The Impact of Renal Revascularisation on Renal Dysfunction

H. van Damme[1], F. Jeusette[2], A. Pans[1], J.O. Defraigne[1], E. Creemers[1], A. Albert[2] and R. Limet[1]

Departments of [1]Cardiovascular Surgery and [2]Biostatistics, University Hospital of Liège, Belgium.

Aim: To determine the value of kidney revascularisation in patients with impaired renal function and correctable renal artery stenosis, the authors reviewed their surgical experience from 1978 to 1990.
Patients and Methods: The study population included 23 patients with ischaemic nephropathy whose preoperative baseline creatinine level exceeded 20 mg/l (range 21–65 mg/l). This represents 20% of all patients operated on for renal artery disease during the same time interval. Preoperative risk profile, operative mortality, impact on hypertension and on renal function, and late survival were analysed. Renal function response to kidney revascularisation was defined as favourable (20% or more reduction of serum creatinine), moderate (stabilised serum creatinine values) or bad (further deterioration of renal function). All patients had atherosclerotic renal artery disease, involving a solitary kidney in five, both kidneys in 15 and one of the two kidneys in three patients. Hypertension was present in 74%. Revascularisation was unilateral in 10, bilateral in nine and associated with controlateral nephrectomy in four patients.
Results: Four patients died postoperatively (three myocardial infarctions, one stroke). Four patients needed postoperative short-term dialysis. After operation, renal function improved in 13, stabilised in six and deteriorated in four patients (of whom two died). Follow-up among the survivors averaged 46 months. The mean serum creatinine value at last follow-up visit was 26.2 mg/l, a decrease of 7.7 mg/l compared to preoperative values (p < 0.05). Overall, 69% of azotemic patients submitted to renal revascularisation manifested a favourable response (45% improved and 24% stabilised). Three patients required long-term dialysis. The 5-year survival rate was 48%.
Conclusion: These data suggest that kidney revascularisation in patients with ischaemic nephropathy can restore or stabilise renal function, preventing evolution and end-stage renal disease and dialysis dependency.

Key Words: Renal insufficiency; Renal revascularisation; Renal artery surgery; Renovascular disease; Ischaemic nephropathy.

Introduction

Although the role of surgery for treating renovascular hypertension has been challenged by drug therapy improvement, the usefulness of kidney revascularisation in preservation or recovery of renal function has been clearly stressed ever since the reports of Dean et al.[1] and Schreiber et al.[2] on the natural history of renal artery disease. Their observations emphasised the progressive nature of atherosclerotic renal artery disease, with subsequent loss of functional renal mass. Among 41 hypertensive patients with atherosclerotic renal artery disease, selected for medical treatment, Dean et al.[1] observed, progressive worsening of renal function (100% or more increase in serum creatinine, 10% or more loss in renal length or 50% or more decrease in glomerular filtration rate) in 17 cases for whom operative management was ultimately war-

ranted. In four patients, a significant stenosis progressed to total occlusion over a period of 28 months. Adequate pharmacological blood pressure control could not obviate deterioration of renal function. In the study of Schreiber et al.[2] (non-surgical approach of 85 renovascular patients), 44% developed gradual progression of the stenosis and 16% of the lesions ultimately evolved towards total occlusion over a mean follow-up period of 52 months. Tollefson and Ernst[3] estimated the progressive narrowing of the diameter at about 5% per year in medically observed patients with renal artery stenosis.

The sudden unexplained deterioration of renal function in an older vascular hypertensive patient should always raise suspicion of progression of renal artery disease to critical stenosis or thrombosis.[4–7] In selected azotemic patients, renal large vessel disease should be considered as a potentially treatable cause of renal failure and elective revascularisation as the most appropriate management. Mailloux et al.[8] pre-

Please address all correspondence to: C.H.U. Liège, Domaine Universitaire du Sart-Tilman, 4000 Liège (Belgium).

sumed a renal vascular cause in 16.5% of patients with end-stage renal disease, the third most frequent cause after glomerulonephritis and diabetes. In an angiographic survey of end-stage renal disease, Scoble et al.[9] observed that 6% of the renal insufficiencies were caused by ischaemic nephropathy, and that 14% of the patients on dialysis over the age of 50 years had ischaemic nephropathy.

