

# **RENAL UNIT HANDBOOK**

**ROYAL INFIRMARY**

**EDINBURGH**

**Fourth Edition February 2007**



# - C O N T E N T S -

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## INTRODUCTION

In 1989, two renal SHOs drew up a handbook for staff in the unit. Don Collie went on to become a neuroradiologist, but his co-author John Hodge utilized his experiences in a future career as a scriptwriter for such films as *Shallow Grave*, *Trainspotting* and *A Life Less Ordinary*. Their handbook started with the line: 'Welcome to the Renal Unit; you will find it many things but never spacious'. Thanks to Dr Gibson for recalling this.

The first edition of this handbook was commenced in 1999 and circulated in draft versions and then a rare first edition before its final publication in blue folders in January 2001. It, and subsequent editions, provide ready access to useful protocols. Almost all of these are guides rather than absolute rules – but this varies. All must be interpreted according to circumstances. There will be errors. If you spot anything that should be revised, please send comments and corrections to Dawn Hibbert, 29167, renal@ed.ac.uk). Volunteers to fill gaps are also welcome.

We used to list contributors individually, but now there are so many that this would be an incomplete list of people on the unit. So both thanks and apologies to everyone who has contributed. Main authors are increasingly being credited page by page on our webpages.

Web-based access to the protocols in this handbook is available at any time. These and much patient information are available from EdREN, the Unit website, at:

<http://www.edren.org>

Internet and Intranet versions of this handbook are available, including versions that will be suitable for handheld devices. Externally available versions don't contain Section A. Call 29167 or email renal@ed.ac.uk if it isn't obvious where they are.

Neil Turner and Caroline Whitworth, November 2006.

## DISCLAIMER

### **Necessary disclaimer**

We have tried to have everything in this handbook checked by more than one person, but it has been written and typed by fallible humans, and protocols change. It is ESSENTIAL that you read carefully, and interpret everything, particularly drug doses, with great caution. Please flag any errors or areas where there is scope for confusion, so that they can be changed.

## A: THE SERVICE

### MEDICAL STAFF AND RESPONSIBILITIES

#### Consultants

Consultants	Email	Code	Ext No	Bleep	Secretary
Dr CE Whitworth	caroline.whitworth@luht.scot.nhs.uk	CEW	21235	#6641	21235
Prof N Turner	neil.turner@ed.ac.uk	ANT	29167/6	5690	21235
Prof AD Cumming	allan.cumming@ed.ac.uk	ADC	29311	5084	21246
Dr P Gibson	paddy.gibson@luht.scot.nhs.uk	PHG	21237	#6619	21239
Dr RG Phelps	richard.phelps@ed.ac.uk	RGP	29164	#6832	29167
Dr J Goddard	jane.goddard@luht.scot.nhs.uk	JG	21256	5945	21267
Dr J Hughes	jeremy.hughes@ed.ac.uk	JH	26563		21246
Dr W Metcalfe	wendy.metcalfe@luht.scot.nhs.uk	WM	27411		21246
Dr J Neary	john.neary@luht.scot.nhs.uk	JN	21245	5085	21244

#### Associate Specialist

Dr M G Dimova	mariana.g.dimova@luht.scot.nhs.uk	MD	21248	2196	21234
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#### Middle grade

Specialist Registrars - normally 4 or 5 in RIE Renal (others elsewhere)  
On-call work is also supported by clinical research fellows  
Staff grade – one at the RIE; others possibly in the future  
FY2/CMT1+2 - 7 in Renal Medicine but one always attached to Transplant

	Reg bleep (FY2/CMT in brackets)
Acutes	#6394 or 07699 611603 (2144)
Ward	5190 (2172)
Transplantation	5285
Dialysis	5221

#### Work Areas

The areas of in-patient responsibility are each covered by a consultant and SpR (changing monthly):  
Ward 115 & Acutes, including ITU and hospital referrals  
Ward 117 & Renal Transplantation (inc. liver HDU patients requiring renal support)  
General ward patients (206)

## End-stage programmes – areas of responsibility

Dialysis Unit, RIE	Dr J Goddard Dr R G Phelps Dr J Neary
Dialysis Unit, WGH	Dr C E Whitworth
Dialysis Unit, SJH	Dr P Gibson
Dialysis Unit, BGH	Dr W Metcalfe
Community Dialysis Programme	Dr P Gibson & Dr J Neary
Transplant Clinic RIE Mon	Mr M Akyol
Transplant Clinic RIE Wed	Dr C Whitworth
Transplant Clinic RIE Fri	Dr W Metcalfe
Transplant Clinic SJH	Dr C P Swainson Dr P Gibson

## Transplant Consultants

Mr J L R Forsythe Sec (Donna)	(john.forsythe@luht.scot.nhs.uk)	#6617	21707 21715
Mr M Akyol Sec (Moira)	(murat.akyol@ed.ac.uk)	#6738	21701 21714
Mr K K Madhavan Sec (Carolyn)	(kk.madhaven@luht.scot.nhs.uk)	#6389	21702 21719
Prof O J Garden Sec (Val)	(o.j.garden@ed.ac.uk)	#6412	23661 23661
Mr J Casey Sec (Moira)	(j.casey@ed.ac.uk)	#6302	21700 21714
Miss L Marson Sec (Donna)	(lorna.marson@ed.ac.uk)	#6417	21715
Mr Ernest Hidalgo Sec (Moira/Donna)	(ernest.mdalgo@luht.scot.nhs.uk)	#6308	21738
Mr Jim Powell Sec (Donna)	(j.j.powell@ed.ac.uk)	#6404	21715
Transplant secretary Donna Wright (donna.wright@luht.scot.nhs.uk)			21715
Transplant Unit Fax			21739

## Transplant Coordinators

Jackie Bradie (Jackie.bradie@luht.scot.nhs.uk)	# 6618	21706
Jen Lumsdaine (jen.lumsdaine@luht.scot.nhs.uk)	# 6824	21703
Christine Jansen (christine.jansen@luht.scot.nhs.uk)	# 6615	21704
Liz Waite	# 6530	21705
Alison Glover	# 6541	21727

## OTHER STAFF

		Bleep	Ext No
<b>Management</b>			
Service Manager	Julia Holmes		23640
Chief Nurse	Lee McGuinness		23642
Clinical Nurse Manager	Karen Adams		23641
Mngmnt Asst - Sandra Ogilvy	(sandra.ogilvy@luht)	5915	23618
<b>Haemodialysis</b>			
Dialysis Unit, RIE	Sr Johanna Mackenzie		21207
	(Room 1 21201; Room 2 21210; Room 3 21211; Room 4 21198)		
Dialysis Unit, SJH	Sr Janet Johnston/Chris Dixon		01506 523 887
	(01506 419 666 x 3994 dial; 3995 sister; 3996 docs)		
Dialysis Unit, WGH	Sr Jacqui Sneddon		31879/32566
Dialysis Unit, BGH			01896 826637
<b>Nursing</b>			
Ward 115	Sr Sarah Hardy	4008	21153
Ward 206	Sr Audrey Killeen	4029	21221
Education Coordinators	Anne Petherick	5607	21292
	Mags Campbell	5983	21215
Anaemia/Dial Coord - Wendy King	(wendy.king@luht)	5487	21214
Vasc Access - Rona Lochiel	(rona.lochiel@luht)	5983	21214
Conservative Mgt. - Johnny Webster	(john.webster@luht)	#6295	21218
<b>Community Dialysis Team</b>			
Office/ pager	pager 07659 5391 16		21219
Lisa Winfield	(lisa.winfield@luht.scot.nhs.uk)		
Katy Thomas	(katy.thomas@luht.scot.nhs.uk)		
Sharon Anderson	(Sharon.anderson@luht.scot.nhs.uk)		
<b>Dietitians</b>			
Susan Reed	(susan.reed@luht.scot.nhs.uk)	5790	21255
Hazel Elliot	(hazel.elliott@luht.scot.nhs.uk)		
Hazel Ferenbach	(hazel.ferenbach@luht.scot.nhs.uk)	5057	21254
Hayley O'Kane	(hayley.okane@luht.scot.nhs.uk)		
<b>Renal Social Work</b>			
Ann Murdoch			27850
<b>Pharmacists</b>			
Lorna Thomson	(lorna.thomson@luht.scot.nhs.uk)	8006	22908
Jane Pearson	(jane.pearson@luht.scot.nhs.uk)	2294	22909
<b>Administration</b>			
Appointments			21231
Ward Sec/Holiday Dialysis - Gary Friary	(gary.friary@luht.scot.nhs.uk)		21234
Computer Manager - Klavs Zarins	(klavs.zarins@luht.scot.nhs.uk)		21226
Asst Data Manager - Alison Stevenson	(alison.stevenson@luht.scot.nhs.uk)		

### Centralised email addresses

renal@ed.ac.uk – to Prof Turner's sec; messages will be redirected.  
 rie.renaladvice@luht.scot.nhs.uk – email advice line primarily for GPs.  
 holidaydialysis@luht.scot.nhs.uk - forwarded to the staff member responsible.

## OTHER CONTACTS

### Radiology

Inpatient X-ray	23719/20
A+E X-ray	23801/2
CT/MRI reception	23800
CT scanning room	23797
CT/MRI sec	23775
USS sec (Wilma)	23752
USS	23759
Portable XRs	page 2155
X-ray sec	23773
Film store	23728
CV labs (Permcath)	23788
Vascular labs OPD3	23605
Junior SpR o/c (USS)	#6186
Sr SpR o/c (CT)	#6187

### Admin

Haden (porters etc)	24242
IS helpdesk	85050
Apex password etc	23763
Medical records	28037/8
WGH Med records	31345

### Wards

115 desk	21151/58
115 docs	21152
117 desk	21171
206 base A	22061
206 base B	22067
206 base (TP)	22068
206 docs	21216/7
206 TP docs	22068

### Dialysis technical

John Ramsay	21251
Technicians	21249/50
Technician on call	07659 406486

### Fax numbers

Renal Unit	0131 242 1233
Ward 206	0131 242 1725
Ward 115	0131 242 2065
Transplant	0131 242 1939

### Labs

Combined labs	27777
BTS	27501
Pathology	27148
Bioch BMS (tech)	page 2221
Haem BMS (tech)	page 6550
Bacteriol BMS	page 2900
ViroI BMS	page 5981
Microbiol SpR	26028
Renal Microbiol SpR	26069
ECG/echo	21813/4
Endoscopy	21600
PFTs	21808

### Other specialities

Cardiology SpR	#6816
Dermatology SpR	#6396
Diabetes	#6800
EtOH liaison	21396
General surgery	page 2254
Haematology	#6466
Liaison psychiatry	21398
Liver/GI SpR	#6361
MoE/Stroke referrals	26927
O+G SpR	page 1625
Orthopaedics	page 2181
Pain team	page 5247
Palliative care	21993
Respiratory SpR	#6408
Urology	page 8181
Vascular SpR	#6440

## CLINICAL RESPONSIBILITIES

### Ward 206 (General Nephrology)

Consultant Ward Round times vary but Tue and Fri mornings are common. There is a multi-disciplinary meeting on Wednesday morning (time subject to change). On other days the FY2/CMT or equivalent is expected to do a ward-round at 0900hrs prompt, discuss problems with the covering middle-grader and inform nursing staff of management changes.

There is a ward phlebotomist and an evening filing clerk.

### Ward 115 and Acutes

Consultant Ward Rounds are done on Tuesday (usually) am 09.00hrs and Friday am 0900hrs (check start time with consultant). On other days the ward doctor ± registrar is expected to go round the patients. After review of patients on 115 and renal wards, patients referred from other wards will be seen. FY/CMTs are encouraged to go round for educational experience but have no responsibility for these patients.

Bloods on Ward 115 are normally done by the nurses.

### Immediate Discharge Letters and Summaries

SHOs are responsible for sending out Immediate Discharge Letters on the day of the patients discharge. These should be filled in by hand legibly - and given to the Ward Secretary to fax to the GP, a copy to pharmacy for the discharge prescription, a copy for the Case Notes. Proton drug screen should be updated at this stage.

### Discharges

Discharge planning is vital and should be done as early as possible to ensure that the process is smooth and arrangements for transport and home care packages etc are in place.

Update Proton immediately to show correct drugs, and check that there is a suitable problem list (but don't wipe out what someone else has carefully composed unless clearly appropriate). Important information for DP or dialysis unit review can be filled in 'Medical Comments'.

Diagnoses and procedures performed should be filled in at the end of the inpatient notes, for the discharge summary and for coding purposes. Add a short term PLAN if appropriate also. Dictated information need be given for more complicated admissions but may not be required for elective admission for simple, uncomplicated procedures such as tunneled line stripping, PD catheter line insertion, AV fistula formation, renal biopsy.

Discharge summaries must be dictated within two days of discharge.

- The name of the consultant normally responsible for the care of the patient (as an outpatient or on dialysis) must be indicated at the end of the discharge letter e.g., "Consultant X is responsible for outpatient care"
- For patients on peritoneal dialysis a copy of the discharge summary should be sent to the Community Dialysis Team

- For patients attending outpatient haemodialysis in
 

OPDA	Copy to Dr Dimova
WGH	Copy to Dr Whitworth
Borders	Copy to Dr Metcalfe
SJH	Copy to Dr Gibson
- And copies should be sent to the sister/ charge nurse in their dialysis unit.

Tapes should be given to the Ward Secretary to be typed up and sent out with a copy of the discharge letter after signature.

## **Middle Grade responsibilities and training**

SpRs rotate around 4 areas on a monthly basis

- Ward 115 and Acutes, including ITU and hospital referrals.  
SpRs are expected to follow the patients on Ward 115 and undertake rounds according to the experience of more junior ward doctors and by agreement with the consultant. They also review patients outside the unit who require renal support or decisions about need for renal replacement therapy and prescriptions need to be passed onto the Ward 115 and Acutes nursing staff as soon as practicable - the day before where possible.
- Transplant  
SpRs attend the ward rounds (joint medical and surgical) at 0830 hrs every morning (0900 Sat, 0930 Sun).
- Dialysis (and/or cover of annual/study leave). Help review patients on the hospital HD programme together with Dr M Dimova, and cover other HD units.
- Clinics – Middle grade attachments are arranged to provide continuity and breadth of experience, and will vary.

### **Training - SpRs**

- All SpRs are entitled to 1/2 day (Years 1-3) or full day off (years 4,5) per week to allow personal study, research or audit activities. This is an average not a fixed session, we aim to provide longer blocks.
- They are expected to attend all sessions of the East of Scotland training programme
- They are expected to gain practical experience in both native and transplant renal biopsy, and must complete DOPS for these and for line insertion.
- SpRs must arrange regular meetings with their educational supervisor to ensure appropriate planning, progress and assessment are made.

### **Training – Foundation and Core Medical Trainees**

A formal teaching programme is in place (current timing 12.30 Wednesdays). This and other aspects may change as MMC changes roll out.

	MONDAY	TUESDAY	WEDNESDAY	THURSDAY	FRIDAY	SAT/ SUN
8.30	Transplant Round	8.30 Transplant Round	8.30 Transplant Round	8.30 Transplant Round	8.30 Transplant Round	
9.00	Renal Clinic, WGH (CEW)	9.00 Consultant Ward Round 206 & 115	9.00 Transplant Clinic OPD1 (CEW)	9.00 Renal Clinic OPD4 (ANT); 1 <sup>st</sup> Thur, WM	9.00 Consultant Ward Rounds 206 and 115	9.00 (9.30 Sun)
9.00	Transplant Clinic OPD1 (MA)	9.00 St. John's Clinic (PHG)	9.00 Discharge Planning/ Med. Elderly Liaison Mfg 206	9.00 Haemodialysis MDT Meeting (MD, JG, JIN, RGP)	9.00 Transplant Clinic OPD1 (WM or CS)	Ward Rounds 115 & 206
9.30	Home Dialysis Clinic (JUN 1 <sup>st</sup> -3 <sup>rd</sup> PHG 2 <sup>nd</sup> -4 <sup>th</sup> )	9.30	9.30			
2.00 (2 <sup>nd</sup> Mon)	Monthly Dialysis Clinic (WGH satellite patients) OPD4 (CEW)	2.00	2.00 Renal Clinic OPD4 (JIN, JG, ADC/JH, RGP/DCK)	2.00 PD Meeting (2 <sup>nd</sup> Thur) HD Meeting (4 <sup>th</sup> Thur, Odd Months)		
4.00	Radiology (1 <sup>st</sup> + 3 <sup>rd</sup> )	3.00		3.00 Unit Meeting	3.30	
4.30	2 <sup>nd</sup> Transplant List (4.30)	Dialysis Moves Meeting	4.00	4.00 Pathology (1 <sup>st</sup> & 3 <sup>rd</sup> wks) Transplant M & M (4 <sup>th</sup> Week)	'Hand-over' Round Wd 206 Sem Room	

## UNIT TIMETABLE

See adjacent timetable.

