and volume depletion (vomiting and diarrohea).
2. Intracellular shift: insulin or bicarbonate treatment, theophylline, beta agonists, rapid blood cell proliferation.
3. Renal loss: urine K >20 mmol/l, diuretics.
4. Mg deficiency, renal tubular disease.
5. Blood taken from drip arm (false)

Symptoms and Signs

1. Often none, or neuromuscular symptoms, eg muscle weakness, absent reflexes, abdominal distension, ileus
2. Metabolic alkalosis – increased bicarbonate (HCO_3)

Investigations

Immediate

1. ECG changes – depressed ST, flat T, U waves, arrhythmias.
2. Repeat serum potassium + U & E on **plasma** sample (lithium heparin) as **K** released from cells during clotting in serum may give an artificially high level.

2. Helpful

- Blood complete

7. Correct volume depletion if present
8. Use haemodialysis / peritoneal dialysis if the above fails.
9. If persistent hyperkalaemia or renal failure present (poor urine output, rising creatinine or acidosis) consider dialysis early – refer to nephrology unit
10. If continuing K retention and dialysis unlikely within a few hrs – start **calcium resonium resin** 15 g orally in water (not fruit juice) 6 hrly

MONITORING AND TREATMENT

1. Monitor plasma U & E and glucose 12 hrly until K level stable and < 6.0 mmol/l
2. Attend to underlying cause, eg drugs if patient in renal failure, refer to nephrology unit.

- HCO_3 – a raised level indicates chronic depletion; if less than 22 mmol/l in absence of GI loss suspect renal tubular acidosis & rdfer to Nephrology unit.
- Urine K – if cause not obvious
- Serum Mg – for persistent urine K loss especially patients with diarrhea or on diuretics

Management

Management should depend on **plasma K**

1. Patients on I/V fluids, steroids and diuretics should be given 60-80 mmol/l of potassium to prevent hypokalaemia.
2. **Plasma K <3.0 mmol/l & asymptomatic**
   - Identify and correct underlying cause. If on **digoxin** or symptoms present, ive **NEO-K** 2 tabs 8 hrly (each tablet contains 12 mmol/K)
   - Fruits containing potassium may be useful, in serve cases
3. **Plasma K <3.0 mmo/l**
   + arrhythmia, paralysis or pre-existing cardiac disease
   - KCL 20 mmol added to 500 ml of sodium chloride 0.9%. Mix thoroughly and give iv at 10 mmol/hr of KCl (250 ml of sodium chloride 0.9%) with continuose ECG monitoring.
   - In intractable cardiac arrhythmia double infusion rate and contact cardiology unit urgently

MONITORING AND TREATMENT

1. K deficit is difficult to estimate monitor replacement with plasma K levels (at least daily if given iv)
2. If cause is not obvious, refer to nephrology or endocrinology unit for further evaluation

HYPERCALCAEMIA

RECOGNITION AND ASSESSMENT

Plasma Calcium > 10.5 mg / dl

Common Causes

1. Primary hyperparathyroidism (10-20%)
2. **Malignancy:**
   - Local osteolytic bone lesions.
   - Humoral hypercalcaemia (tumour – derived PTH related protein, PTHrP, has similar actions to PTH).
   - Common cancers include breast, lung, myeloma, lymphoma, oesophagus, renal, prostate, and head and neck primaries.
3. **Granulomatous diseases**
   - Sarcoidosis
   - TB
4. **Drugs**
   - Vitamin D
   - Vitamin A
   - Thiazide diuretics
5. **Immobilization**
6. **Metabolic causes**
   - Thyrotoxicosis
   - Phaeochromocytoma
   - Milk-alkali syndrome (antacids, calcium carbonate therapy)

Symptoms and Signs

1. Unusual unless Ca^{++}>12mg/l
2. Gl: nausea, vomiting, constipation, abdominal pain
3. Renal: polyuria, polydipsia
4. CVS: hypertension, on ECG altered QT interval, long PR, wide QRS, arrhythmia
5. CNS: various including depression, cognitive difficulties, headache, altered consciousness, acute psychosis

Incestigations

1. Immediate U & E, Ca^{++}, albumin, chest x-ray, ECG
2. Non-urgent to find cause: PTH (EDTA), Blood Complete, ESR, alk phosphatase, phosphorus level, myeloma screen, amylase/ lipase level (pancreatitis)
3. Determine degree of hypercalcaemia after correcting to albumin of 40 g/l = serum calcium + 0.02 (40 albumin g/l) mmol/l

Management

1. **MILD**
   - Immediate treatment not usually necessary but ensure adequate fluid intake, stop thiazides and any vitamin A, D or Ca supplements
2. **MODERATE**
   - **Oral rehydration** if possible: water 2-3 l/day. If oral route inappropriate give 2-3 sodium chloride 0.9% by IV infusion/day. Ca should decrease by 0.5 mmol/l within 48 hr
   - Check U & E & Ca^{++} at 48 hr

HYPERNATRAEMIA

RECOGNITION AND ASSESSMENT

Serum Sodium (Na)> 150 mmol/I

Mechanism

Decreased total body water or increased total body sodium

Symptoms and Signs

Various CNS symptoms from lethargy to coma and fits, dehydration hypovolaemia

Investigations

1. Serum U & E, glucose.
2. Urine U & E, osmolality

Management

Assess volumic status whether hypovolemic or normovolumic

A. If hypovolemic, expectd result will be Urine osmolality (Uosm)> 300 mmol/I or Uosm<300 mmol/I

Casuses

1. Uosm> 300 mmol/I
   - Osmotic diuresis , eg hyperglycaemia.
   - Excess water loss e.g. sweat, osmotic diarrhea
   - Inability to dring, failure of thirst
2. If Uosm < 300 mmol/I