The present retrospective study was carried out to evaluate the potential role of renovascular repair in the management of patients whose renal artery disease was associated with an impaired renal function. Severe renal dysfunction was considered if preoperative serum creatinine level exceeded 20 mg/l.[10–13] Criteria for blood pressure response and renal function benefit are those described by Dean et al.[4] and Elmore et al.[10]

Methods

From 1978 to 1990, a total of 115 patients underwent renal artery surgery in our department. A subgroup of 23 patients with chronically impaired renal function on admission (baseline serum creatinine level > 20 mg/l) was identified. Renal revascularisations for acute renal ischaemia were not included. The presumed cause of renal function impairment was ischaemic nephropathy. In none of the patients was preexisting primary renal parenchymal disease suspected (no urinary abnormalities, no systemic disease). Data analysed included age, sex, blood pressure, drug requirement, duration of hypertension, and selected risk factors that could influence the operative results (diabetes, ischaemic heart disease, stroke and aortoiliacal disease). Preoperative evaluation included aortorenal angiography, serum creatinine level, and isotopic studies. Selection criteria for renal artery repair included preserved but delayed renal excretory function and, in case of a malfunctioning kidney, a kidney length of at least 7 cm and residual perfusion on scintigraphy. Renin levels were not routinely measured and were only available for seven patients. Operative data comprised the surgical procedure, type and extent of complications, need for haemodialysis and the cause of operative death (deaths occurring within 30 days after surgery). Patency of vascular reconstructions was controlled before hospital discharge by isotopic angioscintigraphy and when doubtful, documented by angiography.

Special attention was given to the effect of renal revascularisation on renal function and hypertension, as well as to the duration of the therapeutic effect. A

favourable renal functional response to surgery was defined as a 20% or more reduction of serum creatinine level at first follow-up visit (2 months following surgery). A minimal benefit was considered if, at long term, renal excretory function had been stabilised, while failures included worsened postoperative renal function as well as operative deaths. The renal function at last follow-up visit was compared with preoperative values. With regard to blood pressure response to surgery, we discerned three categories of late outcome (at least 6 months after renal artery repair): cured, improved, and failed. Patients were defined as cured if normotensive (diastolic pressure less than 95 mmHg without medication) and as improved if diastolic pressure lowered at least 20 mmHg with the same or reduced drug regimen. Treatment failures were patients with unchanged or worsened hypertension as well as operative deaths. The baseline values reported for blood pressure and serum creatinine represent averages of at least three separate determinations.

Follow-up data (serum creatinine level, blood pressure, drug regimen, reoperation, and cause of death) were retrieved from medical records. Half of the patients were re-examined at the out-patient clinic during the last 6 months of 1992. For the remaining survivors, the most recent information, including blood pressure and serum creatinine, were obtained by correspondence with their family doctor. The prognostic value of factors likely to affect late survival was analysed.

Statistics

Means and s.D. were computed for continuous findings (e.g. age, serum creatinine levels, duration of hypertension) and frequencies for categorical data (e.g. sex, hypertension). Patient groups were compared by Mann-Whitney U-test, whereas for paired observations a Wilcoxon signed rank sum test was used. The Kaplan Meier method was used for determining the late survival curve. Results were considered to be significant at the 5% critical level ($p < 0.05$).

Results

Preoperative data

Among the 23 patients with chronically impaired

renal function due to renovascular disease, there were 16 men and seven women, aged 42–84 years (mean 63 ± 11 years). Two patients had diabetes type II but, as previously defined, without parenchymal glomerular disease. The average serum creatinine level on admission was 34 ± 12 mg/l (extremes 21–65 mg/l). Based on serum creatinine values, the estimated glomerular filtration rate (EGFR) (Cockroft estimation[14]) ranged from 15 to 50 ml/min (mean 30 ± 9 ml/min). Among the 11 patients with severe renal dysfunction (creatinine >30 mg/l), one was on chronic haemodialysis.

Seventeen patients (74%) of the study group also had drug resistant hypertension (diastolic pressure exceeding 95 mmHg despite optimised medical therapy). None of these patients were on ACE (angiotensine converting enzyme) inhibitors, known to interfere with functional renal impairment. The mean duration of refractory hypertension was 41 months (range 6–96 months). The type of renal artery disease was always atherosclerotic, all patients having clinical evidence of advanced widespread atherosclerosis (e.g. ischaemic heart disease, carotid artery disease or aortoiliac disease).

