# Medical emergencies in rural practice

*Garry Nixon*

*Correspondence to: tigerhill@clear.net.nz*

### Vignette 1

*62-year-old man. History of hypertension. Presents with episode of syncope on standing. No chest pain or SOB. Initial BP 86/60 improves to 110/70 without treatment. Persistent tachycardia 112 bpm. JVP elevated. Chest clear. 02 sat 94% on room air. Bedside troponin and D-dimer both positive. CXR and ECG unremarkable.*

*Anticoagulated and transferred to base hospital for CTPA which confirms a large saddle pulmonary embolus. Treated with fibrinolytics.*

### Vignette 2

*69-year-old lady with history of diverticular disease, IHD, hypertension and smoker. On nifedipine, losartan and aspirin. Presented with 24 hrs of vomiting and lower abdominal pain. P 90 and BP 78/58. Normalised to 145/85 with iv fluids and remained stable. JVP not documented. Tender + lower abdo. No peritonism. No pulsatile masses felt. Treated as diverticulitis. Clinically stable. Hb fell from 127 to 65 over 72 hrs. USS confirmed leaking AAA.*

### Vignette 3

*84-year-old lady. Previous hx of AVR and repair thoracic aneurysm, hypertension, DVT. On warfarin, enalapril, bendrofluazide. Presented with R sided pleuritic chest pain, palpitations. Initial BP 80/50 but rapidly resolved to 130/80. P 67. Initial JVP not documented. CXR no change from old films. INR 2.8. D-dimer positive. Managed initially as probable PE. Following day sudden onset of epigastric and R flank pain. BP dropped to 43/35 P 100. Initially thought was further large PE. But JVP low. Urgent US, no obvious source of blood loss. Repeat CXR widened mediastinum and bleeding into L lung – dissecting thx aneurysm. Not candidate for surgical repair – managed medically. Anticoagulation reversed. BP responded to fluids, after which GTN infusion and beta blockers used to keep systolic BP around 110mmHg.*

### Vignette 4

*72-year-old man. Normally good health. Sudden onset severe generalised abdominal pain.*

*Pale. P 110 106/75. JVP down. Generalised abdominal tenderness. No pulsatile mass. Presumed AAA. Initial Hb 148. Hb 2 hours later 184. Urgent US – free fluid ++ in abdo. Dx Bowel infarction. Several litres of iv fluid prior to and during transfer.*

### Vignette 5

*Female early 60s. Mild treated hypertension only. Metoprolol CR 23.75 mg daily. Very sudden onset sharp chest pain and then collapse. P 40 BP 90/50. ECG 1mm lateral ST depression and lateral T wave inversion. CXR NAD. Troponin negative and D-dimer. BP and P normalised. Managed as ACS with heparin and GTN infusion.*

*6 hrs later BP suddenly dropped to 60/30 P 80. JVP elevated. New diastolic murmur. Marked difference in BP between arms. Repeat ECG unchanged. Repeat CXR widened mediastinum and increased heart size. Pericardial effusion noted on US. Dx thx aneurysm involving the aortic valve, coronary arteries with pericardial tamponade. Transferred urgently for surgical repair.*

***Garry Nixon*** *is a senior lecturer in the Department of General Practice, Dunedin School of Medicine, where he convenes the postgraduate diploma in rural hospital medicine. He also works as a medical officer at Dunstan Hospital, Clyde, Central Otago. He has a particular interest in the management of cardiology problems in the rural setting.*

Some things have got easier. The advent of courses like EMST and clinical guidelines has made the management of trauma and conditions such as acute coronary syndrome almost formulaic. We now approach them systematically, secure in the 'ABCs' we have learnt and 'recipes' we have at hand.

There is, however, a group of serious emergencies that continues to

trouble those of us working in rural hospitals and practices. They tend to be the catastrophic problems that occur deep in the abdomen and chest, often as a major blood vessel bleeds or blocks.

These conditions are notorious for not following the rules; the symptoms and signs they throw up are neither specific nor sensitive. The same applies to investigations we may have on hand like ECGs/CXRs or bedside troponins and D-dimers. It is not surprising that 38% of patients with a dissecting thoracic aneurysm are initially suspected of having another condition, often acute coronary syndrome; 70% of pulmonary emboli are diagnosed at post mortem; or 25% of patients admitted with a diagnosis of non infective exacerbation of COPD have in fact had a PE.[1,2,3,4]

William Osler correctly observed that, '*There is no disease more conducive to clinical humility than aneurysm of the aorta.*' Pulmonary embolus deserves its title as 'the great masquerader'.

Because of the consequences of missing these conditions we feel compelled to 'rule them out' once considered, even if they are well down on the differential diagnoses. When this involves transferring to a base hospital for complex investigations (and having to justify this to the referral centre on the basis of very non specific findings) there is a strong incentive to not even consider them!

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In those cases in which it is obvious something serious is going on, it can be hard to decide exactly what is happening, but knowing what is happening is important in order to provide appropriate initial management. Anticoagulants (and even less so fibrinolytics) are not that helpful if someone is actively bleeding from an aneurysm!

Our urban colleagues also face these difficulties. But they have access to immediate imaging, particularly CT, which often provides them with a definitive diagnosis. As rural generalists we have to rely on the indirect clues offered by a careful history, (repeated) examination and the simple investigations on hand.

This article doesn't repeat what is contained in the textbooks but looks for additional approaches that may help avoid missing these problems in the rural setting.

Because they frequently cause some haemodynamic compromise, assessing volume carefully and thinking about the nature of fluid shifts can provide some vital clues.