   Cause may be Diabetes insipidus
   - Pituitary
   - Nephrogenic

3. **SEVERE**
   - Rehydrate with sodium chloride 0.9% 304 I/24 hrs depending on severity of symptoms and Ca level. Caution in elderly, LVF, or renal impairment. If fluid overload give **frusemide** 20-40 mg IV 12 hrly Large doses of frusemide (160mg) may lower Ca more quickly but are not recommended as electrolytes must be accurately repaced based on urinary loss.
   - Check U & E * Ca at 12 hr
   - If serum Ca^{++}> 13.6 or serum Ca> 12 .0 mmol/I + symptoms. **Disodium Pamidronate** 60-90 mg IV over 4 hr. Ca usually returns to normal within 7 days. The dose of Disodium Pamidronate may be repeated
   - **Calcitonin** 5-10 units/Kg via IV infusion over 6-8 hrs is relatively non-toxic, but its effect wears off after few days. It is rarely effective when **bisphosphonates** have failed to reduce serum calcium adequately

OTHER TREATMENT

STEROIDS: If cause known to be granulomatous disease or calcitriol excess (Vit D intoxication): hydrocortisone 100 mg Iv 8 hrly (or prednisolone 40 mg orally daily). Calcitriol excess usually responds poorly to disodium pamidronate.

Further treatment

If causes not treatable ensure adequate hydration all the time

IMMEDIATE TREATMENT

Encourage oral fluids in conscious patient the safest

1. **If Asymptomatic**
   - Give oral fluids
   - Monitor serum Na Daily
2. **If symptomatic + Hypovolaemic**
   - Sodium chloride 0.45% sufficient to achieve haemodynamic stability (suggested rate 1 L in 2 hr)
   - If the cause is not related to diabetes mellitus the hypernatraemia can then be corrected with glucose 5%
   - If the hypernatraemia is due to diabetic non-ketotic hyperosmolar coma, switch from 0.45% saline to 5% glucose (when blood glucose has fallen to 150-200 mg/dI)
   - If the cause is not apparent at this stage, diabetes insipidus should be considered and patient referred to endocrinology unit.

B. If normovolumic, expected result will be Uosm >300mmol/I, Una >5mmoll/I

Causes

Excess salt administration

Treatment

If Asymptomatic

Discontinue sodium access

If symptomatic +

- **Normovolaemic** Estimate the water deficit from: 0.6 x lean body weight* x (serum Na 140)/140

- Aim to replce 50% of this in 24 hr as glucose 5% Iv
- Monitor serum Na 12 hrly
- Serum Na must not decrease by more than 10 mmol/I in 24 h

HYPONATRAEMIA

RECOGNITION AND ASSESSMENT

Plasma sodium 125 mmol/I

Causes

Common Causes

1. **Low Extra cellular fluid (ECF)**
   - Hypovolumia (DKA, vomiting **&** diarrhea
   - Cirrhosis, adrenal failure, salt losing renal disease
2. **Normal ECF**
   - Nephrotic syndrome, Hypothyroidism, Diuretics NSAID, post op, pain/analgesia
3. **High ECF**
   - Renal failure, cardiac Failure, SIADH

Symptoms and Signs

1. Nausea
2. Cramps
3. Confusion
4. Lassitued
5. Somnolence
6. Fits and Varied CNS manifestations. (Na<110mmool/I)

Diagnosis

1. History including drug ingestion and fluid balance and weight chart
2. Clinical assessment of plasma volume

Investigations

1. Urea and creatinine

2. Urinary sodium and Osmolality

Management

1. **Plasma Na>120 mmol/L**
   - Water restrictions 0.5L
   - **0.9% saline** if volume depleted stop drugs and treat the specific cause.
2. **Plasma Na>110-120 mmol/L**
   - Water restriction<0.5L
   - 0.9 normal saline IL-12 hrly
   - Add **frusemide** 20-40 mg (Oral) if volume over loaded.
3. **Plasma Na<110 mmol/I** or Neurological signs
   - 1.8% or 3% saline IV to raise sodium by 0.5 mmol/I per hour
   - Add **frusemide** 20 mg IV if volume over loaded

HYPOCALCAEMIA

RECOGNITION AND ASSESSMENT

Plasma Calcium <8.5 mg/dl (2.2 mmol/I)

Common Causes

1. Hypoparathyroidism
2. Alkalosis (Metabolic/Respiratiry)
3. Vatamin D Deficiency
4. CChronic Renal Failure
5. Acute Pancreatitis
6. Hypoalbuminaemia

Symptoms and Signs

1. In children there is a triad of carpopedal spasm, stridor and convulsions but only one of them may be present
2. In adults there is tingling of hands, feet and around the mouth. Carpopedal spasm is less common
3. Trousseau,s sign and chovstek's sign may be present in cases of latent hypocalcemia

Investigations

Serum Calcium level especially ionized Ca

Management

1. Alkalosis is reversed by re-breathing of expired air in paper bag or adminstring 5% Co2 in O2
2. Injection of 20 ml of 10% **Ca gluconates** slowly in a vein

3. I/M Injection of 10 ml of Ca gluconate (for prolonged action)
4. Identity and treat the cause…
5. If tetany is not relieved with **calcium** then **magnesium** should be given

CEREBRAL MALARIA

RECOGNITION AND ASSESSMENT

1. It is the most feared complication of *plasmodium falciparum* infection with a substantial mortality
2. It is a diagnosis of high degree of clinical suspicion and should be considered in any febrile patient with impaired conscious state.
3. Though the patients are usually febrile, don't expect typical malarial pattern and the patient may even be afebrile.
4. Patients with cerebral malaria usually present with ***altered sensorium*** ranging from confusion, delirium, stuporous to coma state. ***Onset may be sudden or gradual*** following a seizure (up to 50% in children).
5. It is a diffuse symmetric encephalopathy and focal neurological signs are rare (though presence of hypoglycemia may manifest as focal neurological signs like hemiparesis or focal fits).
6. Signs of meningeal irritation are lacking while primitive reflexes like pout reflex may be present.
7. Fundoscopic examination may reveal retinal hemorrhages (30-40%), spots of discrete retinal opcification (30-60%), papilloedema (8% in children, rare in adults) and cotton wool spots (<5%).