Angiographic data

At angiography, only three patients had truly unilateral renal artery disease, with a non significant stenosis (<70%) of the controlateral artery. In two cases, PTA (percutaneous angioplasty) was attempted, unsuccessfully, prior to surgery. Five patients with a previous nephrectomy had significant renal artery stenosis of their single kidney. Of the 15 patients with bilateral disease, seven had an occlusion of one of the renal arteries. On angiography an excretory parenchymal renogram was not visualised in 10 nonfunctioning kidneys (including three solitary kidneys). Seven had a totally occluded renal artery and three a tight stenosis. Renal flow scan revealed residual perfusion in six of the 10 non-functioning kidneys.

Surgical procedures

The revascularisation was unilateral in 10 patients, bilateral in nine, and a controlateral nephrectomy of a renin producing shrunken kidney (<7 cm in length) with a non-reconstructible artery concomitantly performed in four cases. Of the seven occluded renal arteries, three were repaired. In these cases, the kidney

was at least 7 cm in length and there was evidence of residual glomerular filtration on renal perfusion scan. Distal renal artery patency had been maintained through a network of collateral vessels as assessed by intraoperative Doppler before arterial repair.

Revascularisation was complete (no residual stenosis left) in 21 patients (92%). In two patients, a controlateral stenosis of 70% was left uncorrected. A variety of operative techniques was used: aortorenal bypass in 10 cases (autogenous saphenous vein in eight and Dacron prosthesis in two cases), thrombendarterectomy in nine (ostial thrombendarterectomy in five patients, associated to patch angioplasty in three of them, and transaortic endarterectomy in four patients, always in association with aortic surgery), splenorenal anastomosis in two, hepatorenal bypass in one, and renal autotransplantation in one. The adequacy of renal artery repair was checked by intraoperative continuous wave Doppler. In doubtful cases, the surgeon opted for another strategy (two thrombendarterectomies and one splenorenal anastomosis were changed for venous aortorenal bypass). Concomitant aortoiliac surgery was performed in nine patients.

All nephrectomised kidneys were examined by the pathologist. Histology revealed nephrosclerotic changes in all shrunken kidneys, with hyalinisation of arterioles and patchy ischaemic fibrosis. There was no evidence of glomerulonephritis nor pyelonephritis.

Postoperative outcome

There were four operative deaths, yielding a mortality rate of 17%. The primary cause of death was cardiac in three cases and one had fatal stroke.

Operative mortality was associated with longstanding hypertension or with severe renal dysfunction (creatinine >30 mg/l). Of the 17 hypertensive patients with impaired renal function, three died postoperatively. The latter had suffered hypertension for 66 months, on average, against 20 months for 14 survivors. The main preoperative serum creatinine was 38 ± 5 mg/l for non-survivors and 33 ± 13 mg/l for survivors (p = 0.09). The presence of coronary artery disease appeared to be a determinant for cardiac mortality, since all patients who died from myocardial infarction had a past history of ischaemic heart disease. Combined aortoiliac surgery (n = 9) led to a 22% mortality rate (2/9), against 14% for isolated renal revascularisation (NS). Operative morbidity included two non-fatal myocardial infarctions, three respiratory infections and one anastomotic leak necessitating reoperation.

Five patients required early postoperative haemodialysis, but only on temporary basis, except for the one patient who was already on chronic dialysis before surgery. One patient needed dialysis after primary failure of the arterial repair, and three were temporarily dialysed in spite of patent repair. None of the renal arterial reconstructions resulted in new dialysis dependency. In two patients, early thrombosis of the renal artery repair (one endarterectomy, one Dacron bypass graft) occurred (9% primary failure rate). One of them needed temporary dialysis. One had successful thrombectomy, while secondary nephrectomy was performed in the other patient whose controlateral repair was patent.