## PATIENTS AND REFERRAL PATTERNS

### Dialysis Patients

There are about 250 patients on hospital haemodialysis and 75 patients on CAPD /APD. They are all instructed to phone the dialysis unit or wards for advice. If there is a definite or possibly dialysis-related problem, it is our responsibility to deal with it and see them if necessary. Occasionally it will be appropriate for them to be asked to see their GP for an intercurrent problem. If any dialysis patient is admitted to A&E or another ward then those units should contact the renal unit and arrangements should be made to transfer the patient if appropriate to the renal unit.

All patients on PD should be managed on the Renal wards except in exceptional circumstances.

### Pre-dialysis patients

If patients present with a problem related to their renal failure then they should be admitted to the Renal Unit. Otherwise it may be more appropriate for them to be admitted to other areas of expertise e.g. CCU, urology, orthopaedics. However the Renal Unit should (usually the acute team) liaise with the admitting unit and provide advice etc when necessary.

### Patients on immunosuppression

Have a very low threshold for asking these patients to attend for infection screen, wbc etc.

### Calls from GPs

GPs will frequently call for advice. Any GP requesting an urgent opinion should either be offered immediate assessment of the patient or an urgent clinic review depending on the situation. Discuss with senior if in any doubt. SHOs should usually discuss a problem with a senior member of staff before giving management advice to a GP. Encourage use of the email advice service ([rie.renaladvice@luht.scot.nhs.uk](mailto:rie.renaladvice@luht.scot.nhs.uk)) for non-urgent queries.

### Patients with hyperkalaemia

Following renal clinics, the biochemistry lab may contact the on-call FY/CMT doctor. If out-of hours and the **K>6.5mmol/l, the patient should be contacted and arrangements made for them to attend the ward** for a repeat blood test and appropriate management. If during working hours, then the doctor who saw the patient should be contacted wherever possible, and they should make the appropriate arrangements.

### Acute Referrals

These usually originate from within the hospital or from other hospitals in the region, and are made at SpR level. For RIE referrals, it is appropriate to first review the patient and decide where further management would be most appropriate.

For patients who are referred from other centres, if it is likely that dialysis or that an urgent renal biopsy and further management are required then arrangements should be made to transfer the patient to the renal unit as soon as possible. Otherwise advice may be given to the referring physician with or without a review of the patient. Do not hesitate to involve the relevant consultant in these discussions. Contact should be maintained with the referring hospital and if there is a deterioration in the patients condition then arrangements

can be made to transfer the patient up to the renal unit as soon as possible. If the patient sounds likely to require ITU care, plan this also with the consultant on call for Ward 118 to discuss the best point of admission and ensure that they are warned of a possible admission.

## **TEACHING**

### **Y4 clinical teaching**

Students attend in groups of up to about 14 for two weeks during their fourth year. Full details are provided elsewhere. Dr Gibson is the organiser.

### **Year 1 SSC**

In Year 1 groups of students do projects that ideally should involve some patient contact but require minimal medical knowledge. These are very rewarding to run. They culminate in a poster presentation. We aim to run 1-2 groups each year.

### **SSCs in Year 2**

Dr Phelps organises this course in which groups of 5-6 students explore a subject in depth in a largely self-directed manner for 4 weeks, to produce a website at the end. Potential tutors welcome (1-2 hours per week for 4 weeks). We aim to run at least 2-3 groups each year, but this can involve research staff.

### **SSCs in Year 4**

Twelve week solo projects, part-time. Arranged individually with potential supervisors. Useful audit, research and other projects can be undertaken in this time, leading to publication if successful. We aim to run several of these each year.

### **E-Learning**

Members of the unit have designed several useful and sometimes innovative packages for learning online. Most of these are available through EEMeC – if you are involved in teaching you can get a password for this (and need one also to make case reports). Talk to John Neary or Neil Turner if you have new ideas.

### **Other**

Some members of staff are involved in teaching in Years 1 and 2 of the new curriculum, in teaching on the Medical Assessment wards, and elsewhere.

## **BED POLICY AND ADMISSIONS**

### **Ward 206**

**All elective admissions must be arranged through the ward secretary on 21234**, who will draw up and circulate a weekly admissions list. If more than two admissions to ward 206 are arranged for any given day, consideration must be given to cancellation.

### **Transplant**

Patients with a functioning renal transplant should usually be admitted to Ward 117/206 for management of medical/surgical problems. Prospective recipients of cadaveric or living donor kidneys are admitted. When transplants fail, the care of the patient reverts to the renal team.

### **Medical Day Case**

The unit is open from Monday to Friday evening. Suitable admissions include those undergoing uncomplicated biopsies, angiography/angioplasty, vascular access radiology/intervention, vascular access or CAPD surgery. Medical factors (diabetes, warfarin therapy etc.) or the perceived potential for difficulties with the procedures may preclude the use of this ward. Admissions can be arranged by phone (21234) by the renal ward secretary. Even if you fix it, you should still notify the ward secretary, and liaise with the appropriate teams – e.g. surgeons, radiologists etc. An F-grade nurse practitioner admits and supervises these admissions. To assist with optimal patient management:

- informed consent should be obtained by the physician arranging the admission, preferably in the outpatient clinic
- pathology request forms should also be completed in the same way

## **GUIDELINES ON ADMISSIONS TO RENAL HIGH DEPENDENCY**

Ward 115 is for:

1. Patients requiring more intensive support and monitoring for renal failure, but excluding those needing advanced respiratory support;
2. Patients who can benefit from more detailed observation than can be safely provided on the nephrology/general ward;
3. Patients no longer needing intensive care, but who are not yet well enough to be returned to a general ward;
4. Post-operative patients who need close observations or monitoring for longer than a few hours;
5. All patients requiring continuous renal replacement treatments

All admissions should be discussed with and approved by the consultant in charge or on-call consultant.

If all 6 Renal beds are occupied, with nobody fit to be discharged to the ward, it may be possible to discuss admission of a patient to the non-renal beds on 115 or to the 116 High Dependency Area: in either case, contact Charge Nurse 116.

## PROTON

Proton is a renal data system that was created for renal units in the 1980s, one of the very first Electronic Patient Records (EPRs) in the world. It includes results, drugs, letters, and many other features. It still offers a very high degree of functionality in many units in the UK, although it antedated the computer mouse, the Internet, even the widespread use of PCs. Services are also provided for renal patients in Fife and for the Haemophilia and Scottish Liver Transplant Services. Data are downloaded automatically from Apex and from similar systems in other hospitals. For Transplantation there are links to Dundee, and UKTSSA in Bristol. Data are automatically sent to the Scottish Renal Registry in Glasgow. A local administrator can set up new screens and data flows, and query the system with 'Quarks' when new analyses are requested.

You interact with Proton via a terminal emulator installed on your PC, using the numeric keypad instead of a mouse to navigate the menus.

- Contact the Proton team to get a password, add new patients etc.
- Go to <http://proton.luht.scot.nhs.uk> for a detailed guide on how to use Proton.

## RENAL PATIENTVIEW

Renal PatientView (RPV) is a UK-wide project that gives patients access to selected data from their renal electronic record. Edinburgh was one of the pioneers in its introduction and several hundred patients have access. See [www.renalpatientview.org](http://www.renalpatientview.org)

Any patient who is registered on Proton can gain access to their results (refreshed daily) and information tailored to their main renal diagnosis and treatment (HD, PD, TP, none of these). It also shows drugs, clinic letters, and may expand further in the future. Their GP is sent a similar login.

Leaflets and posters should be available around the unit, or are available from Prof Turner's secretary. Consent forms can be downloaded from [www.edren.org](http://www.edren.org) – see the link just beneath the picture on the home page.

- Patients must sign up for this service, and can withdraw at any time.
- They are free to share their logins with friends and relatives, or in clinics etc elsewhere, and can change their passwords to remove these privileges.
- RPV is only likely to be of continuing value to patients likely to remain on our books for some time, rather than to people coming to clinic just once or twice.
- RPV combines data by CHI number, so data from other renal units that have RPV will be combined in a single record.
- Diagnosis-specific data depends on entering the appropriate EDTA diagnostic code (Patient Details – third screen along is 'Diagnosis')

**Staff** who would benefit from access to records when out of the hospital can have a login that permits them to see the records of all patients in the unit who have signed up. This can be very useful, but raises important issues. See the staff leaflet, and remember –

- Passwords must not be shared, and shouldn't be too easy to guess (use a mixture of lower and upper case letters and numbers in some memorable sequence)

**Local administrators** for the system are Neil Turner, Wendy Metcalfe and Alison Stevenson. Return sign-up forms to one of these, and contact them with any Qs.

## **'NOT FOR RRT' AND WITHDRAWAL OF RRT**

It is sometimes right not to provide RRT where it would not enhance the quality and/or duration of life.

Such decisions should be made explicitly, after discussion with relevant parties, and recorded. The level of the patient's involvement should be clear. GPs should always be involved in decisions concerning long term care, but not necessarily in acute illnesses where dialysis is being withheld primarily because of factors other than renal failure itself (e.g. other fatal illness). The views of relatives frequently inform a decision, and discussions should be noted, but relatives cannot make decisions on behalf of a mentally competent adult, or overrule a decision which is medically clearly correct.

Patients who are uncertain, or who have elected not to have dialysis, should be referred to the conservative care specialist nurse/team.

Further info on the web

## **DEATHS**

- The FY/CMT doctor should call the patient's GP when a patient dies
- And should inform appropriate secretary to cancel clinic
- Autopsies should be requested routinely, but especially in
  - long-term RRT patients
  - acute illnesses that are not fully understood
- Death certificates and cremation forms should usually be completed immediately
- Discuss with consultant what should be written
- Contact the Procurator Fiscal if in any doubt as to whether referral to them is appropriate. A booklet detailing these procedures should be kept with the death certificate books
- FY doctors should not complete Autopsy request forms. Requests for autopsy and completion of assigned forms must be done by more senior grades.

## CLINICS

### Arranging Appointments

General Nephrology			Ext No
RIE	Prof AD Cumming/Dr J Hughes	Wed pm	21231
	Dr RG Phelps/Dr DC Kluth	Wed pm	21231
	Prof AN Turner	Thur am	21231
BGH	Dr W Metcalfe	Mon am	21246
WGH	Dr CE Whitworth	Mon am	21232
St John's	Dr PH Gibson	Tues am	21232

See timetable for times of dialysis and transplant clinics

FY2/CMT doctors may occasionally be asked to assist and it is educationally valuable to attend if you can. SpRs will be attached to specific clinics for a period to enable continuity. Assistance with phlebotomy is provided in most clinics.

Results should be available on Proton by the end of the following day. Letters should be dictated soon after the clinic and the target for them *being received by GPs* is within 2 weeks of the clinic.

Patients have a nominated consultant. Their outpatient follow-up should be with that consultant. They may be admitted as inpatients under other consultants, but subsequent outpatient follow-up remains with the designated consultant. A copy of the discharge letter should be sent to that consultant. Appointments should be made via the above extension numbers.

**URGENTLY TELEPHONED ABNORMAL RESULTS FROM CLINIC** will be directed to the sender within working hours, and to the Renal Registrar out of hours. See above for correct procedures.

## **B: POLICIES AND PROTOCOLS**

## ACUTE RENAL FAILURE

### Acute renal failure 'screen'

All of these tests should be considered in patients with acute renal failure. Be selective, but keep an open mind even if the diagnosis appears to be clear. The list applies also to patients with a lesser degree of renal impairment who have an acute or immunological renal illness. The 'spot' indicates – do it in everyone.

FBC + plats, film, diff (ESR)	• film essential in ARF
CRP	• misleading, CRP replaces
Clotting screen	• additional tests if abnormal
Group & save	•
Biochemistry	•
Calcium	• even a high-normal Ca is abnormal
Myoglobin, CK	• if rhabdomyolysis possible
Blood cultures	• do in almost all with ARF of whatever cause
Other cultures	• wound, sputum, catheters etc
Hepatitis and HIV serology	• Urgent HepB + HIV if may need dialysis; but also for other disease
CMV/VZV	• If to be immunosuppressed; consider EBV and HIV also
ASOT/ throat swab/ other	• if post-strep GN possible
Other serology	• leptospires, syphilis, hantavirus, etc (rarely)
MSU	•
Bence-Jones protein	• patients $\geq 35y$ with poorly explained ARF
Urinary prot	• 24h or spot protein/creatinine ratio
CXR	•
Renal ultrasound	• USUALLY URGENTLY REQUIRED
ECG	• if > 40 or any risk factors for cardiac disease
Pulmonary function	• in systemic disease, acutely and after recovery
Immunoglobulins, prot elect	• in most patients
Complement	• in almost all
ANF, etc	• and DNA antibodies if ANF positive.
ENA	• if suspect interstitial nephritis or atypical SLE
Rheumatoid factor	
ANCA	• all possibly inflammatory disease
Anti-GBM	• all possible RPGN
Cryoglobulins	• if low C4 or otherwise suspected

## Management of acute renal failure

This section covers general management of acute renal failure only. Diagnosis, prevention and specific treatment are not discussed.

Initial management should comprise

- Optimization of circulation where there is any question of its adequacy
- Diagnosis of cause
- Removal of potential nephrotoxins (especially drugs)

Note that there is no evidence that dopamine is of benefit. There are some reasons to suspect that it may be potentially harmful as it impairs splanchnic perfusion. Loop diuretics may increase urine output in those with less severe degrees of renal failure, but there is no evidence that they improve outcome (requirement for or duration of dialysis, or mortality) and some evidence that they can be harmful. Most interventions tested in prevention of ARF after radiographic contrast administration are ineffective or harmful (e.g. loop diuretics), apart from fluid administration alone: and N-acetylcysteine may at least do no harm (see Radiology). Inotropes may be valuable in shock or heart failure.

Indications for dialysis are:

- Pulmonary oedema, or severe volume overload with oliguria
- Hyperkalaemia
- Acidosis
- Symptoms
- Worsening figures with no prospect of early reversal
- Pericarditis

Neither age nor comorbid conditions (that might lead you to question longterm RRT) should be considered as automatically disqualifying dialysis for ARF if there is a substantial chance of recovery. BUT:

- avoid dialysis if aggressive treatment is otherwise inappropriate
- remember that exposure to dialysis membranes may prolong ARF
- there is no evidence that early dialysis improves outcome

### Peritoneal dialysis

Is now rarely used for ARF in the UK, though it can be effective if the patient is not too catabolic and ultrafiltration requirements not too extreme.

### Continuous or very slow treatments (haemofiltration or haemodialysis)

- are better tolerated in haemodynamically unstable
- permit large and variable volumes of fluid removal
- are preferred in patients with encephalopathies

#### **BUT**

- involve continuous anticoagulation
- prolong exposure of patient to artificial membranes
- do not achieve better outcomes
- can also under-provide small molecule clearance (continuous treatments rarely are continuous)

### Intermittent haemodialysis

Patients with ARF need at least as much dialysis, and usually more (because of catabolism) than patients with ESRF. Therefore Kt/V or URR should be at least as good. Dialysis usually need to be more frequent and daily treatments should be regarded as the norm in the early phase, or if fluid fluxes are

substantial (e.g., from feeding).

### Preventing disequilibrium

Disequilibrium is a state of clouding of consciousness, confusion and sometimes fits following dialysis. Disequilibrium is most likely:

- in patients at ESRF after prolonged CRF
- when urea, creatinine etc are very high
- in patients with cerebral disease and in the elderly

If the risk is significant it is sensible to give a low-clearance (eg Kt/V 0.5, or 30% URR) and low intensity (low blood flow and/or small dialyser) treatment initially, intermediate the next day, a full treatment (eg Kt/V 1.2, URR 70%) the third day, if figures permit.

A 'gentle start' is inappropriate if fast removal of small molecules is required – e.g. in severe hyperkalaemia, or for removal of low molecular weight poisons such as salicylate. CVVH is also inappropriate in these circumstances (unless the toxin is of molecular size better removed by haemofiltration).

First dialysis treatments can and should be less cautious in catabolic patients in whom figures are rising fast.

### Haemofiltration

Haemofiltration, whether continuous or intermittent, is less efficient at removal of small molecules including toxins. Prolonged haemofiltration commonly leads to phosphate depletion, replacement may be required, and it clears some drugs (e.g., vancomycin) faster than haemodialysis.

### Amount of dialysis

Detailed calculation of dialysis dose is beyond the scope of this summary, but see the brief description under 'Haemodialysis'. Note that for haemofiltration, Kt is equal to (or for urea, >90% of) the total volume of fluid exchanged.

**Comparisons:** For very crude comparison of small molecule clearance by continuous versus intermittent treatments, the following figures are provided. HD figures are for urea clearance by F8 dialyser, ignoring UF.