Diagnosis

Clinical and laboratory assessment:

1. Peripheral smear for Malarial Parasite, especially requesting for detection of Falciparum and to assess

the level of parasitemia (>5% parasitemial or >10^{o} parasites per milliliters are at increased risk of death).
2. Assess the conscious state, use Glasgow Coma Scale.
3. Assess the hydration status and keep strict intake output record. Consider Acute renal shut-down if patient becomes oliguric (<500ml/24 hrs).
4. Look for evidence of Acute Renal Failure as concomitant ecent (Black water fever) by checking serum creatinine and blood urea on arrival and then daily.
5. Check blood glucose levels immediately and then every 4-6 hrs, especially if the patient is put on Infusion Quinine.
6. Check haematocrit daily, if falls below 20%, arrange and transfuse fresh blood.

IMMEDIATE TREATMENT

1. If unconscious, nurse the patient on sides or put the patient in prone position.
2. Establish IV line.
3. Pass a nasogastric tube (NGT) and feed with **Glaxose-D** fortified fresh juices 80ml/hr. NG feeding will heop in keeping the patient hydrated (1920 ml over 24 hrs) with enough glucose to combat hypoglycemia.

Specific Therapy

1. Commence infusion **Quinine** with a loading dose of 20mg/Kg stat and then 10mg/Kg I/V 8 hrly, given in

MENINGITIS (BACTERIAL)

RECOGNITION AND ASSESSMENT

Symptoms and Signs

1. Headache, vomiting, neck stiffness, photophobia
2. Fever
3. Impaired consciousness, coma and fits
4. Clinical features of septicaemia or severe sepsis

In elderly, confusion may occur in the absence of meningism or even of fever.

Lift-Threatening Features

1. Altered consciousness
2. Focal neurological deficit
3. Raised intracranial pressure
4. Convulsions
5. Concurrent septicaemia (and septicaemic complications)

Investigations

1. CSF-in all patients with fever, headache and meningism **EXCEPT** in patients with focal fits or if SOL is being suspected for some other reason. Under these circumstances get a CT or MRI brain to exclude an SOL and then perform LP even in the presence of papilloedema. Also send CSF for C/S.
2. Blood Complete and differential WBC.
3. U & E, glucose
4. Blood culture (may be positive in 50% cases of pneumococcal meningitis)

10% dextrose water with additional 100ml of 25% D/W (to avoid hypoglycemia) **over four hrs** (i.e. 600 ml infused @ 2.5 ml/min or 40 drops per min via ordinary drip set). Reduce the maintenance dose of Quinine by 50% on day three in case of acute renal shut down. OR

2. Deep **IM Artemether** in a loading dose of 3.2 mg/Kg followed by 1.6 mg/kg OD for five days can also be sued instead of infustion Quinine.

Ancillary therapy

3. Put the patient on a 3rd generation **Cephalosporin** (e.g. Inj **Ceftriaxone** 2 mg I/V stat and daily after test dose) to take care of septicemia, which these patients are prone to develop.
4. In case of acute renal failure haemodialysis should be started as soon as possible.

If not improving

5. Consider exchange transfusion if level of parasitemia is more than 15%.
6. Consider alternate diagnosis like viral encephalitis

DISCHARGE POLICY

1. Patients can be sent home once they are afebrile for 48 hrs, are fully conscious, have startd taking normal meals and have no vomiting.
2. Continue **antimalarials** for ten days.

5. Chest x-ray
6. CT heat scan – if there is doubt aout the diagnosis, if there is altered consciousness, or if there are focal neurological signs.

Differential Diagnosis

1. Subarachnoid hemorrhage
2. Other intracranial sepsis
3. Shystemic sepsis
4. Other causes of confusion or of raised intracranial pressure
5. Encephalitis
6. Malaria

IMMEDIATE TREATMENT

The interval between patient's arrival and commencement of antibiotic treatment ('door-to-needle time') should not exceed 1 hour.

Complicated or Severe Meningitis (With Lif-Threatening Features)

1. In case of Pneumococcal meningitis administer **Ceftriaxone 2mg 12 hrly and add Inj Vancomycin 15mg/Kt if patient dose not improve within 48 hrs.** Also administer Inj **Dexamethasoe** 0.15 mg/Kg concomitantly with first dose of antibiotics and then give 10 mg 6 hrly. If C/S report shows **sensitivity to Penicillin then switch over to benzyl penicillin 2.4 g by IV infusion 4 hrly and continue for 14 days.**
   In cases of penicillin sensitive Meningococcal meningitis use only **Benzyl Penicillin** OR use Inj

Ceftriaxone if penicillin resistant is under consideration. In very young or very old patients or in immunocompromized states also consider Listeria as a possiblility and add Inj Ampicillin 8-12 g/day in divided doses.