After operation, serum creatinine dropped significantly to an average level of 30 ± 17 mg/l ($p = 0.05$). It decreased by at least 20% in 13 patients (57%) and stabilised in six (26%), thus yielding an early beneficial effect of operation on renal function in 83% of the patients. Almost all cases of deteriorating postoperative function (3/4) were observed in the group of patients with severe renal dysfunction (creatinine > 30 mg/l). Two of them died postoperatively (one myocardial infarction, one stroke during dialysis). Of the two other operative deaths, the renal function was stabilised in one and improved in the other. Both suffered fatal myocardial infarction. Of the 19 patients who were discharged from hospital, 12 (63%) left hospital with improved renal function, five (26%) with stabilised serum creatinine level, while in two cases (11%), renal function further deteriorated (Table 1). The greatest improvements of renal function, a reduction of serum creatinine of 15 mg/l or more ($n = 5$), were observed among patients with severe preoperative renal functional impairment (creatinine > 30 mg/l), although these patients were also at highest operative risk. Longstanding hypertension appeared to be a poor predictor of failure to benefit from operation: all four patients whose renal function worsened postoperatively had hypertension for a mean duration of 65 months vs. 22 months duration for the 13 hypertensive patients whose renal function improved or stabilised.

Table 1. Operative outcome

Creatinine (mg/l)	Postoperative renal function			Operative deaths	Total
	Unchanged	Improved	Deteriorated		
20–30	4	7	1	1	13
> 30	1	5	1	3	10
	5	12	2	4	23

Long-term results

Complete follow-up data were available for 17 of the 19 patients discharged from hospital. Two patients were lost to follow-up at 6 and 18 months respectively, when they moved abroad. Follow-up periods ranged from 2 to 14 years (mean 46 ± 31 months). At their last visit, 10 patients (59%) had improved serum creatinine levels, compared to their preoperative value, while a moderate benefit was noted in five patients whose preoperative creatinine level remained stable throughout the follow-up period. In two patients, renal excretory function worsened in spite of an initial technically successful revascularisation. They ultimately became dependent on haemodialysis, after 17 and 36 months. In one of them a late thrombosis of an aortorenal bypass graft was documented 3 years later. Since no residual excretory function was evidenced by isotopic studies, no reoperation was considered. The other had angiographic evidence of graft patency despite progressive renal failure. The three patients requiring long-term dialysis died within the first year of dialysis. The average creatinine level at the last follow-up visit was 26 ± 18 mg/l, thus yielding a significant decrease of 8 mg/l from preoperative values ($p < 0.05$). The most restrictive evaluation of the late operative result gave 43% (10/23) of the patients with improved renal function, 22% (5/23) with unchanged creatinine values, and 35% (8/23) failures, including all in-hospital deaths ($n = 4$) and patients lost to follow up ($n = 2$).

Regarding blood pressure control in long-term survivors who had preoperative hypertension ($n = 14$), 4 patients were cured (28%) after renal revascularisation, six were improved (43%), three remained unchanged (21%) and one worsened (7%). Severe preoperative renal dysfunction (serum creatinine > 30 mg/l) was predictive for unfavourable response in terms of blood pressure control ($p = 0.1$). None of the survivors with preoperative hypertension and creatinine exceeding 30 mg/l ($n = 4$) was cured or improved. No other preoperative variable (e.g. duration of hypertension) was significantly associated with the clinical response rate in terms of blood pressure control.

During the follow-up period, six patients died. Four of these deaths (two patients on dialysis) were due to fatal myocardial infarction. One suffered a fatal stroke while on dialysis and another died of cancer. For the 17 patients followed since hospital discharge, the actuarial 3-year and 5-year survival rates were 75% and 48% respectively. This is lower than the life expectation of an age- and sex-matched general Belgian population (83.6%, 5-year survival)[15] (Fig. 1).
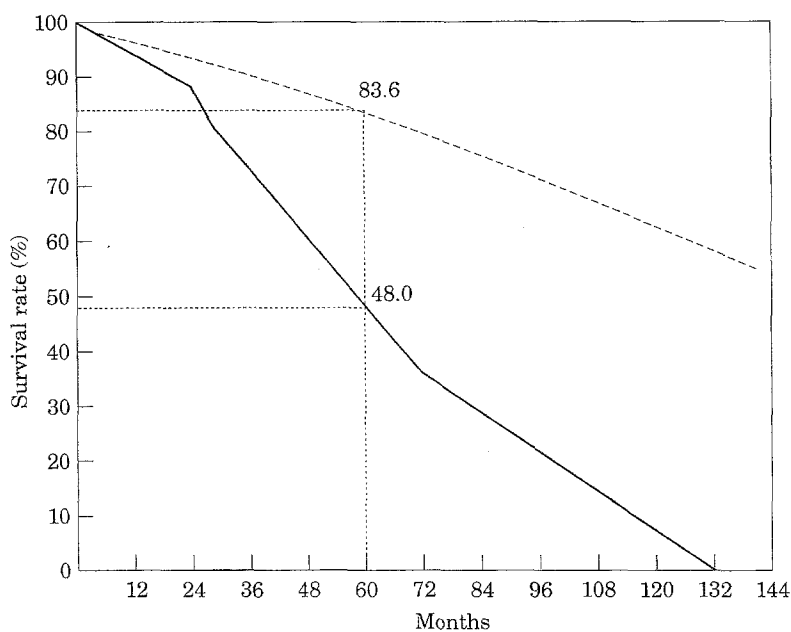
Fig. 1. Survival estimates of 17 patients followed after operation for renal dysfunction (followed for 2 to 14 years), compared to life expectation of an age- and sex-matched Belgian population. (——) patients followed after operation for renal impairment (*n* = 17); (— — —) age- and sex-matched Belgian population.

