Management of medical emergencies

— R B Dyer

Summary

Drawing on experience with emergencies presenting at King Edward VIII Hospital, this article provides a practical approach to the management of some of the commonest medical emergencies. It refers to recent advances in the treatment of Hypertensive crisis, Acute Pulmonary Oedema, Myocardial Infarction, Status Asthmaticus, Status Epilepticus and Hypoglycaemia.

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Curriculum vitae

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Acute medical emergencies embrace the whole range of internal medicine and require a sound knowledge of basic pathophysiology for the institution of rational management. This article draws on experience with emergencies presenting to the medical outpatients department of King Edward VIII Hospital, Durban, the major referral hospital for Natal and KwaZulu. It is intended to provide a practical approach to management of some of the commonest medical emergencies, with reference to some recent advances.

1. Hypertensive crisis

Prompt pharmacologic intervention undoubtedly reduces mortality and morbidity in the hypertensive emergency. However, overzealous treatment can lead to major complications such as stroke and renal failure.[1]

It is important to remember that it is not the level of blood pressure, per se, which determines the urgency, but rather the symptoms and signs of acute cardiovascular damage. Previously normal patients, such as those with acute glomerulonephritis or eclampsia, may develop a hypertensive crisis at relatively low levels of blood pressure eg 160/110, while patients with long-standing hypertension can have diastolic pressures of 150-160 mm Hg and be clinically well.

With these provisos, the clinical characteristics of hypertensive crisis can be summarized as follows:-

— diastolic pressure usually ⩾ 140 mm Hg
— fundal haemorrhages and exudates
— neurological abnormalities (headaches, confusion, somnolence, visual loss, focal deficits, seizures, coma)
— cardiac abnormalities (heaving apex beat, clinical cardiomegaly, acute left ventrical failure)
— nausea, vomiting and symptoms of azotaemia.

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The most important hypertensive emergencies are shown in Table I. Treatment is delayed only long enough for a brief history and physical assessment. This is primarily to exclude a catecholamine induced form of hypertension and to determine the extent of concomitant cerebrovascular and coronary disease which might modify therapy. Patients with hypertensive encephalopathy, phaeochromocytoma, major bleeding, aortic dissection and left ventricular failure should probably have their blood pressures reduced within minutes to hours. With accelerated hypertension, on the other hand, many authorities feel that reduction of pressure over 1 to 3 days is sufficiently rapid.

When one attempts to reduce the blood pressure within minutes then the aim should be a controlled reduction in pressure to a diastolic pressure of around 100 mm Hg.

Table 1: Hypertensive emergencies

Hypertensive encephalopathy
Drug related
— Drug withdrawal (clonidine, methylodopa).
— Drug-food interactions (guanethidine + tricyclic + tyramine).
— Drug-drug interactions (guanethidine + tricyclic antidepressants).
Hypertension complicated by:
— Aortic dissection
— Acute left ventricular failure
— Acute coronary insufficiency
— Cerebrovascular insufficiency
— Epistaxis and other bleeding (eg post-operative)
— Toxaemia of pregnancy
— Acute glomerulonephritis

Treatment of hypertensive crisis

Sodium Nitroprusside

This is the drug of choice in hypertensive encephalopathy being the most potent and reliably effective of the parenteral hypotensive agents. The drug directly relaxes arteriolar and venular smooth muscle, with the increase in venous capacitance reducing venous return to the heart so that, despite a modest increase in heart rate, the cardiac output is unchanged. Nitroprusside is administered by infusion pump with the dose being adjusted according to response. An incremental infusion in the dose range 0,25 — 8 ug/kg/min, increasing every 5 minutes, should control pressure without accumulation of cyanide (to which nitroprusside is broken down before being metabolised to thiocyanate in the liver). Caution should be exercised in renal failure and use of the drug requires intensive care facilities.

Dihydralazine methane sulphonate (Nepresol)

This drug, a direct vasodilator, is very effective when given as a slow IV bolus, dosage 6,25-25 mg. The response is less predictable than with nitroprusside and hypotension may result, but the drug is very useful in the small clinic or emergency situation. Use should be avoided in myocardial ischaemia.

Onset of action 5-10 min IV and 30 min IM.

Not the level of blood pressure per se, but rather the symptoms of acute cardiovascular damage, determines the urgency

Hydralazine Hydrochloride (Apresoline)

Dosage 10-40 mg IV or IM. Precautions as for dihydralazine; can also be given orally but parenteral use preferred in emergencies.

Diazoxide (Hyperstat)

The hypotensive effect of diazoxide is due to a

lowering of peripheral resistance by direct vasodilation of peripheral arterioles. Dosage 300 mg in IV bolus exposes patients to the risk of an excessive fall in pressure. To minimise this, it is probably safer to give smaller boluses of 100 mg every 5-10 minutes until the pressure falls.

Oral Agents

Nifedipine (Adalat)

This calcium channel blocker is of proven efficacy in hypertensive crisis when given sublingually in a dose of 10-20 mg. A gradual drop in blood pressure is produced within 5-10 minutes. The drug may cause side effects of tachycardia and flushing.