2. If meningococcal sepsis is present give **methylprednisolone** 500 mg Iv infusion with first dose of antibiotics
3. Insert CVP line if peripheral IV access is difficult.
4. If lymphocytes predominate in CSF consider partially treated bacterial meningitis, TB meningitis, cerebral abscess, paradural pyogenic collections.
5. If herpes Simplex encephalitis is a possibility, give empirical **acyclovir** 10 mg/kg by IV infusion over 1 hour 8 hrly in addition to the above.

Meningism

1. It can appear in complicated Enteric fever and in systemic viral infections e.g. Mumps or Varicella infections and the treatment is that of underlying disorder.
2. In strongly suspected cases of meningitis start treatment empirically even if CSF examination is not possible, treat with **Ceftriaxone** 2gm 12 hrly.

DISCHARGE POLICY

3. Follow-up in clinic to check for hearing loss
4. Patients with persisting neurological deficit will require rehabilitation
5. Refer to appropriate specialist

ACUTE HOT JOINT, SEPTIC ARTHRITIS AND GOUT

RECOGNITION AND ASSESSMENT

Symptoms

Acutely painful, swollen joint

Signs

Warm, tender, swollen joit (± effusion)
Pyrexia may not be a feature of septic arthritis in the elderly, immuno-compromise, diabetic, renal failure, rheumatoid arthritis

Patients with prosthetic joint and PUO-consider prosthesis infection

Investigations

Immediate

If knee joint swollen, aspirate-see

Practical Procedures–Knee Aspiration

1. Blood Complete
2. Microbiology
3. Blood cultures
4. Swab from any infected skin lesion
5. If gonococci suspected, swab rectum, urethra, and throat

Within 24 hrs

6. ESR
7. CRP

8. Serum uric acid
9. X-ray of affected joint

Differential Diagnosis

1. Septic arthritis
2. Crystal arthritis including gout
3. Acute reactive arthritis
4. Acute inflammatory arthritis-eg rheumatoid arthritis
5. Haemarthrosis

IMMEDIATE TREATMENT

Supportive

1. Adequate analgesia for joint pain: **diclofenc** 50 mg orally 8 hrly (if not contraindicated) plus.
2. Step 1: **paracetamol** 1 g orally 6 hrly if requird
3. Step 2: if not adequate, change to **Tramodol** orally 1 tab 8 hrly.
4. Refer to physiotherapists for ice pack and splint on joint
5. Rehydration
6. If patient already taking low dose **sterodis,** consider increasing to **prednisolone** 10 mg orally daily

Antibiotic Therapy

Start as sonn as joint aspirated

Commonest microbe is *Staph aureus*

1. If immunocompetent patient with no other inflammatory arthritis and only 1 joint involved: **Sodium fusidate 500 mg orally 8 hrly + EITHER**

- **Flucloxacillin 500 mg IV 6 hrly OR (if allergic to penicillin) Erythromycin** 500 mg IV 6 hrly

2. If patient has rheumatoid or other inflammatory arthritis and a septic join: **Flucloxacillin** 500 mg IV 6 hrly + **amoxicillin** 500 mg IV 8 hrly
3. If gonococci isolated **Ofloxacin** 400 mg orally 12 hrly
4. If sepsis syndrome present, see **Sepsis, Sepsis Syndrome and Septic Shock**

MONITORING TREATMENT

1. Pulse, BP, temperature 4 hrly until patient is stable
2. Repeat daily aspiration and culture of joint effusion, while effusion persists
3. WBC, ESR, CRP every 48 hr
4. Liver function every week if using sodium fusidate

SUBSEQUENT MANAGEMENT

Septic Arthritis

Antibiotic Therapy

1. Adjust antibiotics on results of therapy and bacterial sensitivities

Always use two antibiotics for Staph aureus

2. Continue IV antibiotics until apyrexial and no systemic symptoms; then change to equivalent oral antibiotics
3. Oral antibiotics should be continued for 6 week, or longer If joint not settled

Supportive

Rdfer to physiotherapists for passive exercise and rehabilitation.

Failure to respond to therapy

1. Reconsider diagnosis
2. Repeat cultures
3. Refer to rheumatologis/ physician/ orthopedic surgeon especially for consideration of arthrotomy or arthroscopy with lavage if no response within 72hr

Confirmed Gout

1. Gout is confirmed by microscopic identification of urate crystals in synovial fluid
2. Rehydrate. Consider stopping diuretics
3. **Naproxen** 500 mg orally 8 hrly OR **diclofenac** 50 mg orally 8 hrly. If NASAID contraindicated, **colchicine** 0.5 mg orally 6 hrly. Higher doses can be used bur beware of abdominal pain, vomiting or diarrhea.

Do not start allopurinol in acute gout

In difficult or resistant cases contact rheumatologist/ physician/ Orthopaedic surgeon

DISCHARGE POLICY

Refer new cases to a Rheumatologist.

SEPSIS SYNDROME & SEPTIC SHOCK

RECOGNITION AND ASSESSMENT

Sepsis is a systemic response to bacteraemia or septicaemia. It commonly occurs in patients who are immunosuppressed, or who have recently undergone an invasive procedure

Symptoms and Signs

1. Rigors, sweating, fever
2. Headache, muscle pain
3. Features of primary infection; Sources include urinary trace lower respiratory tract, meningitis, skin, intra-abdominal infection, pelvic inflammatory disease, endocarditis, osteomyelitis, sexually transmitted disease

Lift-threatening Features

1. Sepsis syndrome: sepsis with impaired organ function (eg diminishing renal function, impaired cardiac function, hypoxia, acidosis, ARDS, clotting disturbance)
2. Septic shok: sepsis syndrome with systolic BP>90 mm Hg