Discussion

Ischaemic nephropathy, defined as the combined presence of extraparenchymal occlusive renal artery disease and renal dysfunction[1] (arbitrarily defined as serum creatinine level exceeding 20 mg/l[10–13]), has become an important indication for renovascular repair. It represents 10–30% of the large series of renal artery surgery.[4,11,12,16] Dean *et al.*[4] noted that this type of renal insufficiency is characterised by a more rapid decline of creatinine clearance, compared to other causes of renal insufficiency (4 ml/min/month *vs.* 0.5 ml/min/month).

Patients with renovascular disease and deteriorating renal function are extremely ill patients with severe systemic atherosclerosis. They have a poor prognosis due to refractory hypertension, congestive heart failure and evolving renal insufficiency. The inherent high operative risk of renal revascularisation in these elderly polyvascular patients should be balanced against the potential benefits. The only way to answer this question would be a randomised clinical trial of surgical *vs.* medical management of such patients. Up to now, such a study is not available.

Since the Cooperative Study of Renovascular Hypertension (1975),[17] it is well known that operative mortality after renal revascularisation is greatly influenced by preoperative renal function impairment. The mortality rate reached 11.5% for patients with serum creatinine levels greater than 15 mg/l, *vs.* 4.7% for patients with normal renal function. This significant procedure related mortality tempered the enthusiasm for a surgical approach to renal failure, and percutaneous balloon angioplasty (PTA) gained favour.[18–20] We consider percutaneous dilatation as a safe method for correction of short truncal renal artery stenosis. Nevertheless, many atherosclerotic renal artery lesions are not suitable for angioplasty, such as calcified ostial lesions, extensive total occlusions of the main artery and associated aortic occlusive or aneurysmal disease, characterised by an inherent risk of cholesterol embolisation.[20] Weibull *et al.*,[19] however, did not find any difference between the two methods (PTA versus surgery) for selected cases of unilateral ostial renal artery stenosis, with regard to blood pressure response or influence on renal function. Pattison *et al.*[20] also obtained excellent results with PTA in patients with renal failure (improvement in 40%, deterioration in 10%).

In recent series, mortality rates of 2–7% are mentioned, contrasting with the high mortality in our series[10–13] (Table 2). The leading cause of operative death was cardiac. A more adequate preoperative screening for ischaemic heart disease and eventual myocardial revascularisation prior to renal artery surgery might have reduced our operative mortality rate.[12,21] Important factors that predispose to operative death are prior history of ischaemic heart disease,[11,12] and severe renal impairment (creatinine

Table 2. Results of renal revascularisation for renal dysfunction

	Reference	Year	n	Mean creatinine		% Improvement † (early results)	Mortality (%)	Late dialysis (%)	Follow-up (months)	5 year survival (%)
				Preoperatively (mg/l)	Postoperatively (mg/l)					
Elmore *et al.*	10	1974–86	30§	35	24	$70 <^{49}_{36}$	3.3	26.0	32	—
Hallet *et al.*	11	1970–81	98‡	30	20	$69 <^{20}_{49}$	7.1	4.4	42	64%
Hansen *et al.*	12	1987–91	70‡	—*(15 ml/min)	—*(31 ml/min)	85	5.7	4.2	24	40%
Chaikof *et al.*	13	1982–92	50§	31*(25 ml/min)	—*(30 ml/min)	$96 <^{42}_{54}$	2.0	18.4	49	62%
Van Damme *et al.* (this series)		1978–90	23‡	33.9*(30 ml/min)	26	$83 <^{57}_{26}$	17.0	13.0	46	48%

* EGFR = estimated glomerular filtration rate.
† the percentage of favourable response is subdivided in % that had improved renal function (above) and in % whose renal function stabilised (below).
‡ ≥ 20 mg/ml.
§ ≥ 18 mg/ml.

