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Letters to the Editor

Therapeutic implications of diastolic dysfunction in heart failure

Sir,

I read with interest the article by Dean and Poole-Wilson on diastolic dysfunction in heart failure.[1] As was alluded to in the quotation cited at the beginning of the article, Henderson[2] should be credited not only with having distinguished diastolic dysfunction from systolic dysfunction of the heart as far back as 1923 but for having identified the mechanism of heart failure in the aging or aged heart, that is, impaired diastolic function.

Morphological changes in the aging or aged heart can be either expected, e.g. calcific deposits,[3] increased subepicardial fat,[3] nondilated left ventricular cavity,[3] increased ventricular thickness,[4] and increased heart weight,[3,4] or unexpected, e.g. massive cardiac amyloidosis,[3] severe aortic valvular stenosis,[3] hypertensive hypertrophic cardiomyopathy,[4] and left ventricular hypertrophy secondary to systemic hypertension.[4] All may lead to impaired diastolic relaxation of the heart and thus cause diastolic heart failure.

Recognition of diastolic heart failure in elderly subjects not only is important from the diagnostic standpoint but also, as the authors pointed out, has significant therapeutic implications. For example, traditional drugs employed in the treatment of congestive heart failure, such as inotropic agents, diuretics and arterial vasodilators, may not only be ineffective but even prove harmful to such patients.[5]

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Withdrawal of maintenance digoxin from institutionalized elderly

Sir,

I read with interest the article by Macarthur[1] entitled 'Withdrawal of maintenance digoxin from institutionalized elderly'. He states that withdrawal of maintenance digoxin is not indicated in patients with a history of atrial dysrhythmia. However, there is some evidence that pretreatment with digoxin does not reduce the frequency of the paroxysms of atrial fibrillation and can result in longer attacks.[2] The latter is consistent with the action of digoxin in reducing the atrial refractory period and thus reducing the likelihood of reversion to sinus rhythm.[2] In those patients where paroxysms of atrial fibrillation are more likely to occur when vagal activity is high, the additional vagotonic effect of digoxin could result in an increase in the frequency of attacks.[3]

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Stroke due to carotid artery dissection

Sir,

Dissection of the carotid artery is a recognized cause of stroke,[1] but it is rarely discussed in journals and textbooks.[2] We report a patient presenting with fluctuating stroke, in whom the diagnosis was made only after considerable delay.

A 41 year old woman developed a coughing bout, followed by a sharp pain on the right side of her neck and face. She then noticed left-sided weakness, and went to a casualty department. She was allowed home with no definite diagnosis, and later consulted her general practitioner (GP) who also took no further action. Her symptoms were fluctuating, and one doctor thought she was 'hysterical'. Later, her family drove her 60 miles to her mother's home intending to consult the local GP. On the way, her symptoms worsened and she was brought to the nearest hospital, where the on-duty doctors noted signs of a left hemiplegia, but a 'strange personality' was recorded, and hysteria again considered a possibility. She was admitted, and her hemiplegia resolved completely over the next few hours. The following morning, however, she was densely hemiplegic, with left facial weakness and a right Horner's syndrome. A diagnosis of subdural haematoma was considered and she was transferred to a neurosurgical unit, where a computed tomographic scan confirmed right-sided cerebral infarction. Doppler/duplex ultrasound showed reduced internal carotid artery flow suggestive of an obstructive lesion, though no significant atheroma was noted. Subsequent arteriography showed elongated narrowing of the right cervical carotid artery consistent with dissection. She was anticoagulated, and, following a prolonged period of physio-

therapy, she made a good recovery, though still had some permanent disability.

Spontaneous dissection of the carotid artery was first described in 1959,[3] though traumatic dissection has been known for longer.[1] Common presenting features are abrupt unilateral pain in the neck, face or head, hemiparesis, ipsilateral Horner's syndrome, pulsatile tinnitus, and bruit. Angiographically there is irregular narrowing of the cervical carotid artery ('string sign'). Although the overall prognosis is good,[1,4] diagnosis is important as outcome is improved with anticoagulation. In our patient, coughing appears to have precipitated the dissection. The considerable diagnostic delay was largely due to inadequate history-taking, and the possibility of carotid dissection not being considered.

Carotid dissection is probably underdiagnosed, and may indeed be one of the commonest causes of stroke in young patients.[5] The condition is well recognized by neurologists, but often not by physicians, even though it is these latter doctors who deal with most stroke disease. Carotid artery dissection should therefore be suspected especially in younger patients presenting with transient cerebral ischaemia or stroke, associated with unilateral facial, neck pain or headaches. A history of even trivial trauma such as coughing, vomiting or sneezing should further alert the physician, as should the lack of other obvious causes.

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Rare presentation of gastric leiomyosarcoma

Sir,

Gastric leiomyosarcomas are rare tumours and account for 1–3% of all gastric cancers.[1] The presenting features are non-specific and these tumours often present at an advanced stage.[2] We describe a patient with gastric leiomyosarcoma whose main presenting feature was a pyrexia of unknown origin.

A 47 year old male presented with a 2-week history of left sided neck pain, tiredness, headaches, sweating and rigors. His temperature on admission was 40.5°C and physical examination demonstrated no obvious focus of infection. The positive findings on routine blood tests were mild anaemia (haemoglobin 10.8 g/dl), a platelet count of 638×10^9/l, a white cell count of 13.5×10^9/l (70% polys), and an ESR of 54 mm/h. Chest X-ray revealed consolidation in the left lower lobe with slight elevation of the diaphragm. A computed tomographic (CT) scan of the brain was normal, as was lumbar puncture. Blood cultures, however, grew an anaerobic streptococcus and the patient was commenced on intravenous erythromycin. His temperature settled and he was discharged home 3 weeks later.

He was admitted 6 days later with a pyrexia, sweating and rigors. Blood cultures were negative on this occasion. Abdominal examination was unremarkable but ultrasound revealed a mass in the left hypochondrium. CT scan confirmed the presence of a 12 cm mass lying above and medial to the spleen extending to the left hemidiaphragm. Ultrasound guided needle biopsy of the left upper quadrant mass revealed clusters of spindle-shaped cells suspicious of malignancy.

A laparotomy was performed and revealed a large inflammatory mass in the left upper quadrant. This was adherent to the dome of the diaphragm, the spleen, the tail of the pancreas and originated from the fundus of the stomach. The mass was excised with the attached stomach wall, the tail of the pancreas and the spleen. Histology showed that the mass had a necrotic centre with malignant spindle-shaped cells, diagnostic of gastric leiomyosarcoma. His post-operative recovery was complicated by a left basal pneumonia which settled on antibiotic therapy. At discharge he was apyrexial and was well at follow up 4 weeks later.

This is the first reported case of a gastric leiomyosarcoma presented with a pyrexia of unknown origin.[3] The commonest presenting features in patients with gastric leiomyosarcoma are gastrointestinal bleeding, abdominal mass and weight loss. Septicaemia may complicate peptic ulceration due to venous invasion and it is possible that the same mechanism was responsible for the pyrexia in the present case.[4]

The initial presenting symptoms in this case were non-specific and did not implicate the gastrointestinal tract. This resulted in a delay in diagnosis. Preoperative diagnosis is unusual and histological confirmation of gastric leiomyosarcoma was made in only 8% of cases in a recent review.[3] We suggest that a diagnosis of gastric leiomyosarcoma should be considered in patients with a Gram-positive septicaemia and a solid mass in the left upper quadrant.

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