Onset is sudden; usually very sudden; more sudden than the onset of cardiac pain or the other conditions we often confuse with PE. An abrupt onset is the most specific characteristic of the pain of dissecting aortic aneurysm.[4]

The haemodynamic effects also occur suddenly. However by the time we assess the patient they may have compensated and be normotensive. Beware of a history of syncope or near syncope close to the onset of other symptoms, even more so if it is postural. While syncope is a common and often benign presentation it can be a clue to a sinister underlying haemodynamic problem. Be more suspicious if there is not a complete recovery (lowish BP, or even a normal BP in a normally hypertensive patient, persistent tachycardia or a significant postural drop) or it is accompanied by other symptoms such as back, chest or abdominal pain or SOB.

Twenty per cent of patients of PE present with syncope.[1] These patients are more likely to have a large life threatening embolus sitting in a major pulmonary artery. They are less likely to have a distinct pulmonary infarction, and therefore often lack the more specific symptoms of pleuritic chest pain and haemoptysis.

Do not be fooled by the absence of a tachycardia. Supine tachycardia is an insensitive sign of volume loss. Elderly people in particular sometimes do not mount a tachycardic response to shock and are not infrequently bradycardic. Dizziness so severe that it prevents the patient from standing or a postural increase in pulse rate of >30bpm are more sensitive and specific.[5]

Carefully assess the JVP. It will give you vital information, alerting you to a loss of volume if it is down or an obstructive problem or pump failure if it is up. I am not embarrassed to say I can find looking for the JVP difficult. It takes time and care; particularly if it is very low and only seen when the patient is flat or very high and only seen when they are sat up at 90 degrees. But even in an emergency, if the diagnosis is at all unclear, it is worth taking the time to assess it. The result may surprise you and set you off on a completely different diagnostic and management track.

It is easy to understand why it is assumed many of these patients are having an MI. The spectrum of presentations is very similar. The pain associated with a dissecting thoracic aneurysm is slightly more likely to be felt in the chest than the back, despite the classic teaching.[4] PE also masquerades as an acute coronary syndrome, SOB and chest pain both being non specific.[6]

The value of obtaining a history of pain that is sudden in onset has already been mentioned. Another discriminating feature of the pain due to a thoracic aneurysm, if present, is its migratory nature as the aneurysm steadily dissects distally; initially mistaken for an MI, then cholecystitis, then renal colic, even diverticulitis or limb pain.[4] It is perhaps not surprising that the more distal the pain at the time of presentation, the less likely the admitting doctor is to correctly suspect an aneurysm.[4] Some authors suggest the pain is often pulsatile in nature.[7]

Examination can be unremarkable and consequently unhelpful. Having said that, some signs, when present, are highly specific for aortic dissection. But you will only find them if you look carefully. These include the diastolic murmur of aortic incompetence (caused by the aneurysm dissecting into the aortic valve and present in 25% of patients) or a pulse deficit (present in 38% of patients).[4,7]

The simple investigations we have on hand in the rural setting can be equally confusing. Fifty per cent of patients with a thoracic aneurysm will have T wave and ST segment ECG changes, though ST elevation is relatively rare.[7] Similarly a PE can produce the same non specific ECG changes.[1] Fifty per cent of patients will have the classic mediastinal widening of a dissecting aneurysm, however a normal CXR is not unusual.[4] The majority (over 80%) of patients with a PE will have an abnormality on CXR but it is likely to be minor and non specific.[8] Linear atelectasis and small effusions are the most common.[1] Fifty per cent of patients with a PE will have a positive troponin, suggesting a large, more serious, PE.[11,6] D-dimers are even less specific. While a negative D-dimer has a role in ruling out PE, anything that activates the fibrinolytic system (bleeding, infection, inflammation, MI) or even increasing age can give rise to a positive D-dimer.[6,9]

Again it is worthwhile thinking about the haemodynamics. Unless the patient has severe pre-existing heart damage, an MI will cause haemodynamic problems only when a large territory of myocardium is ischaemic (or as the result of an arrhythmia). In either case the ECG will be grossly abnormal. Think again if the ECG changes are minor, non specific or involve a small territory. Differentiate between a left heart problem (wet lungs +/- elevated JVP – much more likely to be a primary cardiac problem), and a right heart problem (elevated JVP and a clear chest – more likely obstructive – PE, cardiac tamponade or tension pneumothorax; though inferior MI causing isolated R ventricular infarction could be a possibility, something that should be obvious on the ECG).

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Unfortunately the problem will sometimes be missed at the time of the initial assessment and the patient may end up being managed locally, it being falsely assumed they have a much less serious problem. It is therefore important to fully reassess them, whenever their condition changes. This means thoroughly re-examining them and even returning to the presenting history. It can also mean repeating blood tests, ECGs and CXRs. For example the vital clue of a new diastolic murmur may only become obvious later in the course of the illness. You will only hear it if you listen specifically for it each time you reassess the patient. The mediastinum may also appear normal on the first CXR but may obviously bulge or widen with later views.

Serial monitoring of Hb may not only alert you to ongoing volume loss it can indicate the nature of the loss. This has important implications for management. If there is active bleeding from a site that can only be controlled surgically you will want to be cautious with fluid replacement. Indeed if you suspect an aneurysm the patient should be kept relatively hypotensive; target systolic BP 110mmHg.[10] Morphine, intravenous beta-blockers and possibly a GTN infusion are probably the best agents to achieve this in the rural hospital. If, however, the patient is losing just salt and water from the intravascular space (manifested as rising Hb as in vignette No.4), much more aggressive fluid resuscitation is indicated.

There are pitfalls in the 'rule out an MI' approach to the patient with chest pain. Picking up those patients with other serious problems occurring deep in the chest and abdomen in the rural setting remains a challenge that requires attention to detail and a willingness to repeatedly reassess the patient. Thinking about the haemodynamic effects and the nature of fluid shifts can provide vital clues.

### Competing interests

None declared.

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