> 30 mg/l).[11–13,22] Severe renal functional impairment (creatinine > 30 mg/l) is the most important predictor of operative death in our series, as well as in others.[11–13,22] In the large series (*n* = 98) at the Mayo Clinic,[11] the operative mortality was 3% for patients with initial creatinine of 20–29 mg/l, but reached 13% for patients with preoperative creatinine values above 30 mg/l. This reflects more severe atherosclerosis at renal and extrarenal levels. One may conclude that successful outcome of renal artery repair is more likely in patients with moderate deterioration of renal function, and that postponing the operation only makes it more hazardous.[13] We also found a significant relationship between longstanding (5-years or more) hypertension and operative mortality. One can explain the increased operative risk by more advanced atherosclerosis and cardiac failure as a result of longstanding hypertension.

The beneficial effect of renal revascularisation is illustrated in our series by a positive postoperative response rate (improved or stabilised creatinine level) in 83% of patients and in 65% of the surviving patients at their last follow-up visit. Considering the 10 patients who were candidates for chronic haemodialysis (preoperative serum creatinine > 30 mg/l, excluding the one patient who was on chronic dialysis), five could be definitely kept off of dialysis during the follow-up period of 46 months, three died perioperatively, and two became dependent on dialysis in spite of successful revascularisation. Longstanding hypertension (duration of 5 years or more) decreased the chance of renal functional improvement after successful revascularisation. This could mean that certain patients had already hypertensive kidney damage (intrarenal small vessel disease). Such nephrangio-

sclerotic changes will not be reversed by renal artery repair. Conversely, patients with accelerated recent onset of hypertension and renal function decline have a greater chance to improve after renal revascularisation.

Seventy-one percent of the survivors who were initially also hypertensive became normotensive (28%) or had improved blood pressure control (43%) during follow-up. Dean *et al.*[4,16] and Hallett *et al.*[11,23] obtained an improved blood pressure control in about 60% of their azotemic patients (creatinine > 20 mg/l) submitted to renal revascularisation. Hansen *et al.*[12] even mentioned an 80% success. This improvement seems to be durable. As in our series, the same author[12] observed less favourable clinical response to surgical correction in patients with longstanding hypertension. In addition, we found that severe preoperative renal dysfunction was predictive for poor blood pressure control. Chaikof *et al.*[13] noted the same finding. One could speculate that some nephrangiosclerotic kidneys were inappropriately revascularised. This assumption could not be verified because renal biopsies were not available. Our selection criteria for kidney salvage are preoperative kidney size of at least 7 cm length, residual isotopic glomerular filtration and disease free distal renal artery. Some authors[6,22–25] insist on the value of renal biopsy in evaluating the viability of nephron units in a poorly functioning kidney and in prediction of functional recovery. If less than 50% of the glomeruli are involved by nephrosclerosis, the kidney is considered salvageable. However biopsy can be misleading and its usefulness is controversial.[10,26]

We also argue for early intervention (serum creatinine below 30 mg/l) to lower the operative risk and

..to assure a favourable functional response after renal revascularisation. In Chaikof et al.'s series,[13] the incidence of late kidney failure and ultimate dialysis (despite initially successful renal revascularisation) was directly correlated to the preoperative azotemia: up to 32% of patients with preoperative chronic severe renal insufficiency (creatinine > 30 mg/l) came to dialysis within 3 years of renal revascularisation. The moderate early improvement in these patients was not sustained at long follow-up. Jamieson et al.,[22] Dean et al.[4] and Mercier et al.[26] also conclude that patients with end-stage renal failure are unlikely to benefit from surgery, because of the irreversible kidney damage (nephrangiosclerosis, glomerular scarring). Once a renovascular patient is on chronic dialysis, renal artery surgery can only exceptionally reverse dialysis needs. This finding is not shared by others. The Cleveland group[27] report nine cases of end stage renal failure caused by atherosclerotic renal artery occlusion, who benefited from renal revascularisation and no longer remained dependent on dialysis. The kidney size was always more than 10 cm in length. This represents 1% of all dialysed patients in their centre. Hansen et al.[12] reported that out of a total of 23 patients with extreme (creatinine > 30 mg/l) renal insufficiency 8/11 patients dependent on chronic dialysis were improved by bilateral revascularisation. Pattison et al.[20] was able to wean 10 of 13 patients off chronic dialysis by percutaneous angioplasty. The duration of the pre-operative chronic dialysis was not mentioned in any of these reports.