Investigations

1. Blood Complete and differential wBC
2. Chest x-ray
3. Biochemical screen (save serum for cortisol)
4. Glucose
5. Arterial blood gases and acid-base

6. INR, APTT
7. Culture blood, urine, faeces (if enteric infections suspected), CSF (if any hint of meningitis; omit if patient confused or intracranial pressure is raised)
8. Bone marrow examination
9. Consider CT, ultrasound and nuclear medicine imaging if source of infection not apparent

Differential Diagnosis

1. Systemic infections: staphylococcal toxic shock syndrome, malaria, viral haemorrhagic fevers, rickettsial infections, systemic inflammatory response syndrome
2. Systemic disease: occult haemorrhage, myocardial infarction, adrenal insufficiency, pulmonary embolism

IMMEDIATE TREATMENT

Consider admission to ICU

Supportive

1. **Oxygen** (highest concentration possible)
2. Insert CVP line and restore CVP to normal with crystalloid, or blood if patient also anaemic.

Antibiotic therapy

3. No obvious source of infection, or suspected origin in respiratory or urinary tract, cardiovascular system, or skin: **Ceftriaxone** 2 g IV daily (+**gentamicin** for life-threatening infection or in patients recently treated with cephalosporin)

4. Intestinal involvement: as above, Where perforation or abdominal abscess suspected add **metronidazole** 500 mg IV 8 hrly
5. If salmonella suspected, use **ciprofloxacin** 750 mg orally 12 hrly, or 200-400 mg IV 12 hrly by infusion over 30-60 min, instead of **cefuroxime** and **gentamicin**
6. Immunosuppressed (including neutropenic patients; **Timentin** 3.2 g IV 4-6 hrly by infusion + **gentamicin** 2.5mg/kg 12 hrly. Do not use single daily dosage regimen. Check gentamicin levels at least twice a week.
   If allergic to penicillin substitute **Ceftazidime** 2 G IV 8 hrly V.
7. **SURGICAL DRAINAGE**, IF APPROPRIATE
8. **OTHER MEASURES**
   Patients with **septic shock** require inotropic agents and further plasma expansion while monitoring pulmonary capillary wedge pressure.
   Contact ICU.

SUBSEQUENT MANAGEMENT

If Improving:

a. Continue oxygen and maintain CVP
b. Continue antibiotics, changing to oral route after resolution of sepsis and ceasing after a further 5 days

If Not Improving:

a. If systolic BP does not rise above 95 mmHg after initial volume expansion, treat as septic shock (see above)

MONITORING TREATMENT

1. Temperature, pulse, BP (1/4 hrly)
2. Urine output, CVP (hrly)

DISCHARGE POLICY

1. Where sepsis syndrome resolves, recovery is usually complete, Outpatient follow-up depends on severity and source of infection

ACUTE POISONING/ DRUG OVERDOSAGE

RECOMMENDATION AND ASSESSMENT

Features of drug overdose

1. Any comatose patient aged 15-55 years is likely to have taken a drug overdose
2. Depressed respiration suggests a centrally acting drug
3. Skin blisters (between knees/ toes) common after barbiturates and tricyclics
4. Hypothermia after exposure to cold or barbiturates
5. Venepuncture marks and pinpoint pupils suggest opioid overdose

Life-threatening features

1. Coma
2. Cyanosis
3. Hypotension
4. Paralytic ileus

Identify likely poison(s)/ drug(s)

Ask patient, relatives, retain any containers found

Investigations

1. U & E
2. Blood gases and acid-base
3. Save blood and urine for toxicological analysis, Urgent measurement of plasma/serum concentrations is essential in the diagnosis and management of poisoning with **ethylene glycol,**

iron, lithium, methanol, paracetamol, theophylline and salicylate.

4. With the exception of paracetamol there is no need to measure the concentrations of these substances unless there is a clear history of ingestion
5. Plasma paracetamol concentration should be requested in all unconscious patients in whom dfug overdosage is considered

IMMEDIATE TREATMENT

Separate guidelines give more detailed advice on the management of overdosage with aspirin, paracetamol, and tricyclic antidepressants.

1. Secure and maintain airway
2. Maintain circulation. If BP>100/60 mmHg with poor peripheral circulation despite being placed in the head down position, give sodium chloride 0.9%and insert CVP line. Do not use vasoconstrictor drugs
3. Give antidote if appropriate
4. Discourage absorption; consider gastric lavage only in patients whose airway can be protected and who have ingested life-threatening amounts of a toxic agent up to **1 hr** previously, provided they are co-operative and have not ingested petroleum distillates or corrosives. See **Parctical Procedures-Gastric Lavage**. Leave **50 g activated charcoal** behind in the stomach bdfore removing tuve
5. If lavage not indicated, **give activated charcoal 50 g** by mouth or nasogaslric tuve to patients who present **within 1 hr** of ingestion, provided airway

can be protected. Activated charcoal does not affect absorption of acids, alkalis, alcohols, cyanide, ethylene glycol, iron or lithium
6. Avoid ipecacuanha – it does not empty the stomach reliably and can be dangerous
7. Stop any regular medications that might enhance the effect of the substance taken in overdose

If life-threatening features are present:

1. Hypoxia or hypercapnia
2. Feeble respiration
3. Respiratory arrest

SUBSEQUENT MANAGEMENT

1. Supportive care alone is required for most acutely poisoned patients
2. After poisoning with certain chemicals/drugs further active measures may be necessary
3. Increase elimination. In patients who have ingested life-threatening doses of **carbamazepine, dapsone, phenobarbital, quinine, or theophylline, give activated charcoal 12.5 ghrly via nasogastric tube, to total of 150-220 g**
4. Consider alkaline diuresis after overdose of **aspirin** if plasma salicylates>500mg/ (>3.6 mmol/I). **phenobarbital** or **barbital** (plasma concentration > 10 mg/I). Alkaline diuresis is contraindicated in patients who are shocked, or have impaired renal function or heart failure.

