

elucidated, hypotension probably results from sudden reduction or cessation of sympathetic activity. Endogenous opioid peptides, released with ACTH from a common precursor in the pituitary and central nervous system, may inhibit sympathetic tone. Plasma endorphins have previously been demonstrated to rise in patients with vasodepressor syncope, and our aim was to examine whether this rise is a primary or secondary phenomenon.

We studied 16 consecutive patients (mean age 61.6 yrs; range 18-80) undergoing tilt-test for unexplained syncope. Regular medication was continued. All tests performed in fasting state, with 70° tilt for up to 40 mins. Blood samples for beta endorphin and ACTH were obtained at baseline (BASAL), and then every 10 mins, at the onset (SYNC) and following syncope. Beta endorphin and ACTH results expressed as mean (SD) pmol/l.

Eight patients were asymptomatic throughout the test and demonstrated no change in plasma endorphins [3.2 (1.0)] and ACTH [3.0 (0.9)].

Vasodepressor syncope was induced in 7 patients, but in 2 patients adequate blood samples could not be obtained prior to syncope. A marked increase in plasma endorphins preceding syncope occurred in 4 of these 5 patients [BASAL 3.9 (1.1), SYNC 8.9 (2.6)]. ACTH rose in parallel with endorphins in these 4 patients [ACTH: BASAL 3.0 (0.8), SYNC 7.5 (4.7)]. The fifth patient (female; 23 yrs) showed no rise in endorphins: [BASAL 5.3; SYNC 3.9] or ACTH [BASAL <3.0; SYNC <3.0]. All 7 patients had an increase in plasma endorphins [27.3 (13.2)] and ACTH [41.0 (20.6)] post syncope.

The one remaining patient developed presyncopal symptoms at 35 mins, without a fall in blood pressure, and showed an increase in endorphins similar to the syncopal group [BASAL 5.4; presyncope 10.1].

Conclusions: Endogenous opioid mechanisms appear to be implicated in the pathophysiology of vasodepressor syncope. An increase in plasma beta endorphins precedes syncope and this observation may have important therapeutic implications.

74 NON-INVASIVE MONITORING OF RENAL TISSUE OXYGEN SUFFICIENCY FOR TRANSPLANTATION

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Storage of organs prior to transplantation results in oxygen deprivation and substrate depletion, dependent upon the duration of the ischaemic period. Ischaemic/reperfusion damage to kidneys is commonly associated with medullary congestion but it is known that the superficial renal cortex is reperfused sufficiently to permit normal tissue oxygenation immediately after transplantation.

We have assessed the mitochondrial redox state in the insitu transplanted kidney and these findings indicate that extended storage results in damage to the respiratory chain.

The study consisted of 2 groups (n=6) of rabbits. In group 1 one unstored kidney was autografted into nephrectomised recipients. In group 2 one 72hr stored (4°C) kidney was autografted into nephrectomised recipients. Two non-invasive methods were used prior to and up to 4 hr post transplantation: 1) surface fluorimetric (SF) measurements of cortical mitochondrial NADH; and 2) near infra-red spectroscopic (NIRS) measurements of cytaa3, the terminal enzyme of the respiratory chain. These were correlated with functional outcome and histological findings.

During the first 10 min of reperfusion cytaa3 became more reduced, markedly in group 1 followed by a slow and progressive reoxidation over the next 4hr in both groups. Significant levels of NADH were measured in both groups prior to reperfusion. In group 2 100% oxidation to NAD occurred within 20 min of reperfusion (5/6), whereas in group 1 a maximum of 70% oxidation was observed over the 4 hr. On reduction of pO₂ the NADH level in group 1 returned to the initial value (6/6); however in group 2 the NAD did not become reduced even after 20 min (5/6). The data is consistent with mitochondrial dysfunction in the stored group and is correlated with poor renal function and medullary congestion. These findings suggest that these measurements could be applied to assess kidney function on an ex-vivo circuit prior to transplantation.

75 DISABILITY AMONG ACUTE ELDERLY ADMISSIONS

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There is controversy about the interface between General Internal Medicine and Geriatric Medicine. At least three admission policies exist (age-related, needs-related and integrated), each aiming to channel disabled patients to geriatric medicine. However, little is known about the prevalence of disability. We therefore studied acute cases using a standard activities of daily living (ADL) measure.

Subjects were consecutive elderly patients admitted acutely to general internal medicine and geriatric medicine. Patients were assessed within 48 hours of admission (most within 24 hours). Data collected included demographic details, problems, diseases (coded using ICD-9 and grouped into broad categories), a measure of previous health status and Barthel ADL Index (BAI) score. The BAI is a measure of disability, covering primary ADL (walking, continence, self-care) and scores range from 0 - 20 (totally dependent - independent).

There were 311 patients with a median age of 80 years (range 65-99). 55% were female. Median BAI score was 13 (range 0-20). About a quarter (26%) were severely disabled (BAI > 8), whilst 14% were independent (BAI = 20). All ADL were affected. For example, 16% of patients had faecal incontinence, 25% urinary incontinence, 38% dependence in toileting, and 54% were unable to walk independently. Older patients were more disabled; median BAI for over 75s was 11, vs 16 for the under 75s (p<0.001, Mann-Whitney U test). Disability was worse among females (median BAI 11.5 vs. 15 for men, p<0.01). This was an age effect. Among the groups of diseases, disability was worse among those with stroke (median BAI 7 vs. 13 for those without stroke, p<0.001), and dementia (median BAI 8.5 vs. for those without dementia, p<0.001). Disability was lower among those with cardiac disease, especially ischaemic heart disease. Disability was higher among patients whose previous health status was worse and for those who had "geriatric problems". Few of these associations were age-dependent.

These results are the first direct UK evidence of high levels of disability among elderly admissions, indicating a need for the assessment and early management of disability. The associations above may indicate ways of targeting such interventions including the involvement of specialists in geriatric medicine.

76 THE POTENTIAL APPLICABILITY OF DRUG THERAPY FOR ACUTE STROKE

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Effective drugs for acute ischaemic stroke may soon be available, but current clinical trials are confined to highly selected cases. To determine the proportion of strokes eligible for therapy, we analysed the data of consecutive cases. **METHODS:** All strokes (World Health Organisation definition) admitted to a district general hospital were assessed clinically and by the National Institutes of Health (NIH) neurological scale. 25% had a CT scan within a week. **RESULTS:** Of 514 strokes in 14 months (mean age 76ys; 230 male), the numbers with exclusion criteria stipulated by current trials were:

Delay >12 hours (stroke onset to ward)-	196(38%)
Minor stroke (arm & leg NIH score <2)-	124(24%)
Pre-existing major neurological deficits-	95(18%)
Unconscious or very drowsy (NIH >2)-	57(11%)
Other severe illness present-	47(9%)
Haemorrhagic stroke-	23(4%)
Brain stem stroke-	20(4%)
Major ECG abnormalities-	19(4%)
Abnormal electrolytes, hypotension, recent myocardial infarction-	17(3%)

None of these features were found in 33(6%), while 106(21%) had >2. **CONCLUSIONS:** If current selection