Modality	Urea clearance
Normal GFR	150 l/day
Daily intermittent HF	15-25 l/day
Continuous HF @ 1 l/hr	24 l/day
Continuous HF @ 2l/hr	48 l/day
Daily HD x 4hr @ $Q_B = 200\text{ml/min}$	46 l/day

### Dietary Management

Is important, and is described further in the DIET section.

### Other Treatment

Patients with acute renal failure should receive H2-blockers or PPIs. Prophylaxis against DVT should usually be used in bed-bound patients.

## ANAEMIA – ERYTHROPOIETIN, IRON, TRANSFUSIONS

### Identification of anaemic patients for Erythropoietin (Epo) treatment

All patients with chronic anaemia associated with chronic kidney disease (CKD) should be investigated for possible treatment, irrespective of the stage of kidney disease and the requirement for renal replacement therapy.

Anaemia should be investigated in patients with CKD when Hb falls below:

- <11.5 g/dl in adult female patients
- <13.5g/dl in adult male patients
- <12.0g/dl in adult male patients >70 years

### Targets for Hb

The British Renal Association, in their 2002 standards document stated that dialysis patients with chronic renal failure (CRF) should achieve a haemoglobin of 10g/dl within six months of being seen by a nephrologist, unless there is a specific reason. To achieve this target it is necessary to aim for a Hb of 11-12.5g/dl with a range of 10-14g/dl.

### Prescribing

EPO for hospital-based patients is currently prescribed from the hospital pharmacy. Community based patients (Pre-dialysis, failing transplant and PD) can receive EPO from the hospital and their GP via a shared care agreement – willingness of the GP to proceed should be confirmed for patients on PD or pre-end stage.

First prescription of EPO is a decision for senior staff. It should only be considered in PD patients after an initial stabilisation period on dialysis of three months, as there is often a spontaneous rise in Hb.

### Assessing iron status

Is difficult and there is not a single fool-proof method. Ferritin <50ug/l unequivocally proves iron deficiency, and <100ug/l is very likely to do so. As Ferritin is an acute phase protein, levels may sometimes be higher even in the presence of functional iron deficiency, and a level of <300ug/l (or Transferrin Sat [Tsat] <20%) should probably lead to IV iron therapy if there is an inadequate response to usual doses of EPO. Expect most patients to run along at least at 100ug/l.

There is evidence to suggest that if Ferritin levels are within normal limits (50-70µg/l), or if the patient is on haemodialysis, the rate of absorption of oral iron supplementation is not sufficient to supply the need of iron for Hb production.

Transferrin saturation (Tsat) is a measure of the ability to mobilise stored iron for red cell production. It can be calculated by

$$\text{TSAT (\%)} = (\text{serum Fe/Total Iron Binding Capacity [TIBC]}) \times 100$$

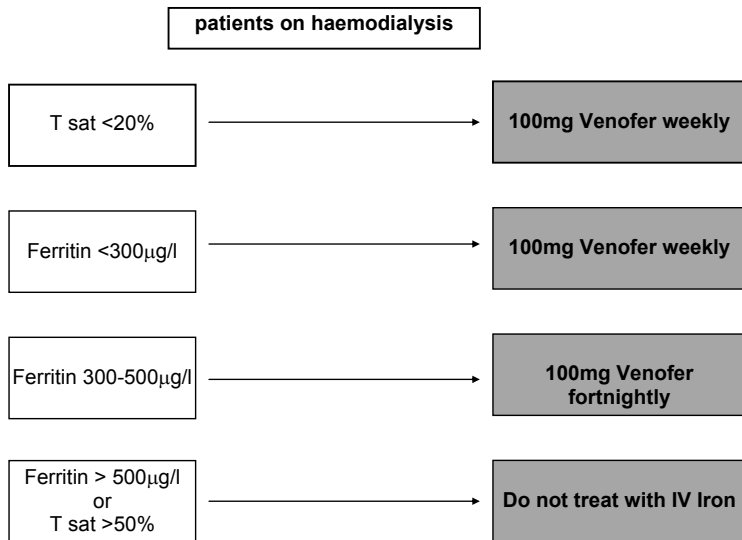
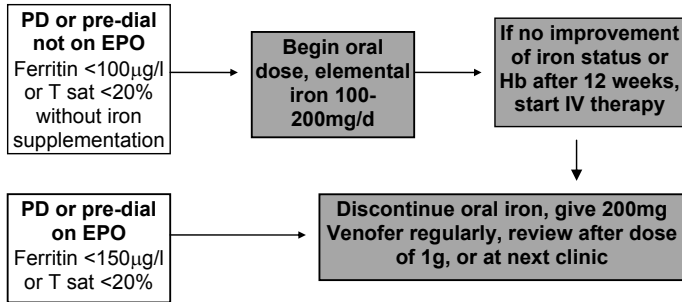
If TSAT < 20% this is indicative of functional iron deficiency even if the ferritin levels are adequate. This may respond to treatment with IV iron.

## Short protocol for Erythropoietin use

- 1. Indication:** Hb consistently below 11 g/dl<sup>1</sup>
- 2. Contra-indication:** Uncontrolled hypertension
- 3. Before starting, check:** For GI / other blood loss  
 FBC / MCV reticulocyte count  
 Ferritin, transferrin saturation, folate & B12  
 Dialysis adequacy  
 PTH, Al<sup>3+</sup>, CRP & TFTs
- 4. Starting dose:** **Haemodialysis:**  
 Epoetin alpha (Eprex)  
 150 units / kg body weight / week  
 as 3 divided doses, intravenously
- Peritoneal or pre-dialysis:**  
 Epoetin beta (Neo-Recormon)  
 100 units / kg body weight / week  
 as 2 divided doses, subcutaneously
- 5. Target Hb:** Population target range: 10 – 12.5 g/dl<sup>2</sup>  
 Hb. should not be allowed to rise above 14 g/dl
- 6. Monitoring:** The intervals (in weeks) between checking Hb & iron studies are:
- |              | Induction | Stable | Dose↑ | Dose↓ |
|--------------|-----------|--------|-------|-------|
| Hb           | 1-2       | 4      | 2     | 2     |
| Iron studies | 4         | 12     | 4     | 4     |
- \* = until stability confirmed
- 7. Blood pressure:** BP is checked every time epoetin is administered.  
 A BP over 170/95mmHg should be discussed with medical staff, but EPO should not be withheld.
- 8. Dose adjustment:**<sup>3</sup> **Induction period** ( Hb <11 g/dl )  
 a. rate of rise ≤ 1 g/dl/month: ↑ weekly dose by 25%  
 b. rate of rise of Hb ≥1.5 g/dl/month: ↓ weekly dose by 25-50%
- Stable period** (Hb >10 g/dl)  
 a. if Hb exceeds 12.5 g/dl ↓ weekly dose by 25-50%  
 b. if Hb falls below 10 g/dl ↑ weekly dose by 25-50%
- 9. Stopping EPO:** If EPO is stopped because of a sharp rise in Hb it must be recommenced at a lower dose 2 weeks after discontinuation.
- 10. EPO Resistance:** EPO doses ≥250units/kg/week<sup>4</sup> should be discussed with senior staff, patients should be investigated for causes of EPO resistance. This should include a retic count.

- 1 Recommended starting Hb in European Best Practice Guidelines (EBPGs) quoting evidence level of A.
- 2 Caution is advised in taking Hb above 12 g/dl in patients with diabetes, particularly those with peripheral vascular disease; evidence level C.
- 3 Modified from EBPGs, evidence level C.
- 4 Definition of EPO resistance in EBPGs is 300 units/kg/week.

## Treatment of iron deficiency – short protocol



## Treatment of iron deficiency

Patients on Epo therapy, and patients on haemodialysis, will almost always require a higher degree of iron replacement than can be delivered by oral supplementation in order to maintain the haemoglobin levels that are recommended. Some pre-dialysis, transplant and peritoneal dialysis patients will not need that level of iron replacement therapy, and oral iron supplements will be sufficient for them. Some patients may not need any iron supplements at all. Oral iron should be discontinued when patient is receiving IV iron. We use iron saccharate (Venofer™) when intravenous iron is required. IV iron therapy should be ongoing, rather than intermittent treatments, when a need has been identified.

Patients who have never had Venofer™ before must be given a supervised test dose before commencing a course of treatment. Give 20mgs over 1-2mins (more conveniently as part of infusion). Wait 15 mins. Proceed with remainder of dose thereafter. *Note that it is sensible to give at least a single dose to all patients who are to move to satellite unit dialysis, so that treatments can be continued there if necessary, without the need to return to the centre for a test dose.*

Severe allergic reactions are rare but when they occur are usually anaphylactoid. Discontinue infusion; give 0.5ml 1:1000 adrenaline i.m, and oxygen as initial treatment if this occurs followed with chlorphenamine and hydrocortisone as required.

Patients on haemodialysis can receive IV iron whilst on dialysis, at any time during the procedure. Those requiring iron who are non dialysis patients can receive 200 mgs (Venofer™) in ward or clinic as a slow bolus IV injection (20mgs (1ml) per minute, or diluted with 100mls N saline and given over 30 minutes on the basis of their last iron studies). Blood should be taken for repeat iron studies before it is given.

*The full protocol for the administration of Venofer™ should be referred to. Available on web.*

## Blood transfusions

Transfusions should be avoided as far as possible in patients who are on (or who in the future may be on) the transplant list.

All blood which is given to patients with renal disease who may be transplant candidates, should be treated to remove white blood cells. The Blood Transfusion Service now routinely provides leucocyte-depleted products, thanks to BSE and nvCJD. The white cell content of each unit will be less than  $5 \times 10^6$ .

## ANTMICROBIAL POLICY

### Common infections

The following are all initial recommended therapy pending microbiology reports. Treatment should be changed according to sensitivities. An asterisk\* means adjust dose for renal function; see below.

Infection	Recommendations	Duration	Comments
<b>UTI</b>			
uncomplicated	Co-amoxiclav 375mg tid If penicillin allergic	3 days	
systemic upset	Ciprofloxacin 250mg bd	3 days	if hospital acquired
	Co-amoxiclav 375mg tid Or ciprofloxacin 250mgbd	10-14 days	
prophylaxis	Co-amoxiclav 375mg at night or Cephalexin 250mg at night	6 months	relapse or reinfection seek specialist advice
<b>Pneumonia</b>			
Community Acquired	Amoxicillin 500mg tid * oral Penicillin allergic Clarithromycin 500mg bd *		IV therapy only in patients who are severely ill
Hospital Acquired	Ceftriaxone 1-2 g* plus clarithromycin 500mg bd iv *		
Aspiration	Co amoxiclav 1.2g tid iv* + metronidazole 500mg tid*		Clarithromycin if penicillin allergy

<b>Septicaemia</b>			
	Gentamicin iv * + amoxicillin iv 1g tid * +metronidazole iv 500mg tid *		
<b>Cellulitis</b>			
Serious Infection	Amoxicillin 500mg tid oral* Flucloxacillin 500mg qid oral  Flucloxacillin 1g qid iv		Clarithromycin if penicillin allergy

<b>Clostridium difficile</b>			
	metronidazole 400mg tid oral  vancomycin 125mg qid oral	10 days	if no response to metronidazole
<b>Candidiasis</b>			
Oral	Nystatin 100,000u 1ml qid		
Vaginal	Clotrimazole 500mg PV Fluconazole 150mg oral	stat stat	for recurrent infection

<b>Line and Exit Site Infections - HD and CAPD catheters</b>			
uncomplicated	Flucloxacillin 500mg qid oral	7 days	clarithromycin if penicillin allergy
systemic upset/sepsis	Flucloxacillin 500mg qid iv + gentamicin 1.5 mg/kg *		clarithromycin if penicillin allergy dose gentamicin according to blood levels
<b>A-V fistula infection</b>			
	Flucloxacillin 250mg qid oral Penicillin V 250mg qid oral		IV if indicated

<b>PD peritonitis</b>			
Bacterial	Vancomycin 30mg/kg as single dose, 6 hour dwell. + Ciprofloxacin oral 500mg twice daily		Do not measure vanc. levels, send fluid for WCC, gram stain and culture. Change APD to standard 4-exchange CAPD
Fungal	amphotericin 0.5mg/l/ exchange IP plus Flucytosine 50mg/l/exchange	Can be up to six weeks	The priority is usually catheter removal, and this is then temporising therapy. See section on PD peritonitis.
Yeast	Oral fluconazole 200mg	2 weeks	
<b>Nasal Carrier</b>			
Staph Aureus	Mupirocin 2% bd to both nostrils	5/7 per month indefinite	Screen PD pt prior to catheter insertion; treat if 2/3 swabs positive
MRSA	Mupirocin 2% tid to both nostrils	5/7 per month indefinite	Screen PD pt prior to catheter insertion; treat if 2/3 swabs positive
<b>Hepatitis Immunisation</b>			
Hepatitis B	HBvaxPRO 40mcg/ml	0,1 and 6 months	All patients on RRT or for whom RRT is likely should be immunised. Booster if level <10 at 8 months

## Dosage reduction required for Renal Failure

(For further advice contact clinical pharmacist - bleep 8006/2294)

Drug	Creatinine Clearance ml/min	Dose	Comments
Aciclovir IV	25-50 10-25 <10 dial	5-10 mg/kg 12hrly 5-10 mg/kg daily 2.5-5mg/kg daily IV 2.5-5mg/kg daily IV	On HD days give a dose after dialysis, not during/just before
Oral	10-20  <10 dial	200mg 6-8hrly 400-800mg 8hrly (zoster) 200mg 12hrly (simplex) 400-800mg 12hrly (zoster) 200/400mg 12hrly	Give after HD
Amoxicillin	<10 ml/min	250mg tid	On HD days give a dose after dialysis, not during/just before
Benzylpenicillin	10-20 <10 or dial	75% normal dose 20-50% normal dose max 3.6g per day	On HD days give a dose after dialysis, not during/just before
Ceftazidime	31-50 16-30 6-15 <6 or dial	1g bd 1g daily 0.5-1g every 24hr 500mg-1g every 48hrs	On HD days give a dose after dialysis, not during/just before
Cefotaxime	<10 or dial	0.5- 1g 8-12 hourly	On HD days give a dose after dialysis, not during/just before
Clarithromycin	< 10 or dial	250mg bd Oral/IV	On HD days give a dose after dialysis, not during/just before
Ciprofloxacin	<20 or dial	100mg bd IV 250mg bd oral	On HD days give a dose after dialysis, not during/just before
Co Amoxiclav	10-30  <10 or dial	1.2g 12 hourly IV or 375mg 8hrly oral 1.2g stat then 600mg- 1.2g every 12hrs 375mg 8hrly oral	On HD days give a dose after dialysis, not during/just before
Flucloxacillin	<10 ml/min	as in normal renal function max 4g daily	
Flucytosine	20-40 10-20 <10 or dial	50mg/kg 12 hourly 50mg/kg every 24h 50mg/kg once, then by levels	Aim for trough 25-50microg/l (0.5-1g doses normally adequate)
Gentamicin	<20	1.5mg/kg (after dialysis if on HD)	Dose interval according to levels, see next page

Meropenem	10-20 <10 or dial	500mg 8 hourly 500mg daily	On HD days give a dose after dialysis, not during/just before
Metronidazole	<10	500 mg IV bd or 400mg bd oral	Recommended no reduction if on dialysis, but give dose after, not during/just before
Trimethoprim	15-25  <15 or dial	200mg bd for 3 days - then 100mg daily 100mg daily	On HD days give a dose after dialysis, not during/just before
Vancomycin		15mg/kg for IV 30mg/kg for PD fluid	Dose interval according to levels (except in PD use), see below.

**CrCl / eGFR** – for historical reasons manufacturers have made dosage recommendations by CrCl rather than eGFR. Using eGFR is usually more accurate than using estimated CrCl in most *stable* patients. eGFR DOES NOT ACCURATELY INDICATE RENAL FUNCTION IN ARF or severe illness, or in unusual circumstances (amputation, wasting). The same applies to Cockcroft-Gault or other estimates of CrCl. Patients with changing renal function are particularly likely to be over- or under-dosed and treatments should be reviewed frequently.

**Dialysis** Note that 'dialysis' in the table above assumes minimal residual native renal function. In general, drugs that are removed by HD or HDF should be administered after a treatment. Some drugs (e.g. vancomycin) may be removed by haemofiltration even though they have negligible clearance by conventional dialysis. Check with pharmacists or a reference source if in doubt.