MONITORING TREATMENT

1. Monitor conscious level, temperature, respiration, pulse and BP until they return to normal
2. There is no need to monitor drug concentrations other than to guide the use of measures to enhace drug elimination

Psychiatric reviw

All patients admitted after acute self-poisoning or deliberate drug overdose should he offered an interview with a member of Psychiatry unit within 24 hrs of admission or regaining consciousness

DISCHARGE POLICY

When discharged from hospital patients should have:

1. Been conscious and alert with normal vital signs for at least 6 hr
2. No evidence of significant organ dysfunction as a result of poisoning/ drug toxicity
3. Been interviewed by a member of the Psychiatry Team
4. Follow-up appointment in psychiatric clinic (if recommended by psychiatrist)
5. Follow-up appointment in medical clinic (if persistent sequelae of poisoning require review)

ASPIRIN

RECONITION AND ASSESSMENT

Symptoms

1. Tinnitus
2. Deafness
3. Blurring of vision
4. Epigastric pain, nausea and vomiting
5. Restlessness
6. Tachycardia
7. Hyperventilation
8. Hyperthermia
9. Sweating
10. Dehydration
11. Vasodilatation
12. Pulmonary oedema
13. Confusion
14. Coma
15. Delirium
16. Hypotention

Investigations

1. U & E creatinine
2. Blood gases and acid base
3. Blood glucose (capillary)
4. Plasma salicylate (if coincident) paracetamol overdose requiring infusion, sample before administration of N-acetylecysteine)
5. Urine pH

IMMEDIATE TREATMENT

1. Consider gastric lavage only if stated dose> 10 g and presentation within 1 hr of overdose. See **Pracitcal Procedures – Gastric Lavage**
2. Give **activated charcoal 50 g** via stomach tube after lavage
3. If gastric lavage not indicated, give **activated charcoal 50 g** orally or via nasogastric tube to patients who present within 1 hr of ingestion.

SUBSEQUENT MANAGEMENT

1. If patient has symptoms or signs of aspirin overdosage give further activated charcoal 12.5 g hrly via nasogastric tube to total of 150-220 g
2. If plasma salicylate < 3.6 mmol/L (500 mg/L), rehydrate orally (IV if vomiting)
3. If plasma salicylate > 3.6 mmol/ L (500 mg/ L) especially in presence of acidaemia and/ or altered consciousness, consider alkaline diuresis (see below) provided **renal function normal** plasma creatinine >1.7 mg/dl (150 umol/L) and there is no evidence of heart failure or shock
4. If alkaline diuresis contraindicated and plasma salicylate level >5.1 mmol/L (700 mg mg/l) consider haemodialysis. Contact nephrology unit.

Alkaline diuresis

1. Insert urethral catheter

2. If hypertensive and CVP low (<-3 cm), correct hypovolaemia with IV infusion of sodium chloride 0.9%
3. Give **sodium bicarbonate** 1.26% IV 500 ml/hr for 3 hr. Add **potassium chloride** 20 mmol to each 500 ml infused to prevent hypokalaemia
4. **Adjust infusion rate to maintain urine pH 7.5 – 8.5**
5. Continue until plasma salicylate < 3.6 mmol/1 (500 mg/L)

MONITORING TREATMENT

1. Measure plasma salicylate 4 hrly until clearly falling
2. During alkaline diuresis, check U & E. blood glucose, acid-base hrly

PARACETAMOL

RECOGNITION AND ASSESSMENT

Symptoms and Signs

1. Usually none
2. Nausea and vomiting occur within a few hrs of ingestion of a hepatotoxic dose

Investigations

1. Plasma paracetamol 4-16 hr after overdose (but not before or after this interval) is a reliable guide to the need for treatment
2. If patient presents > 8 hr after overdose, request baseline
   - Blood complete, IINR
   - U & E, liver function, phosphate
   - Acid-base (venous sample)

IMMEDIATE TREATMENT

Gastric lavage/emesis is not indicated. Activated charcoal is more effective at reducing absorption

1. If patient is thought to have taken > 12 g or 150 mg/kg and presents within 1 hr of dosing give **activated charcoal 50 g** orally or via nasogastric tube
2. Compare plasma paracetamol with treatment graph
   - If above, on, or even slightly below the "treatment line", give **IV acetylcysteine in 5% glucose: 150 min: then 50 mg/kg in 500 ml over 4 hr; then 100 mg/kg in 1 liter ober 16 hr**

- Use high risk treatment line for patients taking enzyme inducing drugs (**eg barbiturates, phenytoin, carbamazepine, rifampicin**) or who abuse alcohol: they are at higher risk of hepatic necrosis

3. If 8-15 hrs hrs have elapsed since a potentially dangerous dose (< 12 g or 150 mg/kg) and the paracetamol concentration is not yet known, give **acetylcysteine** at once while awaiting laboratory result
4. If patient presents 16-24 hrs after a potentially dangerous dose (> 12 g or 150 mg/kg), give **acetylcysteine** according to the regimen above at once. Treatment can be stopped 24 hrs after ingestion if:
   - Patient asymptomatic
   - Plasma paracetamol < 10 mg/l
   - INR normal
5. If patient presents after > 24 hrs and has taken a potentially dangerous dose (> 12 g or 150 mg/kg), or is symptomatic, or has abnormal laboratory results, give **acetylcysteine** (standard regimen). Repeat investigations at end of standard regimen and continue with doses of 50mg/kg in 500 ml over 8 hr if patient has, or is at risk of developing, fulminant hepatic failure. Insert urinary catheter to monitor urine flow and rehydrate to maintain urine output> 100 ml/hr. If unresponsive to IV slowly (not more than 4 mg/min) and consider low-dose **dopamine**.

