



# Kidney news

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## PREVENTION OF PROGRESSION OF CHRONIC RENAL FAILURE.

### Introduction

What can be done to detect early the presence of renal failure – especially since it is often symptom free? Early detection is based on being suspicious of renal involvement in systemic diseases, and those at patient groups at risk.

Estimating from international cross-sectional studies, the number of people with renal abnormalities (any one, or combination of: impaired GFR, haematuria, proteinuria) is as many as 4000 new patients in NZ per annum.

There is not enough renal physician expertise in NZ to meet the growing needs for prevention of progression of CRF. The importance of involvement of the general practitioner in the clinical management of renal patients is paramount to reduce the huge burden of end-stage renal disease. Success of such a preventative and early detection programme requires close liaison between the renal physician and the general practitioner.

This issue of **kidney news** is to help educate the GP (and other non-renal physician medical practitioners) in the early detection and management of renal patients to delay and minimise the progression of CRF.

### What can be done?

A summary of the points are:

1. Optimal blood pressure control
2. Minimisation of proteinuria
3. Avoidance of nephrotoxins
4. Management of serum lipids
5. Optimal control of diabetes mellitus
6. Cessation of smoking
7. Avoid diabetes mellitus/overweight
8. Monitor at-risk populations

### **Prevention is at three levels:**

1. primary prevention (prevention of renal damage, and is the most important). Examples include avoidance of nephrotoxins, early and successful treatment of proteinuria, dietary management to avoid obesity, manage lipid levels, prevent development of diabetes mellitus, low salt diet to avoid development of hypertension.
2. secondary prevention (prevention of the progression of renal failure). Once established, minimise the further insult and damage from hypertension, proteinuria and nephrotoxins.
3. tertiary prevention (minimising the complications of ESRF).

### What are the modifiable factors and goals according to the evidence?

1. Good control of diabetes mellitus – HbA<sub>1c</sub>, 7% (possibly as low as 6.5%).
2. Optimal BP control - < 140/80; and in diabetes mellitus <130/70mmHg.
3. Cessation of tobacco smoking.
4. Avoidance of obesity – BMI < 25.
5. Dietary adjustments with reduced sodium intake (<100mmol/24 hours); the lower the better; and reduced protein in the diet (which helps reduce sodium intake, especially when red meat is reduced).
6. Optimal management of lipid profile.

### Good control of diabetes.

**Exercise** assists both weight loss in the obese diabetic, and the utilisation of glucose. The optimal weight is guided by the **BMI, best BMI < 25 kg/m<sup>2</sup>**. Some obese type 2 diabetics can reduce their medication dosage with weight loss. When the type 2 diabetic patient develops proteinuria and reduced renal function they have boarded the slippery slope to death. **75% will die of cardiovascular death, the remainder (25%) will progress to ESRF.** Many of these deaths are avoidable by avoiding obesity, and maximising diabetic control (**HbA<sub>1c</sub> < 7%**), and minimising proteinuria (see below).

### Optimal blood pressure control.

Whilst it is widely known angiotensin converting enzyme inhibitors (ACEIs) and angiotensin-2 receptor blockers (ARBs) have protective benefits over and above their BP control, cross-sectional studies still show up to 20% of diabetic patients are not on these medications – more than the number of patients with significant renal artery stenosis.

**Most patients with renal failure require three or more BP medications to address their hypertension adequately.** ACEIs and ARBs do have renoprotective benefits in non-diabetics. Thus these two classes of anti-hypertensives should be the first **line agents** for all patients with renal impairment and/or proteinuria – unless contraindicated.

Diuretics and β-blockers and vasodilators and calcium channel-blockers may then be added. β-blockers will be preferred in IHD.

Dietary sodium intake is often overlooked in hypertension. Despite the no-added salt in the cooking or at the table diet, dietary sodium analysis is often over the **recommended upper limit of 100mmol sodium/24-hours.** **The 24-hour urine collection is an easy way to assess the daily sodium intake.**

Left ventricular hypertrophy (LVH) occurs in the hypertensive patient, but also is a **direct result of**

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**anaemia per se.** Early referral for the initiation of erythropoietin (NZ criteria are Hb<100g/l, and GFR <30ml/min/1.73m<sup>2</sup> BSA (<45ml/min/1.73m<sup>2</sup> in diabetics)) is also important to reduce the cardiovascular morbidity risk of anaemia.

**Minimise proteinuria.**

Now evidence suggests ACEIs and ARBs in combination, and **at doses above their antihypertensive effects are effective at lowering proteinuria.** We may be using doses adequate for BP control, however not adequate to maximise the treatment of proteinuria.

**Proteinuria is a strong predictor of both cardiovascular death in renal failure, and progression to ESRF.** **Dietary protein manipulation** (with the guidance of an experienced renal dietician), **use of hypoproteinuria agents** (immunosuppressives (corticosteroids and cytotoxics), ACEIs and ARBs) are all important.

**Avoidance of nephrotoxins.**

COX-2 inhibitors are **not more renal friendly** than NSAIDs. Fibrates should be avoided in CRF.

**Minimisation of vascular disease.**

Oral folate 1-2 mg daily reduces the cardiac morbidity effects of the hyperhomocysteinaemia of CRF. Pragmatically **folate 5mg daily** is recommended in all patients with CRF.

**Cessation of smoking and reduction of hyperlipidaemia** are also priorities. Once CRF is established, **statins** are the only safely indicated lipid lowering agents available. Primary and secondary prevention of cardiovascular events with the **additional of aspirin** also.

**SUMMARY**

Multiple interventions are required to prevent renal disease establishment and its progression. Lifestyle changes (eg. exercise, dietary manipulation, attainment of optimal BMI, cessation of smoking), multiple medications for anti-hypertensive (often 3 or 4 different agents in advanced renal failure), anti-proteinuric agents (immunosuppressives and/or ACEIs and ARBs), aspirin, and statins. In addition, phosphate binders and calcium supplementation, with oral vitamin D all add to the huge polypharmaceutical armamentarium each CRF patient needs to be prescribed and administered daily. Multiple laboratory tests are also required to monitor the progress.

**Monitoring.**

As a guide, blood and urine test intervals are:

GFR >60ml/min	6-12 monthly
GFR 30- 60ml/min	3 monthly
GFR 15-29 ml/min	2-3 monthly
GFR < 15ml/min	monthly

Any sudden change in monitored parameters should alert the GP to contacting the renal physician early if the change cannot be fully explained. The GP must not feel isolated. The GP,patient:renal physician triad must be a close, frequent communicative, complex, informative long-term relationship.

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## Qualifications

BSc (Biochemistry, Otago) 1981

MBCbB (Otago) 1984

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MRCp(UK) 1993

## Interests

Investigation of renovascular disease and hypertension

Management of urinary tract infections

Investigation of urinary calculi

Investigation of proteinuria and haematuria

Early detection, investigation and management of impaired renal function.

Renal nutrition.

**For All Appointments  
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## Consulting Rooms At:

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**Takanini Care Accident & Medical Clinic**

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TAKANINI

**Waitemata Specialist Centre**

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TAKAPUNA

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