It is often stated that severe bilateral disease or ischaemia of a solitary kidney are necessary to cause functional renal impairment.[7,21] In rare cases, uni-lateral tight stenosis can produce hypertension, severe enough to destroy the non-ischaemic controlateral kidney by nephrosclerosis. Unilateral renal artery repair can retrieve renal function in such cases.[4] Nevertheless, most authors[4,5,12,16] observed a better beneficial renal function response in patients who had their bilateral renal artery lesions or single kidney revascularised, compared to the renal function response in patients where only one of the two kidneys had a lesion needing correction. Dean et al.[16] found a significant difference between unilaterally and bilaterally revascularised severely azotemic patients (creatinine > 30). In the latter, the mean serum creatinine dropped from 51 to 28 mg/l, while it remained unchanged at 43 mg/l in the former group. In our study, as well as in the recent series of Chaikof et al.,[13] there was no evidence of such relationship between the anatomic extent of the renal artery disease and the result of its repair: bilateral repair did

not reveal a propensity for greater success than unilateral kidney revascularisation.

Late survival was largely affected by associated cardiovascular disease.[4,11-13,26] Failure to improve severe renal dysfunction also predisposes to early death during follow-up. In the series of Dean et al.,[4] 44% (4/9) of the follow-up deaths were patients on chronic dialysis. In our series, half of the late deaths were patients on dialysis and myocardial infarction was the main cause. The 5-years survival is 50–60% and is superior to that obtained by medical manage-ment of similar patients with ischaemic nephropathy, as suggested in the non-randomised study of Novick et al.[21] The estimations of Mailloux et al.[8] are even more convincing: azotemic patients, almost 60 years and older, with uncorrected renal artery stenosis who progressed to dialysis, had a 5-years life expectation of 12%. In a large review on the outcome of dialysed patients, Held et al.[28] calculated an annual death rate of 34% for dialysed patients aged 65 years or older. The bad prognosis of terminal renal insufficiency in elderly atherosclerotic patients makes revascularisa-tion worthwhile to avoid dialysis. A more aggressive approach to the diagnosis and adequate treatment of associated ischaemic heart disease, not only allows operative risk to be reduced but also increases life expectancy.[11,30]

Acknowledgement

References

1 DEAN R, KIEFFER R, SMITH B et al. Renovascular hypertension. Anatomic and renal function changes during drug therapy. Arch Surg 1981; 116: 1408–1415.
2 SCHREIBER M, POHL M, NOVICK A. The natural history of atherosclerotic and fibrous renal artery disease. Urol Clin N Am 1984; 11: 383–392.
3 TOLLEFSON D, ERNST C. Natural history of atherosclerotic renal artery stenosis associated with aortic disease. J Vasc Surg 1991; 14: 327–331.
4 DEAN R, TRIBBLE R, HANSEN K, O'NEIL E, CRAVEN T, REDDING J. Evolution of renal insufficiency in ischemic nephropathy. Ann Surg 1991; 213: 446–456.
5 KREMER HOVINGA T, DE JONG P, VAN DER HEM G, DE ZEEUW D. Relief of renal artery stenosis: a tool to improve or preserve renal function in renovascular disease. Nephrol Dial Transplant 1990; 5: 481–488.
6 SHEIL A, MAY J, STOKES G, JOHNSON J, TILLER D, STEWART J. Reversal of renal failure by revascularisation of kidneys with thrombosed renal arteries. Lancet 1973; 20: 865–866.
7 YING C, TIFFT C, GAVRAS H, CHOBANIAN A. Renal revascularisa-tion in the azotemic hypertensive patient resistant to therapy. N Engl J Med 1984; 311: 1070–1075.

8 MAILLOUX L, BELLUCI A, MOSSEY R et al. Predictors of survival in patients undergoing dialysis. Am J Med 1988; 84: 855–862.

