accepted at most transplantation centres because of decreased renal function in elderly subjects, and possibly because of an increasing risk of undiagnosed malignant or other systemic diseases. The normal changes in renal anatomy and physiology associated with advancing age have been well described, and renal function reserve declines linearly with time after age 30, thus effectively shortening the useful life of the donor organ.[3] Although initially results with older donors were not as good as with younger donors, satisfactory function and survival rates have now been reported with donors over age 55.[1,4-6] In our experience older donors, living and cadaveric, may be used with confidence if the donor's renal function was normal and proteinuria or diabetes were not present. The use of organs from older donors, especially in elderly recipients, may be enhanced by minimising other potential risk factors such as long ischaemic times, and possibly by the use of cyclosporin A monotherapy as light immunosuppressive therapy.

J. M. CAMPISTOL
M. MAÑALICH
J. ANDREU
F. OPPENHEIMER
M. J. RICART
J. VILARDELL

Renal Transplant Unit,
Hospital Clinic,
Barcelona 08036, Spain

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INFANTS AND VERY YOUNG CHILDREN AS KIDNEY DONORS

SIR,—We cannot agree with Dr Ruder and colleagues (July 15, p 168) that kidneys of donors younger than three years of age should not be used for transplantation and that kidneys of donors aged 3–6 years carry an increased risk of graft failure.

The mean waiting time on dialysis for a renal transplant candidate averages more than 3 years at centres in the Eurotransplant Organisation. In 1986, in the same programme, 251 (3%) patients died while waiting for transplantation.[1] Therefore, all potential grafts should be used even if the kidneys come from brain-dead children.

Between June, 1963, and June, 1989, 1840 renal grafts were done at our institution; among them, 39 were from donors aged 0–6 years. 20 recipients were treated by conventional immunosuppressive regimen (Conv) while 19 had cyclosporin therapy (CsA). The 3-year graft survival was 49% in the Conv group but was 79% in the CsA group. From the start our general policy has been to use en-bloc kidneys from donors less than 2 years old whereas for older donors, a single kidney is usually given to one recipient. However, there were some exceptions to this rule. During the CsA period, 5 recipients (mean age 23·2 [SD 5·5] years) received en-bloc kidneys (donor age: 2·5 years, 4·5 years, and 17, 20, and 20 months), and 14 (mean age: 31·8 [3·8] years) received a single kidney (donor age: mean 36·2 [5·2] months; range 17–72). 3-year graft survival rates were 80% in the en-bloc kidney group and 78% in the single kidney group; acute tubular necrosis rate was 0% in the former and 28% in latter group.

Despite its small size, this series shows that excellent results can be achieved with kidneys from donors less than 6 years old. In our hands, graft survival reaches the percentage observed by Ruder et al for renal grafts from donors more than 6 years old.

J. P. SQUIFFLET
P. GIANELLO
S. NESA
G. P. J. ALEXANDRE

Department of Renal and Pancreatic Transplantation,
University of Louvain Medical School,
B-1200 Brussels, Belgium

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UNIVERSITY OF WISCONSIN PRESERVATION SOLUTE AND BRADYARRHYTHMIA

SIR,—Dr Prien and colleagues (June 10, p 1319) report the occurrence of bradyarrhythmia after release of the vascular clamps in 20 recipients of a cadaveric kidney preserved with University of Wisconsin (UW) solute. Since we started using kidneys preserved with this solute, we have seen 4 cases of well-documented severe bradyarrhythmia (20 per min or less) at the time of reperfusion of the transplanted kidney, which resulted in an abrupt fall of blood pressure. In one patient a grade III atrioventricular block was seen on the electrocardiogram. In 2 patients the cardiac arrhythmia resolved spontaneously within seconds, whereas in the other 2 atropine and/or isoprenaline was needed. In 3 patients a sharp increase in serum potassium to 7·5 mmol/l was recorded. Up to now, only 2 centres (Munster and Leuven) using UW solute within the European collaborative study have reported this occurrence (Ploeg R, personal communication). We can only guess why this problem has not been reported by others. In Munster as well as in Leuven all kidneys are carefully flushed again with freshly prepared UW solution before reimplantation. This process could result in a supplementary load of fresh adenosine, the constituent proposed to account for the bradyarrhythmia. In centres where reflushing is not done routinely, the amount of active adenosine entering the circulation could be lower.

Because of the potential hazard of these UW-solute induced cardiac disturbances, especially in high-risk cardiac patients, UW-preserved organ recipients should be carefully monitored.

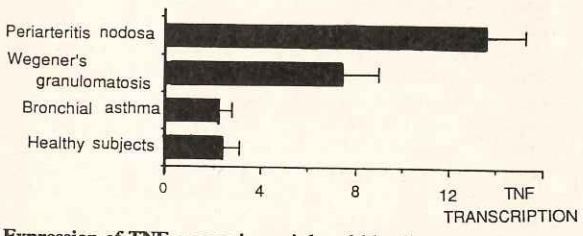
Y. VANRENTERGHEM
L. ROELS
E. VANDERMEERSCH
R. CHRISTIAENS
J. GRUWEZ
P. MICHIELSEN

Leuven Collaborative Group for Transplantation,
University Hospital Gasthuisberg,
B-3000 Leuven, Belgium

ENHANCED TRANSCRIPTION OF TNF IN SYSTEMIC VASCULITIS

SIR,—Despite immunological and pathological studies in systemic vasculitis,[1] the pathogenesis of this condition remains elusive. The cytokine tumour necrosis factor alpha (TNF-α, cachectin) has a wide range of biological activities[2] and has been associated with acute and chronic inflammatory states and implicated as the exogenous mediator of shock.[3] In our studies of cytokine systems and cellular activation in peripheral blood mononuclear cells from patients with systemic vasculitis (eg, periarteritis nodosa and Wegener's granulomatosis), we have found that the transcription[4] of the TNF-α gene is enhanced (figure) while transcription of the actin gene was normal (data not shown). We also looked for DNA amplification and gross translocation of the TNF-α gene by Southern blot analysis but found neither (data not shown).

TNF-α inhibits endothelial cell growth, contributing to vascular damage,[5] and in-vivo administration of this cytokine is associated with severe endothelial damage and vascular leak syndrome.[6] Our



Expression of TNF-α gene in peripheral blood mononuclear cells from 5 patients with periarteritis nodosa, 6 with Wegener's granulomatosis, 6 healthy controls, and 5 controls with asthma.

TNF-α specific transcription was evaluated by nuclear run on transcription assay. Purified nuclei from mononuclear cells were incubated with ^{32}P-ATP, and isolated RNA was hybridised with dotted human TNF-α specific probe[4] on filters. Tests were in duplicate. Autoradiographic images were quantified by densitometry and expressed in units per 10^6 cells.