(see **Prescribing Regimens**) insert CVP line to monitor response to IV fluids only if INR normal

Acetylcysteine can cause a pseudo allergic reaction (wheezing, flushing, and hypotension). If this occurs, stop infusion and give chlorpheniramine 10mg IV (over 1 min) and hydrocortisone 100 mgIV. Once reaction has subsided, recommence infusion at half the original rate

MONITORING TREATMENT

Patients who present within 8 hrs of overdose

1. INR, AST/ALT and plasma creatinine 24 hrs after overdose or when antidote treatment complete

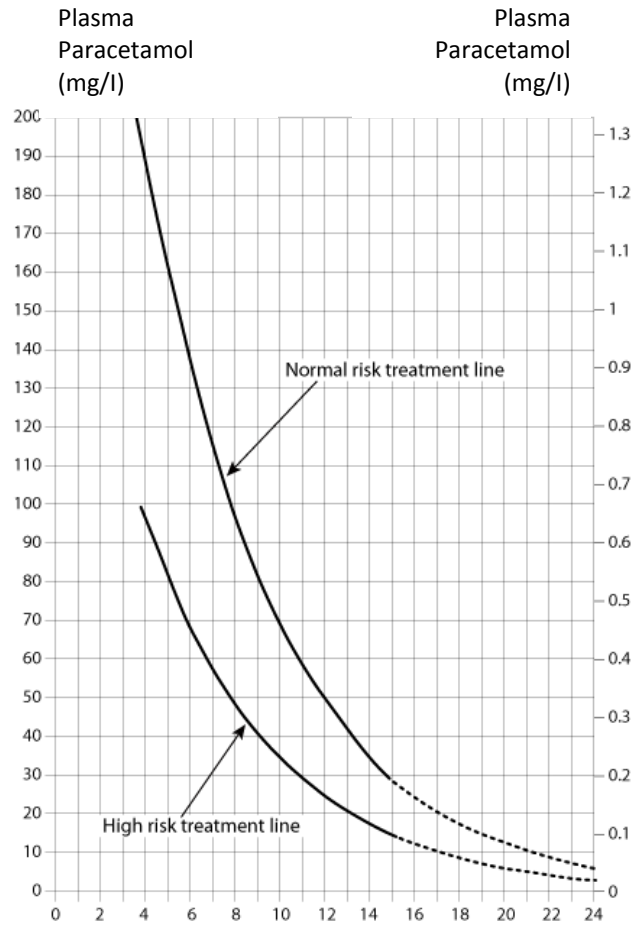
Patients who present 8-15 hrs after overdose

AST/ALT, urea, plasma reatinine and phosphate when antidote treatment complete; if abnormal, repeat at 48 hr after overdose

Patients who present > 16 hr after overdose

1. INR, AST/ALT, plasma creatinine and phosphate when antidote treatment complete and at 48 hr after overdose
2. Urine output
3. Blood glucose – finger prick (4 hrly)
4. Blood gases and acid-base daily
5. Observe for signs of encephalopathy (mental confusion, drowsiness, spatial disorientation, asterixis)

Treatment graph for paracetamol overdose



Plasma paracetamol concentrations in relation to time after dosage as a guide to prognosis Acetyecysteine is indicated in patients with values on or aboce the appropriate treatment line

Life-threatening features

1. A poor prognosis is indicated by:
   - INR> 3.0, blood pH < 7.3
   - Scrum creatinine > 2.2 mg/dl (200 umol/L)
2. If any of these features are present after overdose, seek advice from a gastroenterologist
3. Haemorrhage should be treated with fresh frozen plasma
4. Hypophosphataemia usually occurs after paracetamol poisoning and correlates well with the degree of hepatic-damage

DISCHARGE POLICY

Presenting within 8 hr of overdose

Discharge if 1NR, AST/ALT and plasma creatinine normal at 24 hrs after overdose, or after antidote treatment complete

Presenting> 8 hr after overdose

1. If INR, AST/ALT and plasma creatinine normal after antidote treatment complete, asymptomatic patients may be discharged
2. If INR. AST/ALT and/or plasma creatinine raised after antidote treatment, monitor and observe until these indicaes are falling and are not dangerously abnormal

TRICYCLICS

RECOGNITION AND ASSESSMENT

Symptoms

1. Dry mouth
2. Blurred vision
3. Drowsiness

Signs

1. Excitement, visual hallucinations, delirium
2. Ataxia, athetoid movements
3. Muscle twitching
4. Dilated pupils
5. Hypertonia,
6. Hyperreflexia, extensor planters
7. Convulsions
8. Coma
9. Sinus tachycardia, cardiac arrhythmias
10. Prolongation of PR, QRS, QT interval
11. Hypotension
12. Hypothermia or hyperpyrexia
13. Depressed respiration
14. Urinary retention, paralytic ileus
15. Skin blisters

Investigations

1. ECG
2. U & E
3. Blood gases and acid-base

IMMEDIATE TREATMENT

1. Consider gastic lavage only in patients who have taken a potentially serious overdose (> 750 mg) and who present within 1 hr of ingestion. See Practical Procedures – Gastric Lavage. Give **activated charcoal 50 g**
2. If gastric lavage not indicated, give **activated charcoal 50 g** orally or via nasogastric tube to patients who present within 1 hr of ingestion

SUBSEQUENT MANAGEMENT

1. Correct metabolic acidosis (pH< 7.2) if accompanied by hypotension (systolic BP < 100 mm Hg), using 1.26% **sodium bicarbonate** (150 mmol/1) give 50 mmol over 30 min and repeat acid-base after a further 30 min
2. Treat ventricular arrhythmias with **sodium bicarbonate** 1.26% by IV infusion. Use anti-arrhythmic drugs only if arrhythmia accompanied by circulatory failure unresponsive to plasma expanders and inotropes. Use **lignocaine or phenytoin, NOT quinidine, procainamide or disopyramide.**
3. Use **diazepam** 5-10 mg IV for convulsions, but beware of respiratory depression.