9 SCOBLE J, MAHER E, HAMILTON G et al. Atherosclerotic renovascular disease causing renal impairment. A case for treatment. Clin Nephrol 1989; 31: 119–122.

10 ELMORE J, RAY F, DILLIHUNT R, HERBERT W. Renal failure and advanced atherosclerotic lesions. Salvage by vascular reconstruction. Arch Surg 1988; 123: 610–613.

11 HALLETT J, FOWL R, O'BRIEN P et al. Renovascular operations in patients with chronic renal insufficiency: do the benefits justify the risks? J Vasc Surg 1987; 5: 622–627.

12 HANSEN K, STARR S, SANDS E et al. Contemporary surgical management of renovascular disease. J Vasc Surg 1992; 16: 319–331.

13 CHAIKOF E, SMITH R, SALAM A et al. Ischemic nephropathy and concomitant aortic disease: a ten-year experience. J Vasc Surg 1994; 19: 135–148.

14 COCKROFT D, GAULT M. Prediction of creatinine clearance from serum creatinine. Nephron 1976; 16: 31–41.

15 INSTITUT D'HYGIÈNE ET D'EPIDÉMIOLOGIE — MINISTÈRE DE LA SANTÉ PUBLIQUE BELGE. Probability of death in Belgium, 1987.

16 DEAN R, ENGLUND R, DUPONT W et al. Retrieval of renal function by revascularisation. Study of preoperative outcome predictors. Ann Surg 1985; 202: 367–375.

17 FRANKLIN S, YOUNG J, MAXWELL M et al. Operative morbidity and mortality in renovascular disease. JAMA 1975; 231: 1148–1153.

18 FLECHNER SM. Percutaneous transluminal dilatation: a realistic approach in patients with stenosing lesions of the renal artery. Urol Clin N Am 1984; 11: 515–527.

19 WEIBULL H, BERGQVIST D, BERGENTZ S et al. Percutaneous transluminal renal angioplasty versus surgical reconstruction of atherosclerotic renal artery stenosis: a prospective randomized study. J Vasc Surg 1993; 18: 841–852.

20 PATTISON J, REIDY J, RAFFERTY M et al. Percutaneous transluminal renal angioplasty in patients with renal failure. Q J Med 1992; 85: 883–888.

21 NOVICK A, POHL M, SCHREIBER M, GIFFORD R, VIDT D. Revascularisation for preservation of renal function in patients with atherosclerotic renovascular disease. J Urol 1983; 129: 907–912.

22 JAMIESON G, CLARKSON A, WOODROFFE A, FARIS I. Reconstructive renal vascular surgery for chronic renal failure. Br J Surg 1984; 71: 338–340.

23 HALLETT J, SCHIRGER A, BOWER T et al. The current role of surgical revascularisation for combined renovascular hypertension and renal insufficiency. Intern Ang 1992; 11: 64–68.

24 ZINMAN L, LIBERTINO J. Revascularisation of the chronic totally occluded renal artery with restoration of renal function. J Urol 1977; 118: 517–521.

25 CASES A, CAMPISTOL J, ABAD C et al. Reversal of renal failure by revascularisation in atheromatous renovascular disease. Am J Nephrol 1988; 8: 479–482.

26 MERCIER C, PIQUET P, ALIMI Y, TOURNIGAND P, ALBRAND J. Lésions occlusives des artères rénales et insuffisance rénale chronique: limites de la chirurgie de revascularisation. Ann Chir Vasc 1990; 4: 166–170.

27 KAYLOR W, NOVICK A, ZIEGELBAUM M, VIDT D. Reversal of end stage renal failure with surgical revascularisation in patients with atherosclerotic renal artery occlusion. J Urol 1989; 141: 486–488.

28 HELD P, PAULY M, DIAMOND L. Survival analysis of patients undergoing dialysis. JAMA 1987; 257: 645–650.

29 McCREADY R, DAUGHERTY M, NIGHBERT E et al. Renal revascularisation in patients with a single functioning ischemic kidney. J Vasc Surg 1987; 6: 185–190.

30 NOVICK A, STRAFFON RA, STEWART BH, GIFFORD RW, VIDT DG. Diminished operative morbidity and mortality in renal revascularisation. JAMA 1981; 246: 749–753.