## Therapeutic drug monitoring

<b>VANCOMYCIN</b>	PEAK 20-30 mg/l TROUGH <10mg/l
Peritoneal fluid single dose	Do not measure levels
IV treatment	<p>Take PEAK level 2 hours after the end of the FIRST infusion</p> <p>Take 2nd level 24 hours after the start of the infusion</p> <p>From these levels it is possible to predict when the blood level will be under 10mg/L</p> <p>Check blood level and redose</p> <p>Vancomycin is not removed by dialysis but it is removed by haemofiltration, shorter dosing intervals required on CVVH.</p>
<b>GENTAMICIN</b>	PEAK 5-10 mg/ml TROUGH <2mg/ml
IV treatment	<p>Check PEAK 1 hour after injection/infusion</p> <p>Take 2nd level 24 hours after the injection</p> <p>From these levels it is possible to predict when the blood level will be under 2mg/l</p> <p>Check blood level and redose</p> <p>Gentamicin is removed by dialysis (one dialysis session approximately equal to one half-life).</p>

## ANTICOAGULATION

### Initiation of full anticoagulation with heparin

In patients with renal failure the use of unfractionated heparin is still recommended for full anticoagulation. The use of LMW heparins in fully anticoagulating doses is not recommended as their action is prolonged and may be potentiated by uraemic bleeding tendency.

- Give bolus of intravenous heparin 5,000 units (5ml of Pumphep, which contains 1000 units/ml). For a major pulmonary embolus give 10,000 units (10ml Pumphep)
- Set up intravenous infusion of heparin at 1250 units/h (1.25ml/h Pumphep)
- Check APTT ration after 2-6h. Therapeutic range is 1.5 - 2.5
- Monitor platelet count on alternate days

<b>Adjust heparin infusion rate as follows:</b>		
<b>APTT ratio</b>	<b>Heparin infusion (1000 units/ml)</b>	<b>Recheck APTT</b>
> 5.0	Stop for 1h, then decrease by 500 units/h	2-6h
4.1 - 5.0	Decrease by 300 units/h (0.3ml Pumphep/h)	2-6h
3.1 - 4.0	Decrease by 100 units/h (0.1ml Pumphep/h)	2-6h
2.6 - 3.0	Decrease by 50 units/h (0.05ml Pumphep/h)	2-12h
1.5 - 2.5	No change	within 24h*
1.2 - 1.4	Increase by 200 units/h (0.2ml Pumphep/h)	2-12h
< 1.2	Decrease by 400 units/h (0.4ml Pumphep/h)	2-6h

\* Sooner if APTT has been unstable

- Checking APTT daily is the mandatory minimum frequency for all patients receiving intravenous heparin.
- Continue heparin until oral anticoagulant is established and the international normalised ratio (INR) is stable within the appropriate therapeutic range.

### Special circumstances

- Urgent reversal of anticoagulation: contact haematologist.
- Lupus anticoagulant may render APTT results meaningless, and require more complex assays.
- For further information on these or other issues please contact the consultant haematologist.

## Warfarinisation

<b>Schedule for induction of warfarin therapy</b> From Fennerty et al, Br Med J (1988) 297:1285-5		
<b>Day</b>	<b>INR</b> (best taken 0900-1000)	<b>Dose in mg</b> (best given 17-1800)
<b>1</b>	<1.4 This schedule is not applicable if INR is 1.4 or higher - give smaller loading dose	10
<b>2</b>	<1.8 1.8 >1.8	10 1 0.5
<b>3</b>	<2.0 2.0-2.1 2.2-2.3 2.4-2.5 2.6-2.7 2.8-2.9 3.0-3.1 3.2-3.3 3.4 3.5 3.6-4.0 >4.0	10 5 4.5 4 3.5 3 2.5 2 1.5 1 0.5 zero
<b>Day</b>	<b>INR</b>	<b>Predicted maintenance dose (mg)</b>
<b>4</b>	<1.4 1.4 1.5 1.6-1.7 1.8 1.9 2.0-2.1 2.2-2.3 2.4-2.6 2.7-3.0 3.1-3.5 3.6-4.0 4.1-4.5	>8 8 7.5 7 6.5 6 5.5 5 4.5 4 3.5 3 miss out next day's dose then give 2mg
<b>4</b>	>4.5	miss two doses then give 1mg

## Alternatives to heparin

These may be used in patients with heparin-induced thrombocytopenia (HIT) or sometimes in those at high risk of haemorrhage.

**Epoprostenol** (prostacyclin, Flolan®) is a potent vasodilator that inhibits platelet aggregation. A half-life of about 3 mins means that it must be given by continuous infusion but that its effects wear off quickly. Hypotension, flushing, headache, nausea and vomiting, and other symptoms may occur.

The freeze-dried drug is diluted first with the accompanying diluent and then with saline to 2000 nanograms/ml. Infusion rate is 1-5ng/kg/min, gradually building up the dose over 30 mins before connecting to the machine. At tolerated doses it is usually found to be less effective than heparin at preventing clotting in extracorporeal circuits.

**Danaparoid** (Orgaran®) is a heparinoid. In the UK available on a named-patient basis. Acts by blocking factor Xa. Has a prolonged action that is greater in renal failure because it is normally renally excreted; monitor Xa levels. Difficult to reverse. Side effects are similar to heparin. In HIT, cross-reactivity may occur in 10%. The following has been used during haemofiltration (an unlicensed indication):

- Bolus 2500U i.v. then a continuous infusion at 600U/h for 4h, then 400U/h for 4h, then 200-600U/h to maintain anti-Xa levels at 0.5-1.0U/ml.
- If patient is <55kg, bolus 2000U should be followed by 400U/h for 4h then 150-400U/h. Further instructions from renal pharmacist (or kept on renal HDU in Edinburgh).

**Lepirudin** is a recombinant hirudin (anti-thrombin). Activity can be monitored by APTT (aim for ratio 2.0-3.0 versus control) but this does not correlate precisely with plasma hirudin levels. Prolonged excretion in renal failure means that full anticoagulation is practically continuous for patients on alternate day dialysis, and there is no easy way of reversing the anticoagulation. Side effects related to anticoagulation but also fever, allergy, and injection site reactions. Use in haemodialysis is not surprisingly unlicensed but the following protocol has been used:

- 0.08-0.1 mg/kg for first dialysis as slow i.v. bolus followed by 50% of this dose at subsequent dialyses.

A hepatically metabolised hirudin with shorter half-life is in development.

## Heparin-induced thrombocytopenia (HIT-II)

This is caused by platelet factor 4 antibodies, which can activate platelets. Existing tests often give positive results in patients who do *not* have the syndrome of:

- Low platelets
- Thrombotic events (venous and arterial)

In patients at highest risk, the acutely ill, there are usually alternative possible explanations for thrombocytopenia. Because current alternatives to heparin may be hazardous in themselves, it is important to consider the balance of probabilities and risks.

Reference: The Heparins: all a nephrologist should know. NDT 20:2036-42 (2005) (Hetzl & Suker)

## BLOOD PRESSURE IN RENAL DISEASE

There is strong evidence that 'lower than usual' targets are beneficial in renal diseases, but especially in those associated with significant proteinuria. Any limits set are arbitrary, but for example, the UK CKD Guidelines recommended target maximum BPs as shown below. These are very close to other recommendations.

- **130/80** for patients with diabetes mellitus and microalbuminuria (but note that diabetics with microalbuminuria benefit from ACE inhibitors at all levels of blood pressure, including normal levels)
- **130/80** for non-diabetic patients with chronic renal failure
- **125/75** for those with chronic renal failure of any aetiology if they also have proteinuria >1g/d (Prot/Creat ratio > 100mg/mmol), unless this lower target is contraindicated.

ACE inhibitors are proven to be particularly effective at protecting renal function in patients with proteinuria. A2R antagonists are likely to be equally effective. Non-dihydropyridine calcium antagonists (verapamil, diltiazem) have some theoretical (not proven) advantage if patients cannot tolerate ACEI or A2R blockade.

Blood pressure targets should be individualised, as patients have different circumstances. For example, patients of black race should possibly have lower targets as the risk of end organ damage is greater. Very young patients may merit lower targets.

It is sometimes useful to consider average blood pressure at different ages – although it must be noted that there is no evidence to support using these as therapeutic targets:

	Age	18-24	25-34	35-44	45-54	55-64	65-74
MALE	Systolic	125	128	128	134	141	145
	Diastolic	62	69	74	79	80	78
FEMALE	Systolic	117	117	121	130	139	149
	Diastolic	62	66	70	73	74	73

Figures are for Scotland, 1998

## CODING (RENAL DISEASE INDEX)

The ERA-EDTA codes are used for registering patients on the Proton database. They have a number of shortcomings and anomalies but we are stuck with them. These are the 1995 version.

<b>Primary renal disease</b>	<b>CODE</b>
<i>Primary glomerulonephritis</i>	
Glomerulonephritis; histologically NOT examined	10
FSGS with nephrotic syndrome in children	11
FSGS with nephrotic syndrome in adults	17
IgA nephropathy (proven by IF, not code 76 and not 85)	12
Membrano-proliferative GN; type I (proven by IF and/or EM - not code 84 or 89)	15
Dense deposit disease; MPGN type II (proven by IF and/or EM)	13
Membranous nephropathy	14
Crescentic (extracapillary) glomerulonephritis (type I, II, III)	16
Glomerulonephritis; histologically examined, not given above	19
<i>Pyelonephritis</i>	
Pyelonephritis associated with neurogenic bladder	21
Pyelonephritis due to congenital obstructive uropathy with/without vesico-ureteric reflux	22
Pyelonephritis due to vesico-ureteric reflux without obstruction	24
Pyelonephritis due to acquired obstructive uropathy	23
Pyelonephritis due to urolithiasis	25
Pyelonephritis due to other cause	29
Pyelonephritis - cause not specified	20
<i>Interstitial nephritis</i>	
Nephropathy (interstitial) due to analgesic drugs	31
Nephropathy (interstitial) due to cis-platinum	32
Nephropathy (interstitial) due to cyclosporin A	33
Drug induced nephropathy (interstitial) not mentioned above	39
Lead induced nephropathy (interstitial)	34
Gout nephropathy (urate)	92
Nephrocalcinosis and hypercalcaemic nephropathy	93
Interstitial nephritis (not pyelonephritis) due to other cause, or unspecified (not mentioned above)	30
<i>Familial/hereditary renal diseases</i>	
Polycystic kidneys; adult type (dominant)	41
Polycystic kidneys; infantile (recessive)	42
Medullary cystic disease; including nephronophthisis	43
Cystic kidney disease - other specified type	49
Cystic kidney disease - type unspecified	40
Hereditary/Familial nephropathy - type unspecified	50
Hereditary nephritis with nerve deafness (Alport's Syndrome)	51
Cystinosis	52
Primary oxalosis	53
Fabry's disease	54
Hereditary nephropathy - other specified type	59

<b>Primary renal disease</b>	<b>Code</b>
<i>Congenital diseases</i>	
Renal hypoplasia (congenital) - type unspecified	60
Oligomeganephronic hypoplasia	61
Congenital renal dysplasia with or without urinary tract malformation	63
Syndrome of agenesis of abdominal muscles (Prune Belly)	66
<i>Vascular diseases</i>	
Renal vascular disease due to malignant hypertension	71
Renal vascular disease due to hypertension	72
Renal vascular disease due to polyarteritis (include MPA)	73
Renal vascular disease - due to other cause (not given above and not code 84-88)	79
Renal vascular disease - type unspecified	70
<i>Secondary glomerular/systemic disease</i>	
Cryoglobulinemic glomerulonephritis	78
Diabetes glomerulosclerosis or diabetic nephropathy	80
Myelomatosis/light chain deposit disease	82
Amyloid	83
Lupus erythematosus	84
Henoch-Schoenlein purpura	85
Glomerulonephritis related to liver cirrhosis	76
Wegener's granulomatosis	74
Goodpasture's Syndrome	86
Systemic sclerosis (scleroderma)	87
Haemolytic Uraemic Syndrome (including Moschowitz Syndrome)	88
Multi-system disease - other (not mentioned above)	89
<i>Miscellaneous</i>	
Tubular necrosis (irreversible) or cortical necrosis (not code 88)	90
Tuberculosis	91
Gout	
Nephrocalcinosis and hypercalcaemic nephropathy	
Balkan nephropathy	94
Kidney tumour	95
Traumatic or surgical loss of kidney	96
Other identified renal disorders	99
Chronic renal failure; aetiology uncertain	00

Death and malignancy codes can be viewed online on the electronic version of this handbook, and downloaded from the Scottish Renal Registry Website.

## DIET

### Documentation on the Renal Database

Two Proton screens are maintained and updated by Dietetic staff:-

- Nutritional status
- Dietary therapy

Malnutrition or undernutrition is prevalent in patients with renal disease, the prevalence increasing as GFR falls. The cause is multifactorial but intake often improves on starting dialysis. However malnutrition remains common on dialysis, where it is a strong predictor of mortality.

### Constituents of food important in renal disease

#### Protein

In CKD our policy is to estimate protein intake, and make dietary recommendations to achieve intake in the range 0.8-1g/kg of ideal body weight. This is not a low protein diet, but may in some patients involve a reduction in intake. In others it will require increased intake.

On HD intake is increased to 1-1.2g/kg ideal body weight (ibw) to compensate for small increased losses and a tendency to under-nutrition.

On PD intake is increased to >1.2g/kg to compensate for peritoneal protein losses, which are variable but at times high.

A typical daily intake in the UK is 60-80g; normal requirement is only 45-55g (assuming adequate energy intake).

The main sources of animal protein are meat, fish, cheese, eggs, and milk; and of non-animal protein are nuts, beans, pulses, soya products.

Food	Protein content (g)
500ml cow's milk	17
100g meat, poultry, cheese, nuts	25-30
100g fish	20
1 egg	8
1 yogurt	7
135g baked beans (small tin, in tomato sauce)	7

#### Sodium

Typical daily intake in the UK is 150 - 200mmol. Daily requirement is less than half of this. Only around 10% of this is naturally-occurring in fresh/food - the remainder is added in cooking and food processing or as table salt after cooking.

Salt substitutes mainly consist of potassium chloride and are therefore not usually suitable for patients with renal failure.

For almost all renal patients without extra losses we recommend an intake of 80-100mmol/day. We refer to this as 'no added salt' but it also requires avoiding pre-added salt. Lower intake than this is probably desirable but

may compromise energy intake.

### **Fluid**

It is impossible for patients to comply with fluid restrictions if their salt intake is high.

HD – urine output plus 500mls/d

PD – normally urine output plus 750mls/d, but depends on ultrafiltration.

### **Potassium**

Typical daily intake in the UK can vary from 50 to 150mmol. Intake should only be limited if blood tests show it's necessary, as the fruit and vegetable contribution to potassium intake is important for general health.

Potassium is found in many foods but particularly high in fruit, fruit juice, and potatoes and vegetables which have not been boiled.

CKD – restriction not usually required until GFR<20, unless on ACE inhibitors, and their continuation thought important.

HD – most patients require some restriction.

PD – some patients require restriction.

### **Phosphate and Calcium**

Typical daily intake of phosphorus in the UK is 35-40mmol.

Phosphate is commonly found in association with protein – milk, yogurt and cheese being particularly rich. However, there are some other foods that contribute phosphate, e.g., oatcakes; also offal, shellfish, nuts, milk chocolate, eggs, scones, Horlicks). Other sources are convenience foods that have phosphates added by food manufacturers.

Patient information on all these topics is linked from the handbook webpages.

## Dietary prescriptions in renal failure

Status	Protein	Energy	Fluid	Sodium	Potassium	Phosphate
<b>CM</b>	0.8g – 1 g/kg Ideal Body Weight (IBW)	35kcal/kg IBW min (unless overweight )	Normal (some exceptions require restrictions)	'No added salt' 80-100mmols	Restricted only if blood levels high	Restriction may be required if levels high or dietary intake excessive
<b>Nephrotic Syndrome</b>	1g /kg IBW	35kcal/kg IBW	May require restriction	'No added salt' 80-100mmols	Unrestricted unless levels high	Unrestricted unless levels high
<b>HD</b>	1g – 1.2g/ kg/IBW	35kcal/kg IBW	500mls + UO	'No added salt' 80-100mmols	Most require some restriction <1mmol/kg	Dietary restriction and phosphate binders
<b>PD</b>	>1.2g/kg IBW - higher in peritonitis	25-30kcal/kg IBW (300-600kcal from PD fluid)	Based on ultra filtration - or 750mls + UO	'No added salt' 80-100mmols	Unrestricted for most patients	Dietary restriction and binders often required
<b>Transplant</b>	1g/kg IBW Higher 1-2 wks post-op	Depends on BMI and need for weight gain/loss	High fluid requirements normally	'No added salt' 80-100mmols	Unlikely to need restriction	High intake advised post-op
<b>Acutes</b>	0.17-2g Nitrogen per kg	BMR + stress and activity factors	Depends on UO and RRT	'No added salt' 80-100mmols	Based on blood levels but intake often low anyway	Be aware of hypophosphataemia – refeeding syndrome

\*CM = conservative management;

UO = urine output;

## Dietetic Referral Criteria

### Chronic renal failure

Patients with any of the following should be referred to the renal dietitian for individual dietary assessment and advice.