Captopril (Capoten)

There is evidence that sublingual captopril is effective in hypertensive emergencies.[2] Tachycardia and flushing are not a problem. Dosage 6,25 to 12,5 mg.

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2. Acute Pulmonary Oedema

Acute pulmonary oedema is almost always the result of acute left heart failure due to:

— Myocardial dysfunction (coronary artery disease, cardiomyopathy, myocarditis).
— Left ventricular systolic overload (hypertension, aortic stenosis).
— Mitral stenosis.
— Dysrhythmias.

Rarely, pulmonary oedema can result from central nervous system disturbances, pulmonary infection or infarction, inhalation of irritant gases, drowning and overloading the circulation with intravenous infusions.

Treatment of acute pulmonary oedema

1. Prop patient up.
2. Oxygen 40-100% (via nasal prongs if available).
3. Frusemide (Lasix) 40-100 mg IV
4. Morphine 10 mg IV slowly.
5. Aminophylline 250 mg IV over 20 minutes has a direct vasodilator effect on arteries and veins and increases myocardial contractility through a positive inotropic effect. Bronchospasm may be relieved. Toxic effects include hypotension, convulsions and cardiac arrhythmias.
6. Digoxin is valuable in the presence of supraventricular arrhythmias, particularly atrial fibrillation.
7. The underlying cause must be treated.

3. Acute myocardial infarction (AMI)

The acute management of this condition has undergone a dramatic rethink in recent years with the introduction of thrombolytic therapy.[3] The remarkable GISSI[4] trial on early use of streptokinase in AMI and the subsequent ISIS — 2 trial,[5] published in August 1988, have demonstrated unequivocally the benefits of this form of therapy.

In the latter study, at five week follow-up, there was a 26% reduction in mortality from AMI in those who had received streptokinase in the first 24 hours, a 20% reduction in those who had received Aspirin and a 40% reduction in those who had received both drugs. Those patients treated with both drugs inside 4 hours showed an almost 60% reduction in mortality.

Based on such compelling evidence it seems clear that modern management of the acute myocardial infarction should consist of:[6]

1. Pain relief — Morphine
2. Treatment of complications:
— left ventricular failure
— arrhythmias
— cardiac arrest
3. Immediate initiation of thrombolytic therapy in all patients seen in the first 24 hours, preferably inside 6 hours. This should consist of:
— Streptokinase 1,5 million units intravenously over one hour.
— Aspirin 160 mg daily for one month. The major contra-indications to streptokinase therapy are recent bleeding, surgery, recent cerebrovascular accident or uncontrolled hypertension. These contra-indications may not be as absolute as once thought.
4. The use of beta blockers has been shown to reduce mortality in a subset of patients with AMI but their routine use has not yet found widespread acceptance.

4. Acute severe asthma (Status asthmaticus)

This implies any attack of immobilising asthma which is unresponsive to usual modes of therapy, ie refractory to bronchodilator therapy. The condition carries a significant mortality rate which

has been shown to be due, in part, to a failure of doctors to appreciate gradually worsening airflow obstruction and a tendency to underestimate the severity of an acute attack. It has been observed, with justification, that "the best time to treat status asthmaticus is 3 days before it happens".[7]

Observations such as these, have led to the realization that the clinical symptology of asthma encompasses a spectrum ranging from completely normal to status asthmaticus (Figure I).

Figure I: Spectrum of symptoms in asthma

Normal (clinically and functionally)	Asymptomatic (but with functional abnormalities)	Symptomatic (with functional abnormalities)	Acute (severe asthma)

A patient may occupy a different point in this spectrum at any given time, and the goal of treatment is obviously to move the patient towards the "normal" end. It is thus very important to treat mild and moderately severe asthmatic attacks vigorously. The therapeutic regimen is fairly simple:[8]

1. B2 Stimulant Nebulisation

This is the preferred route of administration. Salbutamol solution (1 ml) is added to water or saline (4 ml) and the patient is nebulised. Repeat in 30 minutes if relief incomplete.

2. Aminophylline infusion

This can be used as first line therapy or as an adjunct to nebulisation with a B2 stimulant. Aminophylline 5-6 mg/kg weight is infused over 15 minutes to avoid toxicity. If a theophylline containing preparation has been taken in the last 24 hours, then the loading dose should be halved.

3. B2 Stimulant IV

Not recommended as first line therapy as tachycardia and aggravation of ventilation-perfusion inequalities in the lung may result. Only use as first line therapy if other drugs are not available.

4. Adrenaline

Has been used in acute asthma for over 80 years. It should be used with caution and only in the young with no cardiovascular abnormality and a

pulse rate 100/min. Administration is subcutaneous (1 ml Adrenaline 1:1000).