MONITORING TREATMENT

1. Continuous ECG monitoring until heart rat <200 min, QRS complex returns to norma width, and

conduction defects resolve. Arrhythmias are unlikely to arise de novo 12 or more hrs after on overdose

2. If metabolic acidosis occurs, monitor acid-base hrly until abnormality returns to normal.

CONTENT

Publishing Medical Textbooks

Honorable lecturers and dear students,

The lack of quality textbooks in the universities of Afghanistan is a serious issue, which is repeatedly challenging the students and teachers alike. To tackle this issue we have initiated the process of providing textbooks to the students of medicine. In the past two years we have successfully published and delivered copies of 116 different books to the medical colleges across the country.

The Afghan National Higher Education Strategy (2010-1014) states: *"Funds will be made ensured to encourage the writing and publication of text books in Dari and Pashto, especially in priority areas, to improve the quality of teaching and learning and give students access to state-of- the-art information. In the meantime, translation of English language textbooks and journals into Dari and Pashto is a major challenge for curriculum reform. Without this, it would not be possible for university students and faculty to acquire updated and accurate knowledge"*

The medical colleges' students and lecturers in Afghanistan are facing multiple challenges. The out-dated method of lecture and no accessibility to update and new teaching materials are main problems. The students use low quality and cheap study materials (copied notes & papers), hence the Afghan students are deprived of modern knowledge and developments in their respective subjects. It is vital to compose and print the books that have been written by lecturers. Taking the situation of the country into consideration, we need desperately capable and professional medical experts. Those, who can contribute in improving standard of medical education and Public Health throughout Afghanistan, thus enough attention, should be given to the medical colleges.

For this reason, we have published 116 different medical textbooks from Nangarhar, Khost, Kandahar, Herat, Balkh and Kapisa medical colleges and Kabul Medical University. Currently we are working to publish 20 more medical textbooks for Nangarhar Medical Faculty. It is to be mentioned that all these books have been distributed among the medical colleges of the country free of cost.

All published medical textbooks can be downloadable from www.ecampus-afghanistan.org

The book in your hand is a sample of printed textbook. We would like to continue this project and to end the method of manual notes and papers. Based on the request of Higher Education Institutions, there is need to publish about 100 different textbooks each year.

As requested by the Ministry of Higher Education, the Afghan universities, lecturers & students they want to extend this project to the non-medical subjects e.g. Science, Engineering, Agriculture, Economics, Literature and Social Science. It is reminded that we publish textbooks for different colleges of the country who are in need.

I would like to ask all the lecturers to write new textbooks, translate or revise their lecture notes or written books and share them with us to be published. We assure them quality composition, printing and free of cost distribution to the medical colleges.

I would like the students to encourage and assist their lecturers in this regard. We welcome any recommendations and suggestions for improvement.

It is mentionable that the authors and publishers tried to prepare the books according to the international standards but if there is any problem in the book, we kindly request the readers to send their comments to us or authors to in order to be corrected in the future.

We are very thankful to German Aid for Afghan Children its director Dr. Eroes, who provided funds for 20 medical textbooks in previous two years to be used by the students of Nangarhar and other medical colleges of the country.

I am especially grateful to GIZ (German Society for International Cooperation) and CIM (Centre for International Migration & Development) for providing working opportunities for me during the past three years in Afghanistan.

In Afghanistan, I would like cordially to thank His Excellency the Minister of Higher Education, Prof. Dr. Obaidullah Obaid, Academic Deputy Minister Prof. Mohammad Osman Babury and Deputy Minister for Administrative & Financial Affairs Prof. Dr. Gul Hassan Walizai as well as the chancellor of Nangarhar University Dr. Mohammad Saber for their cooperation and support for this project. I am also thankful to all those lecturers that encouraged us and gave all these books to be published. At the end I appreciate the efforts of my colleagues in the office for publishing books.

Dr Yahya Wardak
CIM-Expert at the Ministry of Higher Education, March, 2013
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Message from the Ministry

of Higher Education

In the history, book has played a very important role in gaining knowledge and science and it is the fundamental unit of educational curriculum which can also play an effective role in improving the quality of Higher Education. Therefore, keeping in mind the needs of the society and based on educational standards, new learning materials and textbooks should be published for the students.

I appreciate the efforts of the lecturers of Higher Education Institutions and I am very thankful to them who have worked for many years and have written or translated textbooks.

I also warmly welcome more lecturers to prepare textbooks in their respective fields. So, that they should be published and distributed among the students to take full advantage of them.

The Ministry of Higher Education has the responsibility to make available new and updated learning materials in order to better educate our students.

At the end, I am very grateful to German Committee for Afghan Children and all those institutions and people who have provided opportunities for publishing medical textbooks.

I am hopeful that this project should be continued and publish textbooks in other subjects too.

Sincerely,

Prof. Dr. Obaidullah Obaid

Minister of Higher Education

Kabul, 2013

Book Name	Guideline for Treatment of Emergency Cases
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