1. CKD stages 4 and 5 (GFR <30ml/min/1.73m<sup>2</sup>)
2. Hyperkalaemia, Serum K<sup>+</sup> ≥ 5.5mmol/l on an upward trend, not acidotic.
3. Starting an ACE Inhibitor with serum K<sup>+</sup> ≥ 5.0mmol/l
4. Malnutrition related to uraemia
  - BMI < 20kg/m<sup>2</sup>
  - Unintentional weight loss >5% in 3 months
5. Serum PO<sub>4</sub> ≥ 1.8mmol/l
6. Patients with moderate to severe nephrotic syndrome requiring no added salt diet ± nutritional support.
7. Renal stones – patients with calcium oxalate or uric acid stones who will benefit from dietary information, at the dietitian's discretion.

### General nephrology and transplant clinics

- Refer in writing, giving relevant information.
- Please ensure the patient is aware that they have been referred to the dietitian and is willing to attend.
- After receipt of referral, patients will be seen at their next nephrology clinic appointment if possible. If necessary, request earlier contact in your referral.
- Patients with hyperkalaemia will be sent basic written low potassium dietary information as first line advice before being seen.
- Dietary advice for non-renal conditions such as cholesterol lowering, weight reduction, IBS and eating disorders: will not be accepted, clinic staff can provide written information on general healthy eating such as “Your weight, Your Health”, but for more detailed information refer to a community dietitian.
- Basic information on a *no added salt* diet is available for all staff to issue as appropriate.

### Follow Up Policy

- Patients with stages 4 and 5 CKD will usually be kept under follow-up.
- Others and patients who miss appointments will usually be discharged and the referrer will be informed by letter.

### Haemodialysis

Before referring please check dietetic therapy entries in Proton, as often patients will have recently seen the dietitian.

- Newly started onto haemodialysis for dietary education
- K >6.5 mmol/l
- PO<sub>4</sub> >1.8 mmol/l on more than one occasion
- Consistent inter-dialytic weight gain >2.5 kg
- Unintentional weight loss >5% in 3 months
- BMI <20 kg/m<sup>2</sup>
- Poor appetite/GI symptoms for 2 weeks or more.

Patients will be seen on dialysis at a convenient time. All patients will be routinely reviewed 6 monthly as per QIS standards.

### **Peritoneal dialysis**

- First check Proton diet screen.
- Newly started onto peritoneal dialysis for dietary education.
- K >5.5 mmol/l
- PO<sub>4</sub> >1.8 mmol/l on more than one occasion
- Persistent fluid overload related to fluid intake
- Unintentional weight loss >5% in 3 months
- Poor appetite/GI symptoms for 2 or more weeks
- BMI <20 kg/m<sup>2</sup>

PD patients will be seen for initial education when they are training or for review at their next clinic appointment. Please specify if patients require earlier input. All patients should be reviewed 6 monthly as per QIS standards.

### **Management of Hyperkalaemia**

- Upon receipt of referral for hyperkalaemia a letter will be sent out to the patient with basic low potassium dietary information, requesting the patient to phone for further advice. This will be documented on Proton. We won't routinely telephone the patient.
- When the patient calls back, telephone advice will be given and documented on Proton.
- Patients will usually be seen at their next Nephrology appointment.
- If the patient does not phone for further advice they will have a copy of the basic low potassium dietary information and will be seen at their next Nephrology clinic appointment.

Extra resources on the web.

## FOREIGN TRAVEL FOR DIALYSIS PATIENTS

### HAEMODIALYSIS

HD requires considerable forward planning, especially if requested at units close to popular holiday destinations. Requests should be in writing, include relevant patient and dialysis provision details and, if possible, propose alternative dates. Only dialysis centres judged unlikely to infect patients with HepB/HIV/HepC are acceptable. Dialysis requests to foreign centres should, therefore, explicitly request confirmation that the unit conducts virus surveillance and segregates infected patients. When the risk of infection exists, or is uncertain, patients must be made aware of risk and upon return to Edinburgh unit be treated as HepB positive (segregated) and suspended on transplant list for six months.

### CAPD

Dialysis at destinations in the developed world can usually be organised, provided a holiday address is available for delivery and storage of PD fluid.

Patients should

- i. carry a supply of antibiotics and diluents
- ii. know how to assemble and administer in case of peritonitis
- iii. carry a letter for Customs - and -
- iv. know how to contact local Nephrology Service

### Infection Prophylaxis

Patients with renal failure make relatively poor antibody responses to immunisation but should still be offered immunisations as recommended for the geographical area of travel, with one or two particular exceptions:-

- a) immunosuppressed patients should not receive live virus vaccines - these include yellow fever, conventional oral (Sabin) polio vaccine, and oral typhoid vaccine. If polio cover is required obtain the killed (Salk) polio vaccine.
- b) **Hepatitis B** - All patients should have been immunised. Check current immune status and discuss with Department of Virology. If negative and travelling to a high risk area, immunoglobulin may be required.
- c) **Malaria**  
Prophylaxis is difficult because Proguanil is contra-indicated (potential marrow toxicity). No ideal regime is available. Up to date information should be sought from renal pharmacist or medicines information.

**Insurance:** Dialysis patients travelling abroad must buy adequate health insurance, particularly if travelling to the USA. The National Kidney Federation website [www.kidney.org.uk](http://www.kidney.org.uk) is one possible source of information about suitable insurance companies.

## GFR ESTIMATION

Glomerular filtration rate (GFR) is the usual method for measuring renal excretory function. Most commonly it is estimated (eGFR), or surrogates are used such as creatinine clearance (CrCl).

### Normal values for GFR

120±25 ml/min (95th centiles. Males approx 5mls higher, females approx 5mls lower). Therefore values >90 are normal for all. Converting for average surface area (per 1.73m<sup>2</sup>) removes the sex difference.

With age, GFR tends to fall (to approx 100ml/min/1.73m<sup>2</sup> at age 70), although serum creatinine does not rise much in healthy individuals (Fliser et al, *Kidney Int* 51:1196-1204, 1997). This fall is mostly due to subclinical pathology.

### Estimating GFR

A variety of methods have been used. All are based on serum creatinine estimations. Although the Cockcroft-Gault formula is widely used, it was developed to permit estimation of CrCl, not of GFR. The best validated method comes from the MDRD Study data (*Ann Int Med* 130:461-70, 877-84, 1999). The study derived a number of equations. The abbreviated, or four-variable equation includes age, sex, creatinine, and race (black or not black). Adding more variables (albumin, urea) adds little to accuracy.

It is important to be aware of the limitations of these equations:

- Accuracy - even for the MDRD equation, which is the best there is, the confidence limits are wide. 90% of values are within 30% of the true value; 98% within 50%.
- Extremes - none of the methods for estimating GFR without actual measurements of it are likely to be accurate in extreme examples of low muscle mass, or other unusual circumstances.
- Stability - for all methods, [creat] must not be changing quickly.
- Systematic errors - MDRD is better at low GFRs.
- Age – the Schartz equation (requires height) should be used in children.

Most UK labs now report eGFR when returning creatinine values. When available, these values should be more accurate than calculations performed by you, as they should incorporate lab-specific correction factors. If not, here are some resources:

- The UK Renal Association Website – <http://www.renal.org/eGFR>
- Online calculator from nephron.com offers a simple abbreviated MDRD, and has a useful menu of alternatives including quaint US units and also Cockcroft-Gault. Take care to set units for [creat] correctly.

### Measuring GFR

For direct measurement of GFR, isotope tests have largely taken the place of inulin clearance. Like inulin, these markers (eg 51Cr-EDTA, 99Tc-DTPA) are cleared almost entirely by glomerular filtration, and measures of their disappearance rate from the circulation, or appearance in the urine, can be used to estimate GFR.

In the future, other serum markers may become useful as more direct indicators of GFR. Cystatin C, a 13kDa cysteine protease that is produced at a constant rate by all nucleated cells, looks promising.

## Reciprocal of creatinine plots

Plots of the reciprocal of creatinine are useful as they can be used to give an approximate prediction of the date of ESRF for many patients, and to identify changes in the rate of progression. The rate of creatinine production in an individual changes slowly if at all. Download a blank reciprocal creatinine plot from <http://www.renal.org/eGFR> – but note that plots of GFR vs time should be linear and in the future will probably replace 1/creatinine.

## Creatinine clearance

Creatinine clearance overestimates GFR in a variety of circumstances, because of tubular secretion. This is particularly important at low levels of GFR. To calculate it, pay close attention to units and remember that there are 1440 minutes in 24h (details of calculations below). However 24 hour urine collections are error-prone and have been shown to be on average less accurate for estimating GFR than deductions from plasma creatinine.

**Calculations:** everyone knows it's UV/P but the units get a little confusing. This formula shows SI units.

$$CrCl(mls/min) = \frac{UV \times 1000 \times 1000}{P \times 1440}$$

UV	Amount of creatinine in 24h of urine is reported by the lab in mmol/24h. x1000 to convert to micromols
P	Serum creatinine is reported in micromols/l. x1000 for mls
1440	Number of minutes in 24h

The Cockcroft-Gault equation aims to predict creatinine clearance from knowledge of serum creatinine, age and weight. See Calculator 2 (mentioned above) or do it the hard way:

$$\frac{(140 - \text{age}) \times \text{weight(kg)} \times 1.23 \times (0.85 \text{ if female})}{\text{Creat(micromol/l)}}$$

Weight should be lean body mass, but attempting to calculate that makes the equation unreasonably complicated. Estimate lean body mass for extremes of size, or use the MDRD equation instead, or best of all, measure it.

**Note that these estimates/measures of CrCl are not corrected for body size.**

Extra resources on the web.

## GOUT AND HYPERURICAEMIA

Uric acid levels are raised in chronic renal failure, and in view of that, gout sometimes seems less common than expected.

- Asymptomatic hyperuricaemia should usually not be treated.
- Patients with very early onset gout should be screened for predisposing genetic abnormalities.
- Lead poisoning should be remembered as a cause, but is rarely the explanation for gout with renal impairment and hypertension.
- Tophaceous gout may be associated with interstitial granulomatous nephritis with crystal formation – but very rarely except in inherited severe gout.

### Allopurinol

Is more likely to cause toxicity in renal impairment and initial dose should be 100mg. It may rarely cause interstitial nephritis. Its introduction may precipitate acute gout and this needs to be protected against with nonsteroidals or colchicine.

Allopurinol inhibits metabolism of azathioprine. Although some advice suggests reducing azathioprine dose by two thirds, consideration should be given to alternatives, or cessation of azathioprine, if allopurinol is clinically necessary.

### Nonsteroidals

Are not necessarily completely contraindicated in moderate renal failure, if used with monitoring and for a limited period.

### Colchicine

Is the first choice for treatment of acute gout in patients with significant renal impairment. It has a narrow therapeutic ratio, toxicity manifesting as diarrhoea, nausea and vomiting. The BNF recommendations (2mg then 500mcg every 2-3h) will often cause severe side-effects; safer to start with 500mcg tds and increase as necessary.

Lower doses (e.g. 500mcg 1-3 times daily) are useful prophylactically.

### Uricosurics

Are of little value in the presence of significant renal impairment. In addition to the conventional uricosurics probenecid and nenzbromarone, losartan and fenofibrate have some uricosuric activity.

## Acute severe hyperuricaemia

Occurs particularly in tumour lysis syndromes (though prophylactic allopurinol usually prevents it), or in high turnover haematological malignancies such as AML. Causes acute renal failure through crystalluria. Treatment has three elements:

- Remove urate by high intensity haemodialysis – will require long and frequently repeated treatments.
- Prevent uric acid formation with allopurinol – high doses indicated.
- Recombinant uricase (urate oxidase) appears effective, beginning to lower uric acid levels in hours when administered prophylactically.

## HAEMODIALYSIS

### Quantitation and adequacy

Measurements of small molecule clearance have become an accepted way of assessing the adequacy of haemodialysis, because a correlation with mortality has been shown in large studies. It is a Renal Association standard that adequacy is checked monthly on all chronic haemodialysis patients

**Kt/V for urea\*** is the most widely accepted of these measurements, although a simple surrogate for this, urea reduction ratio (**URR**), is more readily measured and is also widely used. There is a calculator for Kt/V in Proton.

- **K** is a dialyser-specific figure for rate of urea clearance on an individual dialyser, at a given blood flow and dialysate flow. **t** is duration of dialysis. **V** is the volume of distribution of urea in a patient.

### Urea reduction ratio method

In the RIE we check Urea Reduction Ratio (URR), calculated as:

$$\text{URR} = 100 \times [1 - \text{Post(Urea)/Pre(Urea)}]$$

The Edinburgh unit participates in a multicentre audit with comparison of URR results between all units in Scotland. This is the protocol approved by the Scottish Renal Association:

- The 'pre' sample is taken immediately after cannulation of the fistula with a dry needle and before the dialysis starts. For central venous cannulae, a sample can be drawn from the line after the Heparin lock has been removed. Take care not to contaminate the samples with saline.
- The post sample is taken by the stop flow method ie. the dialysate flow is stopped for 5 minutes, leaving the blood pump running, before sampling from the port in the arterial blood line. This is to allow time for the blood in the access to equilibrate with the central circulation, but not enough time for the equilibration from the tissue pool

Only these bloods should be marked PRE (001) and POST (002) and are done by staff in the dialysis Units on a monthly basis.

Bloods taken without this stop flow will overestimate urea clearance on dialysis. Hence, while it may be appropriate to take bloods pre-dialysis on eg. in-patients to save patients venepuncture, and it is often necessary to know post dialysis potassium in out-patients who have not for whatever reason, completed their dialysis prescription, mark these bloods with the time they are taken, not pre/post or the 001/002 codes. This is very important as failure to do so causes problems with download of data to the Registry

### Problems with Kt/V & URR

- Small molecule clearance is not the only or even always the most important factor in determining dialysis adequacy.
- Fluid balance is important for mortality and is not measured by Kt/V or URR. It is more affected by dialysis frequency and duration.
- Calculations that use measurements of blood urea (instead of dialysate urea) are prone to sampling errors, particularly urea rebound.
- V (volume of distribution of urea) is low in wasted, malnourished patients – high Kt/V values may be caused by this, rather than by excellent dialysis.

## Kt/V versus URR

Calculations based on URR alone underestimate Kt/V because no account is taken of the consequences of ultrafiltration. This removes urea, but is 'invisible' by URR monitoring alone.

**Table:** correlation of URR to Kt/V corrected for ultrafiltration (NKF K/DOQI Guideline 2000). Calculated from single-pool, variable volume model with a body weight of 67.3kg, V of 35L, and NPCR of 1.0. Wt/BWt is the ultrafiltration volume/post dialysis weight x 100

$\Delta Wt/BWt$ %	URR Values at Kt/V							
	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.5
0	53	57	61	65	68	71	74	76
1	52	57	61	64	68	71	73	76
2	50	55	59	63	66	69	72	75
3	49	54	58	62	65	68	71	74
4	48	53	57	61	64	67	70	73
5	47	51	56	60	63	67	70	72
6	46	50	55	59	62	66	69	71
7	44	49	53	58	61	65	68	71
8	43	48	52	57	60	64	67	70
9	42	47	51	56	59	63	66	69
10	41	46	50	54	58	62	65	68

### Prescribed dialysis:

Dialysis should be prescribed on an individual basis. The variables influencing the amount of dialysis required are

- **Weight:** Higher weight = more dialysis required. Note that in serial audits, we have previously underprescribed dialysis for large patients
- **Sex:** Men require more than women of the same weight due to greater proportional body water

The variables in the prescription that will improve dialysis adequacy are:

- **Time:** this is the most important variable to achieve adequacy and the time likely to be required needs to be explained to patients BEFORE they choose haemodialysis
- **Blood flow rate (Qb):** aim for high e.g., 400ml/min – monitor venous pressures – allow up to 200ml/min
- **Dialyser surface area :** FX8 is smaller than FX10
- **Dialysate flow rate (Qd) :** either 500 or 800ml/min possible (NB the difference this makes is not quantified and likely to be small but if Qb is 400ml/min, use Qd=800ml/min

To prescribe adequate dialysis, please consult the following chart for a guide. Hours required to achieve  $Kt/V > 1.3$

- Always round up to the higher time and assume that Qb initially at least will be less than you prescribe
- Work on the principle that the average dialysis duration is ~4.5 hrs – start with this and then tailor to the patient on the basis of access performance & measured dialysis adequacy
- Do not prescribe less than 3 hours three times a week except in exceptional circumstances (discuss with named consultant)
- Maximise Qb – particularly important in larger patients
- Do not use FX8 in large people or FX10 in small people (though FX60 may be appropriate - discuss with named consultant)

- High flux dialysis (with FX60 dialyser) or haemodiafiltration (with FX80 and adaptation to dialysis machine) may be appropriate for anuric patients on (or expected to be on) dialysis for a prolonged period or with signs/symptoms of dialysis amyloid to aid middle molecule clearance
- HDF may also be appropriate for larger patients with very long hours (discuss with named consultant)

MEN	FX8				FX10 FX60			
	Qb 250	Qb 300	Qb 350	Qb 400	Qb 250	Qb 300	Qb 350	Qb 400
40kg	3	3	3	3	3	3	3	3
50kg	3	3	3	3	3	3	3	3
60kg	3.75	3.25	3	3	3.5	3.25	3	3
70kg	4.25	3.75	3.5	3.25	4.25	3.75	3.25	3
80kg	4.75	4.25	4	3.5	4.75	4.25	3.75	3.5
90kg	5.5	4.75	4.5	4	5.25	4.75	4.25	4
100kg	6	5.5	5	4.5	6	5.25	4.75	4.25
110kg	6.5	6	5.25	5	6.5	5.75	5.25	4.75
120kg	7.25	6.5	5.75	5.25	7	6.25	5.75	5.25
<b>WOMEN</b>								
40kg	3	3	3	3	3	3	3	3
50kg	3	3	3	3	3	3	3	3
60kg	3.5	3	3	3	3.5	3	3	3
70kg	4	3.5	3.25	3	3.75	3.5	3	3
80kg	4.5	4	3.5	3.25	4.35	4	3.5	3.25
90kg	5	4.5	4	3.75	5	4.25	4	3.5
100kg	5.5	5	4.5	4	5.5	4.75	4.25	4
110kg	6	5.5	5	4.5	6	5.35	4.75	4.25
120kg	6.5	6	5.25	5	6.5	5.75	5.25	4.75

FX8 :  $K = 0.51Qb + 93$

FX10/60 :  $K = 0.55Qb + 87.3$

FX80 :  $K = 0.8Qb + 38$

$t =$  dialysis time (min)

$V = 0.6 \times \text{dry wt (men), (x 0.55 women)}$

#### When prescribed vs delivered dialysis differ:

If the prescribed vs delivered dialysis is very different, consider:

- Access recirculation, interrupted or shortened dialysis, slowed pump speeds, clotting dialysers, delayed re-equilibration of urea (eg in shock or cardiac failure), and errors in assumptions about  $V$ , will often tend to reduce actual dialysis dose. This is particularly likely in acute renal failure.

