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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-