Due to inadequate therapy, or despite therapy, there is a subpopulation of asthmatics who will progress to acute severe asthma. There are a number of clinical clues which are helpful in identifying this group. Patients who have a history of severe asthma or have required previous hospitalisation for status asthmaticus, patients who have required corticosteroid therapy in the past and patients who describe failure of usually effective therapy, are all at increased risk of developing acute severe asthma. Warning signs of a potentially fatal attack include:

— respiratory rate > 20/minute
— pulse rate > 120/minute
— pulsus paradoxus
— cyanosis and confusion
— hypercapnoea
— peak flow < 120 1/minute
— pneumothorax.

Asthma can be staged according to blood gas estimation as shown in Figure 2.

Figure 2

	paO_2	$paCO_2$	pH
1. Mild	N	↓	↑
2. Moderate	↓	↓	↑
3. Severe	↓ ↓	↓	↑
4. Very severe ...	↓ ↓	N	N
5. Life threatening	↓ ↓	↑	↓

Treatment of Acute Severe Asthma

1. Humidified oxygen per mask.
2. Adequate fluid replacement intravenously.
3. Careful examination to exclude pneumothorax (particularly if there is sudden, acute deterioration).
4. Nebulisation with a B2 stimulant.
5. Aminophylline loading dose intravenously (5-6 mg/kg body weight) over 15 minutes. Commence aminophylline infusion of approximately

1 mg/minute.
6. Hydrocortisone 200-400 mg IV push. If not available then predisone 40-60 mg orally should be given.
7. If improvement is not *rapid* then the patient should be transferred to a centre where intensive management facilities are available.

Status asthmaticus carries a significant mortality rate, mostly due to doctors underestimating the severity of an acute attack.

5. Status Epilepticus (Grand Mal Status)

This condition carries a mortality rate as high as 30% in some series and has a substantial long term morbidity.

Complications may occur due to:

— Loss of normal protective movements resulting in burns, drowning, traumatic injuries.

— Vomiting with aspiration and pneumonia.

— Hypoxic brain damage with cerebral oedema. The hypoxic brain damage is thought to be due to a combination of factors. Disorganised neuronal activity leads to an increase in cerebral oxygen consumption. This, coupled with poor respiratory motions and upper airway obstruction, due either to secretions or the tongue, leads to hypoxic brain damage with cerebral oedema.[9]

Aetiology of Status Epilepticus

Patients with status epilepticus not infrequently have a definable aetiology or a reversible precipitant. Thus, symptomatic epilepsy is more likely to lead to status epilepticus than is idiopathic epilepsy. Medication non-compliance among known epileptics is another frequent cause of status.

Management of Status Epilepticus

General Measures

1. Ensure adequate airway.
2. Measure vital signs.
3. Intubate if necessary. The precise timing of endotracheal intubation is debated, but the need becomes more pressing the longer the attack continues and the more respiratory depressant drugs are given.
4. Look for an underlying cause.

Specific Drug Therapy

1. Diazepam 10 mg IV bolus (5-20 mg). Give at 2,5 mg/minute.
Beware of respiratory depression, especially if given with phenobarbitone.
2. Phenytoin 50 mg/minute to maximum of 1 g. Beware of hypotension, tachycardia and respiratory depression (rarely). Dose 15-20 mg/kg.
3. If these measures fail, then it may be necessary to paralyse and ventilate the patient.

6. Hypoglycaemia

Hypoglycaemia is frequently seen in patients presenting to an emergency department. Non-specific symptoms and signs ranging from mild feelings of discomfort to coma and generalised seizures, are common, in these patients. Thus, a high index of suspicion is required and blood glucose estimation is mandatory in all such patients.

The clinical manifestations of hypoglycaemia are related to glucose deprivation of the CNS and/or the physiologic effects of cathecholamine release due to neuroglycopaenia.[10] CNS manifestations are confusion, weakness, inco-ordination, blurred vision, diplopia, headache, focal signs, coma and seizures. Adrenergic effects include sweating, tachycardia, palpitations and pallor.

Hypoglycaemic states can be classified as fasting, postalimentary and drug-induced hypoglycaemia. Fasting hypoglycaemia is commonest in children while postprandial hypoglycaemia more commonly effects adults. However, it is drug-induced hypoglycaemia, mainly due to insulin, oral hypoglycaemic agents or alcohol, which is most important.

The best time to treat status asthmaticus is 3 days before it happens!

Management

After confirmation of a diagnosis of hypoglycaemia, immediate treatment should be commenced.

1. *Intravenous* glucose 25-50 g. Practically this means 2 ampoules of 50% glucose (20 mls/amp).
2. Follow this with an infusion of 5% or 10% glucose solution (rates of 5 mg/kg/minute are adequate in adults).

3. If venous access is a problem then glucagon 1 mg/hr may be useful. However, in alcohol induced hypoglycaemia, glycogen stores are depleted. Indeed, hypoglycaemia may be enhanced in this situation by stimulating insulin release.[11]

4. In alcohol induced hypoglycaemia, thiamine should be added to the dextrose infusion to prevent the development of Wernwicke's encephalopathy.

Medication non-compliance among known epileptics is a frequent cause of status

In dealing with emergencies as these, one should always bear in mind that personal, continuing, primary care is an effective way in preventing the emergencies discussed here. How else can one manage status asthmaticus three days before it occurs!

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