## Dry weight

The post-dialysis weight that allows pre-dialysis blood pressure to remain normal despite interdialytic weight gain and without drugs. At dry weight the patient has no clinical signs or symptoms of hyper- or hypovolaemia. Note that the presence of any oedema usually indicates several kg overweight.

At centres providing long hours of dialysis, it is often reported that blood pressure can be controlled by attention to sodium and fluid balance alone in 90% or more of haemodialysis patients. This is achieved by:

- very frequent clinical review of dry weight
- slow reduction of post-dialysis weight over weeks with concurrent
- reduction of antihypertensive drugs
- lengthen dialysis if hypotension occurs
- lower dialysis sodium to reduce thirst
- remember that dry weight often will rise after an initial fall in new patients, as nutrition improves

Read: *Nephrol.Dial.Transplant* 12:1104, 1997, and 14:121-4, 1999

## Routine monitoring of haemodialysis patients

Pre- and post-dialysis U & E, Creat	monthly
LFTs, Ca, Alb, PD4	monthly
FBC	monthly (fortnightly in RIE or if recent change in EPO dose)
Iron studies	2 monthly
PTH	2 monthly
Lipids	annually, more often if high
HbA1c in diabetics	2 monthly
HepBsAg, HepC Ab	3 monthly
HIV Ab	12 monthly
Aluminium	3 monthly
Cytotoxic antibodies (if on transplant list)	monthly

Patients on HDF have in addition:

Trace metal & micronutrient screen, $\beta_2$ microglobulin, B12 & folate	3 monthly
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### Adequacy targets:

For thrice weekly haemodialysis, the MINIMUM targets for patients with no residual renal function (SRA, RA, DOQI) are:

### **Kt/V >1.2 or URR >65% for all patients on chronic haemodialysis**

To achieve this, **the population mean needs to be Kt/V=1.3 or URR=70%**

Higher values may be beneficial – and the unit average needs to be appreciably higher than these minima. Up to a Kt/V of 1.5-1.7 may be 'good', as long as not due to low V (weight).

Be aware that proton corrects URR to 2 significant places. Thus 65% can be 64.5-

65.4% - While the target is arbitrary, it is based on survival data and 64.5 is not adequate dialysis

### Other targets for monitoring (Renal Association – Guidelines March 2006)

K	3.5-6.5 mmol/l
PO <sub>4</sub>	1.1-1.8 mmol/l
Ca (corrected)	<2.5 mmol/l
Ca x PO <sub>4</sub> product	<4.8 mmol <sup>2</sup> /l <sup>2</sup>
PTH	130-260 (x2–x4 upper limit normal)
Hb	>100g/dl (aim for 110g/dl)
Ferritin	100-800
Transferrin saturations	>20%
Aluminium	<2.2µmol/l
Bicarbonate	20-26mmol/l
Cholesterol	<5mmol/l
HbA1c	<7%
Pre-dialysis BP	<140/90 mmHg
Post-dialysis BP	<130/80 mmHg

### Medications

Patients dialysing at the RIE are reviewed in a multidisciplinary team meeting approximately every 8 weeks.

- Most patients receive their drugs from their GP
- Erythropoietin and i.v. alfacalcidol or iron preparations are supplied by the hospital

## Troubleshooting on haemodialysis

### Hypotension

Usually occurs for one of three broad reasons:

- Patient is below dry weight (q.v.)
- Fluid removal is faster than redistribution can occur (eg, too large weight gains, unstable circulation)
- Some effect of dialysate/ membrane/ extracorporeal circuit on cardiac output and/or peripheral resistance

**Acute hypotension** is usually managed by saline infusion, reducing weight loss; if out of character consider cardiac problems (rhythm, ischaemia). Management for **recurrent hypotension** is something like:

- Advise patient about fluid, salt, etc
- Review dry weight (see 'Dry Weight')
- Consider longer, slower dialysis (unpopular)
- Consider serial ultrafiltration followed by isovolaemic dialysis (lengthens dialysis again; can be used for a time to get nearer to dry weight)
- Review haemoglobin (effect of anaemia possibly via cardiac oxygenation)
- Consider providing oxygen during dialysis
- Tricks with dialysate: (1) cooling (causes increased peripheral resistance); (2) sodium profiling, or ramping, in which the dialysate sodium is altered during dialysis. A higher dialysate Na reduces hypotension (probably by maintaining ECF osmolality) - but reduces Na removal. Start high, lower later helps.

Ultrafiltration rate can also be profiled on some machines.

- Avoid eating and drinking before/during dialysis (reduces peripheral resistance by causing splanchnic vasodilation)
- Omit hypotensive agents on the morning (or evening) before dialysis
- Oral midodrine, an  $\alpha$ 1 adrenergic agonist (currently available in the UK on a named-patient basis)
- Consider haemofiltration or haemodiafiltration (different membranes, but in the case of haemofiltration, also usually a slower treatment - and possibly with more cooling of blood)

### **Cramps**

Muscle cramps are very common during dialysis and can be of sufficient severity that they result in termination of the procedure. Their cause is unclear but the majority occur towards the end of the procedure after a significant volume of fluid has been removed. Their etiology is postulated to involve volume depletion and tissue hypoxia. They are associated with large requirements for fluid removal.

- Acute management aims at restoring intravascular volume by administering hypertonic fluid, most commonly 50% dextrose (50mls), in order to raise plasma osmolality.
- Quinine 2-300mg before dialysis or at bedtime can be tried but is unproven
- Oral agents such as clonazepam, vitamin E, carnitine, or anti-convulsants are sometimes used as prophylaxis but their benefits are even less certain.
- Limitation of inter-dialytic weight gains, ensuring that post-dialysis dry weight is correct and the use of an appropriate dialysate sodium are the best means to prevent this problem. Remember that a higher dialysate sodium will reduce intra-dialytic symptoms at the expense of thirst and weight gains; the converse holds true for a lower dialysate sodium. Sodium profiling may again be of benefit.
- Patients who experience cramps at night may benefit from muscle-stretching for a minute or two. Heat and massage for the cramping muscle can help.

### **Pyrexia**

Pyrexia in haemodialysis patients is usually related to the use of semi-permanent tunnelled access and most commonly due to Gram positive sepsis. It is common for dialysis to precipitate pyrexia in these circumstances. The exit site and tunnel should be checked for discharge, tenderness or erythema, but is commonly not abnormal.

- Take blood cultures from the line and from a peripheral vein, along with routine bloods and an examination for other causes.
- Treat with intravenous antibiotics according to the Antimicrobial protocol, with removal of catheter if recurrent or if it fails to respond promptly.

All patients with ESRF are relatively immunosuppressed. 'Typical' bacterial infections of all types seem to be more common, and infection is the second most common cause of death in dialysis patients. Tuberculosis is possibly as common in haemodialysis patients as it is in patients immunosuppressed after receiving a renal transplant.

### **Non-functioning access**

This is described in the section on Vascular Access

### **Dialysis-related critical incidents**

The most serious acute events include air embolism, line disconnection leading to haemorrhage, acute haemolysis or toxicity related to line kinking or dialysis

contamination, and acute allergic reactions to dialysers or sterilants (e.g., the 'first-use' syndrome attributed to antibody formation to ethylene oxide). If any such crisis occurs and the explanation is not entirely clear, in addition to all the necessary supportive measures:

- Stop dialysis
- Take samples from venous and arterial lines - look for alterations in haematology and biochemistry
- Disconnect the patient. Record their weight and routine observations.
- Keep a sample of dialysate
- Keep the used dialyser
- Take the machine out of use. Inform the dialysis technicians that it was in use when an incident involving a patient occurred (e.g., by a prominent notice on the machine), so that an investigation can be made and evidence preserved.
- Record all the details, including the precise circumstances (patient's position, first symptoms, full history)
- Fill in the appropriate incident report form

## **HYPERCALCAEMIA**

- In chronic renal failure of long standing, hypercalcaemia is usually caused by the use of vitamin D derivatives and phosphate binders, and sometimes by tertiary hyperparathyroidism. [See section on Osteodystrophy]. Usually stopping vitamin D and calcium containing phosphate binders is enough, but be cautious if patient is on a significant dose of vitamin D or has had a parathyroidectomy – calcium may plummet.
- In acute renal failure calcium is usually low-normal. A high-normal or high calcium should lead to suspicion that the renal problem is caused by hypercalcaemia itself, or by the same disease as is responsible for hypercalcaemia – e.g., myeloma.

### **Treatment of hypercalcaemia**

Fluid repletion with saline improves renal impairment caused by hypercalcaemia and has a small effect on calcium level.

Diuretics should be avoided unless adequate volume expansion with normal saline has been achieved. Thereafter loop diuretics can be used with caution, but thiazides tend to increase serum calcium.

Corticosteroids are effective in sarcoidosis, some haematological malignancies and allegedly also in vitamin D poisoning.

Bisphosphonates are effective in all circumstances. Disodium pamidronate can be given as a single dose of 30-90mg over 2-4 hours (maximum infusion rate 20mg/h in renal impairment; maximum concentration 60mg in 250ml 0.9% NaCl). Calcium falls over days, reaching a nadir at 3-5 days, and usually remaining suppressed for several weeks, when the infusion can be repeated. Note that high doses of pamidronate may be nephrotoxic – associated with proteinuria and FSGS.

For dialysis patients, adjusting calcium content of dialysate may be helpful.

## HYPERKALAEMIA

Usual intake of  $[K^+]$  is approximately 1mmol/kg/day, but homeostasis can be maintained at intakes of 20-500 mmol in those with normal renal function.

Remember acute causes of altered homeostasis and elevation:

- Hyperglycaemia (by osmotic effect and by insulin deficiency)
- Acidaemia
- Aldosterone deficiency (including spironolactone, ACE inhibitors, heparin)
- K supplements + K-sparing diuretics

### Treatment of acute hyperkalaemia

#### Intravenous calcium (if there are ECG changes)

10% gluconate or chloride, 10mls over 5 minutes (maximum 2mls/min)

- Give if ECG changes – peaked T-waves, prolonged PR
- Check in 15 minutes and if still abnormal, repeat once or twice
- Does not change  $[K^+]$ ; reduces excitability of membranes

#### Intravenous dextrose

25g (e.g., 50ml 50%) + 5u Actrapid over 20 minutes (maximum ratio of 5g :1 unit)

- Acts in 30 minutes, peak effect 90 minutes, lasts up to 6 hours
- Lowers  $[K^+]$  by 0.7-1.6mmol/l
- Can be followed by slow infusion of 10-50% dextrose (give insulin only if glucose high)

#### Salbutamol

5mg nebulised (or an IV preparation can be given IV)

- Acts in 60 minutes, peaks 90 minutes, lasts up to 6 hours
- Similar to dextrose in efficacy

#### Sodium bicarbonate

Traditionally 50ml of 8.4%; but usually as 1.26%

- Can reduce  $[K^+]$  by 0.2-0.3mmol/l but involves sodium load
- Not routine but may be useful in emergency

#### Dialysis

- Note that above treatments do not remove, they only redistribute  $[K^+]$
- A standard haemodialysis removes 40-60mmol  $[K^+]$
- Removal of  $[K^+]$  by haemofiltration or peritoneal dialysis is much slower

#### Calcium resonium

- Not useful in acute setting but may be short/medium term option if dialysis not desirable or possible. Causes constipation.

#### Diet

- May explain acute hyperkalaemia; important for prevention, see Diet

**See also the section on perioperative management of  $[K^+]$  – under SURGERY**

## HYPERLIPIDAEMIA

All stages of renal disease are associated with increased cardiovascular risk. Isolated microalbuminuria increases the risk of a heart attack or stroke by over 60%, while dialysis increases the risk of death from cardiac disease by up to 100 fold.

Hyperlipidaemia is common in patients with renal disease. Sub-group analysis of the big statin trials has demonstrated a risk reduction for cardiovascular events in CKD stage 3 patients treated with statins, comparable to non CKD patients. However, intervention studies in dialysis patients have failed to show improved outcomes. There is also limited evidence that lipid lowering may slow the rate of progression of renal disease.

### Guidelines for treatment

In the absence of high quality evidence we recommend the following:

- All patients with CKD should have lipids checked annually
- If random total cholesterol >5.0mmol/L then all patients should receive lifestyle advice (addressing all risk factors including diet)

The Joint British Societies Guidelines on prevention of coronary heart disease recommend initiating a statin in patients with established vascular disease and high risk individuals without clinically overt vascular disease at a serum total cholesterol  $\geq 5.0$ mmol/l (LDL cholesterol less than 3.0 mmol/l), and aspirin treatment if BP >150/90.

Pending the outcome of the SHARP trial, statin therapy should be considered in all patients with Stage 1-3 chronic kidney disease with a predicted 10 year cardiovascular risk  $\geq 20\%$ , irrespective of baseline lipid parameters.

Nephrotic patients have hypercholesterolaemia. If they do not respond promptly to treatment of the nephrotic syndrome, they should be started on lipid lowering therapy.

A **statin** should normally be the first line. In combined hyperlipidaemia atorvastatin may have improved efficacy. Watch for myositis and rhabdomyolysis in patients with CRF.

## INFECTION CONTROL

Infection control precautions should be the same for all patients.

White coats are not worn in the unit.

Hand hygiene is the most important infection control measure. Wash your hands thoroughly before and after each patient contact, even if gloves have been worn. Alcohol gel is an acceptable alternative if the hands are not visibly soiled.

Disposable plastic aprons and gloves are widely available and should be used if there is risk of cross-infection.

- Apron to examine patients in all HDU areas
- Gloves as well as apron if performing procedures or handling dressings, fluids etc or needles.
- Be cautious – play safe – when examining patients known to carry MRSA or other resistant organisms. Use an identified stethoscope for MRSA patients.

When worn, all gloves and aprons must be changed between each patient and disposed of in a clinical waste bag.

### Methicillin resistant *Staphylococcus aureus* (MRSA)

All staff are reminded that it is hospital policy to screen for MRSA all known positive patients and patients admitted from:

- other hospitals
- nursing homes
- residential care

All patients known to be either colonised or infected with MRSA are cohort-nursed in single rooms or 4 bed bays in Ward 206. Haemodialysis patients with MRSA must dialyse in Room 1 in ODA or in the single rooms at BGH/SJH Satellite Units. Currently, there are no facilities to dialyse MRSA colonized patients at the WGH Satellite and patients must move to Room 1 ODA.

#### Colonised Patients

It is important to remember that MRSA may colonise one or many sites without necessarily infecting the patient. Patients who are simply colonised may be suitable for the organism eradication protocol. This should be done only after consultation with a microbiologist or Senior Infection Control Nurse.

The protocol is carried out for five days:

- Chlorhexidine gluconate 4% to wash daily (only for patients with intact healthy skin)
- Chlorhexidine gluconate 4% as shampoo twice in five days
- Mupirocin (Bactroban) nasal ointment one application to each nostril three times daily
- Chlorhexidine gluconate mouthwash 0.2% or oral spray 0.2% gargled or sprayed to the throat four times a day

Once the protocol is completed, the patient should be treatment-free (including anti-staphylococcal antibiotics) for 48 hours before screening swabs are taken. Three consecutive negative sets of swabs without intervening antibiotics are required for an eradication to be declared successful.

It is Unit policy that all MRSA patients are screened monthly, unless they are receiving antibiotics or on an eradication regime. Normally, three eradication treatments will be tried. Patients still positive after three eradication treatments are screened 3-monthly.

## **Vancomycin resistant enterococci (VRE)**

The Renal Unit had a large outbreak in 1995, and further cases since 2000. While VRE is generally considered to be an organism of low pathogenicity, we have had a number of cases of VRE septicaemia. It is sensible therefore to make sparing use of vancomycin, cephalosporins and quinolones.

We do not screen for VRE, but should an in-patient be found to have VRE-positive diarrhoea, they must be isolated in a cubicle or cohort nursed in 4 bed bays in Ward 206. It is preferable to segregate MRSA and VRE positive patients in Ward 206 but it is acknowledged this is not always possible. A patient isolated in Ward 206 must remain so throughout that admission and move to bay 1 in the outpatient dialysis area if they are on haemodialysis. At 2 weeks after discharge, if the patient is continent and has good personal hygiene, they should be swabbed for MRSA, and if negative, they may move out into the main ODA bays or WGH/SJH after discussion at the dialysis moves meeting.

BGH patients must be discussed with Dr W Metcalfe. Patients isolated in Room 1 for VRE must be screened monthly for MRSA.

## **Clostridium difficile**

This organism is frequently isolated from patients in the unit. Patients are not routinely isolated if MRSA negative and should they should not be cohort nursed with MRSA and high-risk VRE patients. For patients with diarrhoea, a side room is preferable.

The drug of choice for symptomatic patients is oral metronidazole 400mgs three times per day for at least seven days. In severely ill patients the use of oral vancomycin 125mgs four times a day may be considered after consultation with a microbiologist.

## Blood-borne viruses

Patients infected with blood-borne viruses represent an infection risk within the haemodialysis unit, and may require special consideration. For screening procedures, see Preparing patients for RRT, and Haemodialysis and Peritoneal Dialysis sections.

Because of differences in infectivity, not all viruses present the same risk. Furthermore, negative serology does not exclude virus positivity and high risk patients also require special consideration. The current protocol is as follows:

<b>Hepatitis B</b>	<b>acute dialysis</b> in side room, Hep B machine only <b>chronic dialysis</b> in OPDA side room, high risk machine/Hep B machine only.
<b>High risk individual</b> (eg. IV drug use, return from some foreign countries)	as per hepatitis B/HIV until clearly virus-negative. All potentially positive bloods must be sent as "High Risk"
<b>Hepatitis C</b>	chronic dialysis in isolation area if available, dedicated machine during chronic treatment. Machines can then be safely decontaminated.
<b>CAPD</b>	no restrictions; HBV/HIV patients should be positively encouraged to do PD
<b>CVVH</b>	No requirement to isolate Aquarius machines

## Antibiotic protocols

See Antimicrobial Policy section.

## NEPHROTIC SYNDROME

Proteinuria >3g/day (PCR > 300mg/mmol) is often associated with:

- salt and water retention apparent as oedema
- hypoalbuminaemia
- hypercholesterolaemia
- heightened susceptibility to infection
- increased risk of venous thrombosis

Management directed at the cause of proteinuria requires diagnosis (usually by renal biopsy). Specific treatment, usually immunosuppression, is available for some diseases.

### Complications

**Oedema** is controlled by salt restriction and diuretics.

**Blood pressure** should be reduced to 125/75 or less, using ACE inhibitors and diuretics in first instance.

**Hypercholesterolaemia** usually requires HMG CoA reductase inhibitors if syndrome is lasting.

**Anticoagulation:** as a minimum, immobilised patients should receive heparin prophylaxis .

**Infection:** patients with chronic severe nephrotic syndrome should receive Pneumococcal and meningococcal vaccination.

Penicillin prophylaxis has not been shown to be beneficial.

### Diuretic Therapy

- nephrotic patients are often relatively resistant to diuretics, but respond to loop diuretics - if necessary in high doses. Diuresis is usually enhanced by adding a thiazide such as bendroflumethazide or metolazone, and/or Spironolactone or other distally-acting diuretic.
- monitor therapy by weight, lying and standing blood pressure and general examination (JVP, chest, oedema). A degree of intravascular contraction is inevitable and necessary. Pronounced contraction is signalled by postural drop in blood pressure >20% (or >20/10) and is potentially dangerous. This is particularly likely to occur if rate of weight loss is greater than 0.5-1.0kg/day. Greater rates of loss usually require daily observations.
- a degree of residual oedema and mild postural hypotension is often the best that can be achieved.

Patient information available on the web.

## Treatment protocols

### Sample steroid protocols for minimal change disease/FSGS

Regimen for a first episode of MCD-NS in children:

Prednisolone 60mg/m <sup>2</sup>	daily for 4 weeks
Prednisolone 60mg/m <sup>2</sup>	alternate days for (4-)8 weeks
Then reduce dose by one quarter each fortnight (total 4.5 months)	

Regimen for an adult (MCD or FSGS; protocol recognises slower responses):

Prednisolone 1mg/kg/d	daily for 8-16 weeks, or 2 weeks after complete remission (whichever shorter)
Prednisolone 1mg/kg/d	alternate days for 2-4 weeks
Tail dose	over 3-4 months (first episode)

REMEMBER TO PRESCRIBE AN H2 BLOCKER (adults and children)

Consider bone protection (see section on osteoporosis prevention on steroids).

**Subsequent relapses:** tail more slowly if relapse has been quick; consider leaving on low dose therapy if frequently relapsing.

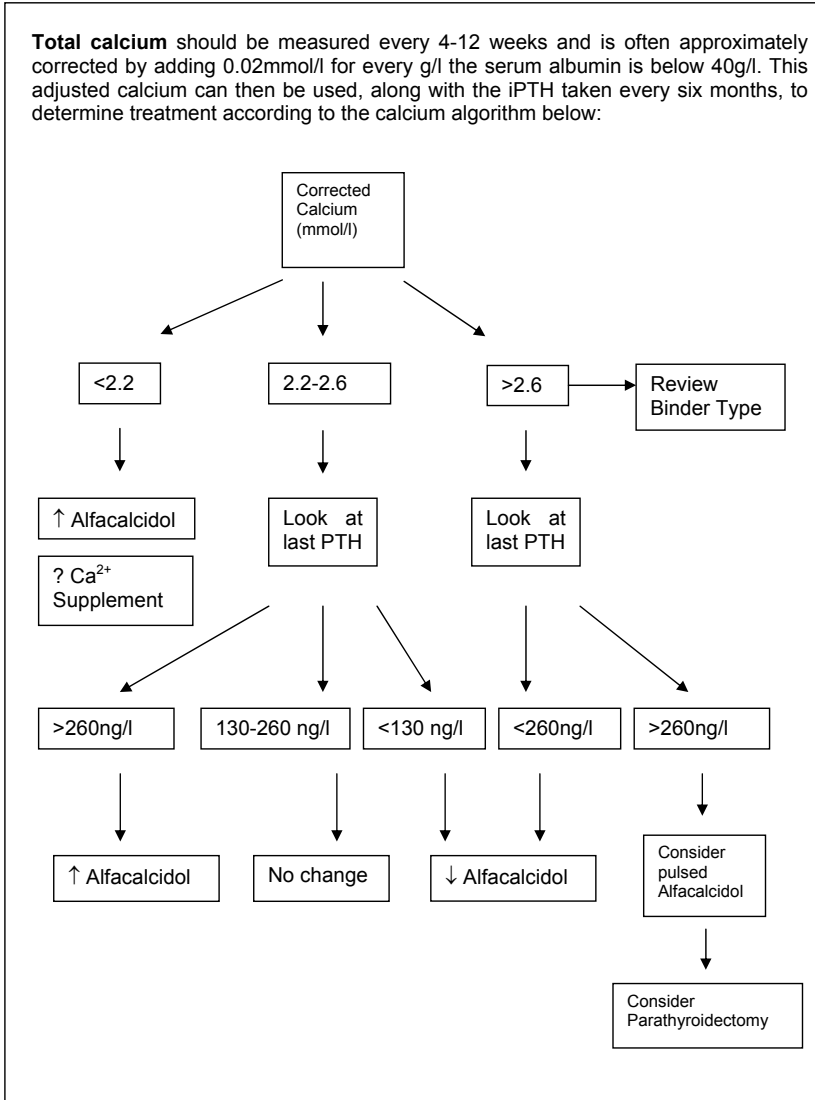
**Frequently relapsing / steroid-resistant / steroid-dependent** patients require discussion.

If response is incomplete, remember coagulation/ lipid disturbances as above, and vaccination.

## OSTEODYSTROPHY – PREVENTION AND MANAGEMENT

Aims of management are maintenance of healthy skeletal architecture, through achieving normocalcaemia and limited hyperphosphataemia, and by controlling PTH. Monitoring frequencies etc are biased towards dialysis patients. Less often in pre-dialysis patients and transplant recipients.

**Total calcium** should be measured every 4-12 weeks and is often approximately corrected by adding 0.02mmol/l for every g/l the serum albumin is below 40g/l. This adjusted calcium can then be used, along with the iPTH taken every six months, to determine treatment according to the calcium algorithm below:



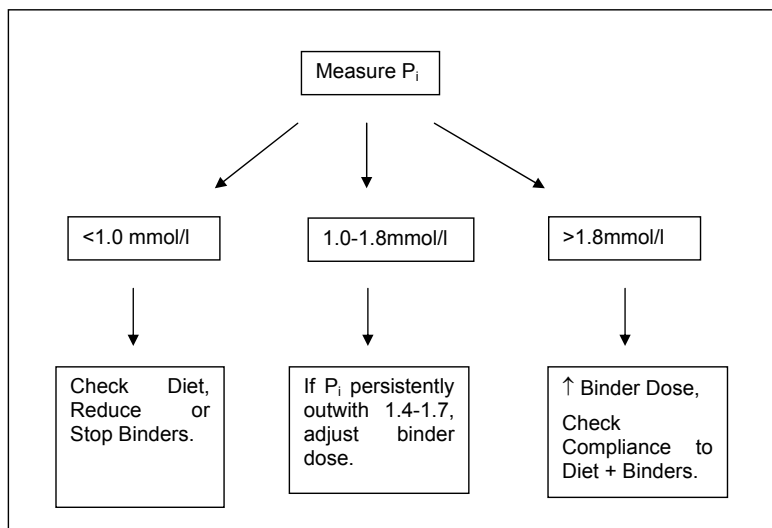
**iPTH** should be measured initially when GFR drops below 40-50mls/min. At this stage diet should be reviewed for both phosphate and protein content. High PTH levels should lead to introduction of alfacalcidol or calcitriol therapy, Calcium levels permitting. The standard starting dose is 0.25micrograms (250 nanograms) daily. High iPTH results (eg >500ng/l) may justify larger doses of calcitriol.

Note that the target range is higher than (2-4x) the normal range, in order to prevent adynamic bone disease resulting from overtreatment.

**Alfacalcidol and Calcitriol** are equally effective and equipotent. Both should be used at doses of 0.25, 0.5 or 1.0 micrograms for daily oral therapy. If the patient goes outwith the protocol limits, they should be taken out of the protocol and treated separately.

**Aluminium** should be measured every three months in patients on Al(OH)<sub>3</sub>. A result of greater than 2.2 micromol/l indicates a high risk of aluminium poisoning and requires cessation of Al(OH)<sub>3</sub>. Levels over 1 should lead to review of therapy.

**Serum phosphate** (Pi, PO<sub>4</sub>) should be measured every 4-12 weeks to adjust therapy with phosphate binders. This can be done using the phosphate algorithm below:



## PHOSPHATE BINDERS

For inpatients, prescribe at 8, 12 and 6 and write 'with food'.

Very approximately, one tablet of each is equivalent (larger dose size for sevelamer):

**Calcium acetate** (CaAc) is the first-line binder for all patients unless they are hypercalcaemic. It is effective but delivers a significant calcium load. Hypercalcaemia may occur, and in the long term the calcium load may accelerate vascular and extravascular calcification. Maximum dose is 6 tablets daily but this may be too much for long term therapy.

**Calcium carbonate** (CaCO<sub>3</sub>) is a probably less effective alternative and contains more calcium, but may be useful if patients dislike the calcium acetate formulation.

**Aluminium hydroxide** (e.g. Alucaps) is an effective binder that is relatively palatable. However it is not suitable for long-term use because of concerns about aluminium accumulation and toxicity in renal failure. Monitor Al levels during long term therapy and be cautious about duration.

**Sevelamer** (Renagel) is an expensive binder that does not cause calcium loading or other known serious long-term toxicity. Used alone it is probably less effective at lowering phosphate and its track record is still relatively short. It is indicated when:

- High serum phosphate levels in conjunction with serum corrected calcium above 2.6mmol/l
- Calcium-containing binders unable to achieve adequate control without high calcium dose ( $\neq$  >6 tablets/day) or hypercalcaemia
- Aluminium hydroxide therapy has been required for more than 3 months

**Other agents** are in development but have not yet found a fixed place in our local practice. See also 'intractable problems' below.

**Phosphate binder type/dose** is decided using measurements of corrected calcium and phosphate (and see above):

**Ca >2.6:** follow the algorithm above for management of alfacalcidol/calcitriol therapy, and consider changing the alfacalcidol or calcitriol dose. Otherwise/also:

- If on CaAcetate, consider change to Al(OH)<sub>3</sub>
- If on non-calcium binders, discuss - review PTH level, other cause
- If intractable and on dialysis, consider lowering dialysate calcium

### Ca 2.16 - 2.6

- If on Al(OH)<sub>3</sub> change (back) to Ca Acetate (if other factors corrected).

**Ca <2.16:** follow algorithm above for management of alfacalcidol/ calcitriol therapy. Otherwise/also:

- If already on Ca Acetate and PO<sub>4</sub> over 1.5, increase CaAc dose
- If Pi below 1.5, give CaAc between meals as a supplement.

### **Intractable problems with calcium and phosphate**

- **Compliance** is always an important question
- **Phosphate clearance on dialysis** is mainly determined by duration - supplementation may be required with continuous treatments or on daily dialysis
- **Parathyroidectomy** is usually required at some point in the lifetime of patients who have spent many years on dialysis
- **Cinacalcet** activates calcium-sensing receptors, leading to reduction of PTH secretion and falls in serum Ca and PO<sub>4</sub> through reduced mobilisation from bone. It seems very effective, but is expensive, track record is still relatively short given the context in which it is used, and its place in long-term therapy has yet to be established.

### **Parathyroidectomy: management of calcium**

Patients with substantial hyperparathyroidism may develop severe hypocalcaemia immediately after parathyroidectomy. This is most likely if bone disease is obvious and severe, in which case it may be minimized by pre-administration of alfacalcidol or calcitriol (see third point below).

- Measure [Ca] after return from theatre and at 2-6h intervals according to results. If normal at 24h, further trouble is unlikely
- If Ca <1.9mmol/l, or if symptomatic, treat by administering 10% Calcium gluconate, 10mls 2-4 hourly by infusion (diluted). Extra Ca can also be given on dialysis or by addition to CAPD fluid
- Prevent and treat by giving oral alfacalcidol (or calcitriol) 1-4 micrograms per day for 3-5 days preoperatively, and continued postoperatively until [Ca] is normal. This is safe for 3 days even if [Ca] is high preoperatively
- Check [Mg] intermittently if [Ca] is causing trouble.

Further information on the web:

- Patient information on phosphate in diet section.
- Shared care protocol for sevelamer.

## Osteoporosis prevention on steroids

The various (many) guidelines were not designed with kidney disease in mind. Locally we have not used bisphosphonates very often in this group, tending to favour vitamin D preparations that are already in use. Two specific concerns are:

- Unknown effects of bisphosphonates on bone already suffering from renal osteodystrophy
- Calcium loading in patients with ESRD

For patients with normal or mildly impaired kidney function, the Lothian Joint Formulary Committee recommendation for patients receiving  $\geq 7.5\text{mg}$  prednisolone or equivalent is:

- Patients over 65 – alendronate 70mg weekly, plus Adcal-D3 1 BD
- Patients Under 65 – Treat if DXA t score  $>1.5$  (osteopenia or osteoporosis). Adcal D3 alone if not. Etidronate is an alternative to alendronate.

Risk factors for osteoporosis, or demonstration of osteoporosis will influence decisions. Note that bone density measurements are of unknown relevance in patients with renal failure, in whom osteodystrophy may confound interpretation.

### COMMENCE PROPHYLAXIS WHEN STEROID TREATMENT IS INITIATED

Risk Factors
Menopause $<45$ years
Personal or family history of low-trauma fractures
Amenorrhoea
Slender build ( $\text{BMI} < 20\text{kg/m}^2$ )
Immobility

#### Alternative Treatments

- When administering pulse methylprednisolone, an alternative: iv. Pamidronate 30mg iv once only – may repeat in 6 weeks.
- HRT in post-menopausal. Oestradiol or testosterone if these are low. (Bone density monitoring recommended if so – but this may be misleading in CRF)
- Alfacalcidol or calcitriol 0.25 mcg/d, 3-7 days weekly if bisphosphonates are not tolerated, or if their unproven safety is of concern (eg in younger patients). Should also receive supplemental calcium. Adcal D3 or Calcichew D3 should be used for those with normal renal function ( $\text{GFR} >50\text{mls/min}$ ).

More info Lothian Joint Formulary guidance, see website.

## PERITONEAL DIALYSIS

PD is an apparently simple form of dialysis that provides the motivated patient with the ability to perform dialysis at home, at work and at leisure. Travel is much easier and many patients value their independence from hospital.

**Continuous ambulatory PD (CAPD):** involves up to 4 exchanges a day of volumes of 1.5-3 litres (usually 2) of fluid. This fluid currently comes from two manufacturers and in multiple formulations as follows:

- |            |   |
|------------|---|
| Baxter:    | 1.36%, 2.27% & 3.86%, all glucose-based<br>Icodextrin (Extraneal) (polymeric high osmolality)<br>Physioneal (bicarbonate buffered, dual chamber bags)<br>Nutrineal (amino acid-based) |
| Fresenius: | Numbers 2,4 & 3 (this order is correct for increasing glucose concentration!)<br>Balance (low GDP, physiological pH)  |

Low calcium versions of most of the above are available.

**NOTE: Icodextrin and blood sugar measurements:** maltose and perhaps other degradation products of icodextrin cause falsely high readings with some commonly used blood glucose monitoring devices. Hypoglycaemia can therefore be mistakenly excluded in diabetics, or hyperglycaemia diagnosed in any patient, with potentially serious consequences. For diabetic patients receiving icodextrin we are providing a monitoring method that avoids this hazard, the Medisense Optium monitor, but monitoring is still likely to be performed elsewhere. Patients must be made aware of this potential problem, and icodextrin should be prescribed with caution to diabetics.

- **Automated PD:** uses one of a number of devices that are ideal for overnight administration of large volumes of fluid
  - Home choice (Baxter)
  - Sleepsafe (Fresenius)
- Quantum a nifty little machine that is able to do a single extra exchange overnight to 'extend' daytime CAPD

### PD catheter insertion

Done by the Transplant surgeons laparoscopically under GA. The patient should be seen by the CDT one week prior to insertion, and the preferred exit site marked with a 5x5 cm box, taking into account belt line and the patient's line of vision. Laxatives, Hibiscrub, and appropriate instructions are given. Antibiotic prophylaxis for this procedure is 1gm IV flucloxacillin in theatre, or 1gm IV vancomycin on the ward, pre-op for those who are penicillin allergic.

## **PD peritonitis**

Signs and symptoms include abdominal pain, pyrexia, cloudy PD effluent, but these may not all be present initially. The large volumes associated with APD may make cloudiness less apparent. Patients with any of these features must be reviewed and samples taken from their PD bag. If they do not have their own transport fix a 2-hour emergency ambulance. A fluid white cell count of  $\geq 100/\text{mm}^3$ , or a differential wcc of  $>50\%$  neutrophils on any total wcc is diagnostic. Initial microscopy is often not helpful but treatment should not be delayed after cultures are sent. Immediate management is:

- vancomycin 30mg/kg dry body weight intraperitoneally, single dose
- oral ciprofloxacin 500 mg bd.

Intraperitoneal antibiotics should remain in-situ for 6 hours; to this end, those patients normally on APD are converted to CAPD. Most patients are allowed home, but admission is required for pain control, systemic upset etc.

Subsequent antibiotics are adjusted on the basis of culture and sensitivity results, and the patient's progress. Failure to improve suggests fungal or secondary peritonitis (see below), or just a bad infection. Catheter removal is often necessary in such cases and should not be delayed.

Further details on web.

## **Fungal peritonitis**

Rare, but serious with high morbidity and mortality. The priority is usually catheter removal. Pending surgery, yeasts should be treated with oral fluconazole 200 mg daily, continued for 2 weeks after catheter removal, and other fungi require amphotericin or flucytosine.

## **Secondary peritonitis**

When there is a failure of clinical improvement, particularly with Gram negative or mixed organisms, a surgical cause should be sought. Where necessary seek a surgical opinion, asking for not only catheter removal if appropriate, but either a laparoscopic search for pathology or a mini-laparotomy.

## **Troubleshooting peritoneal dialysis problems**

### **Exit-site infection**

Treat with oral antibiotics, usually flucloxacillin 250-500mg qid for 2 weeks. Report infection to CDT. Failure to improve may require admission for IV antibiotics and in some cases surgical repair. The incidence of Staph. aureus exit site infection is increased by the nasal carriage of that organism, and both impact on peritonitis rate. An attempt at eradication with nasal mupirocin is of benefit in reducing the infection rate.

### **Poor drainage**

Most poor drainage is temporary and is due to the catheter being caught in amongst loops of bowel. Administration of half a sachet of Picolax whilst stopping PD for a short period is usually curative. Poor drainage due to fibrin deposition sometimes occurs, particularly in relation to peritonitis and may be eased by the use of IP heparin. A minority of cases are caused by adhesions, kinking, malposition or omental plugging and may require surgery. PD catheters are highly mobile, so that the finding of a 'displaced' catheter on X-ray therefore means little in relation to poor drainage. So don't do them unless there's a really strong reason. There is absolutely no need for lateral pelvis films, which give a very high radiation dose.

### **Blocked PD catheter**

The absolutely blocked PD catheter may be salvaged by a urokinase infusion. This can however be painful.

### **Bloody bag**

Bloody PD effluent can be frightening but is usually benign. In women it can occur during menstruation or at ovulation. In most cases the patient can be reassured and sent home. Rarely it may be seen in a leaking aortic aneurysm or other intra-abdominal crisis.

### **PD leak**

A leak of PD fluid from the peritoneum to the abdominal wall may be suspected by increasing flank oedema in the absence of other fluid accumulation, and in association with poor fluid drainage. Proving this can be difficult. Ultrasound may detect a defect in the anterior peritoneum, usually in relation to the catheter. The most sensitive test however is CT scanning of the abdomen using a contrast-filled bag that has been in place for at least one hour prior to the scan (100mls contrast is diluted into one bag; PD nursing team do this on the ward). Treatment is usually by rest but a period of nocturnal APD with no daytime exchanges (to minimize intra-abdominal pressure) may avoid that. Haemodialysis is sometimes necessary.

### **Contaminated line**

Patients who may have (if there is any possibility) inadvertently disconnected their system and contaminated their line should receive prophylactic antibiotics in the form of a single dose of vancomycin 30 mg/l PD fluid given intraperitoneally and left in-situ for six hours.

## **Dialysis adequacy**

Measurement of PD adequacy requires collection of PD effluent, urine (for residual function) and blood. Adequacy should be checked regularly and after any problems, and the regimen adjusted in response. Measurement should not be performed for the first 8 weeks of treatment, but then every 4-12 months. Residual renal function is significant in most people who are doing well on PD.

There is continuing controversy about acceptable targets for PD, but it

seems likely that too little dialysis is bad. Weekly creatinine clearance and Kt/V are the accepted measurements. The latter is more dependent upon PD, the former on residual renal function, and there may be marked discrepancies between the two. Currently accepted minimum targets are: (DOQI and our own):

CREATININE CLEARANCE at least 60 litres per week/1.73m<sup>2</sup> (50 l/week in low transporters) (normal creat clearance is around 1000 litres per week)

TOTAL Kt/V at least 2.0 per week (1.7 in low transporters)

For a description of how to measure Kt/V and creatinine clearance on PD, contact the CDT, or read UpToDate.

### **Peritoneal Equilibration Testing (PET)**

The PET test describes how rapidly solutes are absorbed from the peritoneum in a standardised manner. Basically, higher rates of solute transfer are good for biochemical clearance of small molecules, but bad for ultrafiltration. Low transporters may receive poor dialysis; they should benefit from long dwell times (eg CAPD). Fast transporters achieve inadequate ultrafiltration, and may benefit from rapid-cycling PD (e.g., APD). The PET test is performed using a standard 2 litre 2.27% or no. 4 bag, which is left in-situ for 4 hours. Blood and PD fluid samples are taken for creatinine and glucose at 0, 2 and 4 hours and are used to assess D/P ratios. See the CDT for the full protocol, and for tables to interpret the results, or the very good section in UpToDate.

### **Routine monitoring for PD patients**

- U & E, LFTS, FBC, glucose 1-3 monthly at clinic as indicated
- HepBs Ag, HepC Ab, HIV Ab – annually
- Iron studies as indicated (as for HD)
- PTH at least annually, more often if evidence of bone disease
- Lipids – at least annually (more often if elevated)
- Adequacy – at about 3 months and then 6-12 monthly. Repeat after peritonitis, change of regimen, or if previous result inadequate
- PET test – at 3 months then as indicated
- Tubing change – every 6 months for Baxter system, annually for Fresenius

## POISONING

Haemodialysis is effective at removing a number of low molecular weight, water-soluble poisons with a low degree of protein binding. It is indicated when elimination by other routes is unacceptably slow, especially if renal failure is contributing to this. The following agents are usefully removed:

Inorganic acids (Acetic, Phosphoric, Formic)  
Alcohols (ethanol, methanol\*)  
Barbiturates  
Chloral Hydrate  
Ethylene glycol\*  
Thallium

**Lithium** – is the ideal poison for removal by dialysis. Renal tubular reabsorption leads to a renal clearance of 10-40ml/min when hydration is adequate, whereas haemodialysis can achieve clearances of up to 150ml/min. Some suggested indications for dialysis are:

- Lithium level of  $\geq 4\text{mM}$
- Lithium level of  $\geq 2.5$  with severe symptoms or in the presence of significant renal impairment or sodium retention (eg heart failure, liver disease)
- if the level is falling slowly

Rebound is normal, because of intracellular stores and the fact that slow-release preparations are commonly responsible for poisoning. Try 6 hours of HD on a large kidney with maximal flow rates. Check levels 1-2h later.

**Salicylates** Although there is a high degree of protein binding at therapeutic levels, this is saturated at toxic doses, and salicylates become more widely tissue distributed, extending half life 3 to 4-fold to 15-30h. Alkalinization of plasma and urine are beneficial.

Dialysis should be considered when

- salicylate levels are  $>800\text{mg/l}$
- impaired renal function or fluid overload
- serious toxicity (eg coma, or deterioration despite treatment)

\* Ethanol should be co-administered to inhibit metabolism to toxic product

Haemoperfusion over activated charcoal is more effective if poisons that are protein-bound in the circulation bind to it well. This applies to (for instance), theophylline, some anticonvulsants, procainamide. Practically this is now so rarely undertaken that obtaining the charcoal cartridge may be difficult.

Poisons Units will give detailed advice for specific drugs. The Unit has a log-in for the online database TOXBASE, which is viewable online at <http://www.spib.axl.co.uk>

User name: H849

Password: RENAL9QZ

## Pre-dialysis management of CRF

The aim is to:

- Prevent progression of CRF so far as possible
- Prevent complications of CRF
- Ensure timely and appropriate planning of RRT when necessary (see next section)

<b>Fluid balance</b>	prevent hypervolaemia (Na restriction, diuretics) avoid hypovolaemia (no oedema, postural hypotension) watch out for sodium-losing patients who will benefit from Na supplementation.
<b>Hypertension</b>	q.v.
<b>Diet</b>	q.v.
<b>Hyperlipidaemia</b>	q.v.
<b>Acidosis</b>	prescribe $\text{NaHCO}_3$ to keep plasma bicarbonate $\geq 20$ if Na load permits
<b>Osteodystrophy</b>	prescribe alfacalcidol (calcitriol equally effective) when there is hypocalcaemia or when PTH $> 2x$ normal in the presence of normal serum calcium. $\text{PO}_4$ should be kept $\leq 1.8$ mmol/l by dietary restriction and the use of phosphate binders. See Renal Osteodystrophy.

Planning – see next section

## PREPARING PATIENTS FOR RENAL REPLACEMENT THERAPY

The appropriate time to prepare someone for dialysis can be difficult to judge. It is essential that preparation should not be left until too late; it may, however, be inappropriate to burden people with this information too early.

- advise patients (and others e.g., spouse, as appropriate) re need for RRT
- introduce concepts of PD and HD early
- emphasise the importance of patients' preference
- discuss transplantation (including pre-emptive transplantation)
- remember that No RRT is one option for management

### **About 6-12 months before expected ESRF, refer to**

- Social worker - download a proforma and copy of clinic letter.
- Community Dialysis Team: a written referral (use downloadable proforma and copy clinic letter, including:
  - Relevant PMH
  - Any medical contraindications to PD/HD
  - Very brief social history
  - Urgency of referral
- Transplant Team (unless clearly contraindicated).  
See next page for additional investigations

The CDT will arrange visits to see PD and HD (repeat if necessary), and will arrange invitation to a Patients' Evening. They will see potential PD patients at home; those who are likely to do HD can be seen at clinic visits.

Get feedback from above visits/referrals, and at monthly Pre Dialysis MDT meeting check understanding.

**Hepatitis B immunization** should be carried out by request to the patient's GP (see antimicrobial policy).

**AV access** (fistula) should be created at least 6 months before starting HD to ensure that it is successful and mature. Some may require multiple operations.

- Refer to Vascular Access Coordinator (Ext 21199) and use specific referral proforma.

**CAPD catheter** should be inserted at least 2 weeks before starting PD

- Operation dates are arranged through either the transplant or vascular surgical team. There is a coordinator for vascular access.

**See below for blood tests required** Cytotoxic antibodies should be checked each OP visit for patients on the transplant list, or monthly.

**Data accuracy** Keep computer screens updated with dialysis / transplant decisions made, and other information requested. Liaise with consultants or computing staff if unclear how to do this.

## Pre-dialysis and pre-transplant investigations

Proforma suitable for inserting into front of notes

Test	Mandatory		Date	Comment
	pre-dial.	pre-trspl.		
<b>VIROLOGY</b>	EBV is not routinely tested for but can be investigated on saved samples.			
Hep B, C				
HIV				
CMV				
VZV and EBV				
<b>IMMUNOLOGY</b>	Measure CABs at each visit in patients on the transplant list (monthly for HD patients)			
Tissue typing				
Cytotoxic antibodies				
Blood group				
<b>RADIOLOGY</b>	No routine imaging of bones required pre-transplant, but PTH should have been measured. Surgical team may request pelvic XR in some.			
CXR				
Renal imaging				
Other				
<b>UROLOGY</b>	Pre-transplant, consider further investigation if history of recurrent infection, obstruction, or bladder dysfunction			
<b>CARDIOLOGY</b>	Pre-transplant, echo mandatory if any history of cardiac disease, and refer if symptomatic or high risk.			
ECG				
Echo	?			
Other (eg ETT)				Mandatory if for SKP
<b>GASTRIC</b>	If any symptoms or previous history to suggest increased risk of ulceration			
Endoscopy				
<b>REFERRALS</b>				
Community dialysis team				
Social assessment				
Vasc. access/ PD cath				
Transplant team				

This page can be downloaded from the web version of the handbook.

## PRESCRIBING

### Specific Points

Doses and intervals of many drugs are altered in renal failure and should always be checked. Renal pharmacists are the best sources of information on anything non-routine. Important examples of drugs that require special consideration are:

**Antibiotics** – see antimicrobial section for more detail

**Aminoglycosides, Vancomycin** – doses in dialysis patients are days apart.

**Tetracyclines** – only doxycycline can be used in renal failure.

**Nitrofurantoin** should not be prescribed in renal failure.

**Cimetidine** should be avoided in favour of ranitidine or other H2 blockers.

**NSAIDs** should generally be avoided in significant renal impairment, though in mild/moderate CRF they can be used after discussion and with monitoring if alternatives are much less effective. Avoidance is not essential for dialysis patients but risk of GI bleeding is probably already increased in ESRD.

**Opiates** – the effects of almost all except for fentanyl are very much prolonged in renal failure and there is a great tendency to accumulation. Use with caution.

**Aluminium or bismuth**-containing compounds should be avoided in renal impairment. (Al hydroxide with caution – see osteodystrophy)

**ACE inhibitors** may cause hyperkalaemia in renal failure (check in 3 and 7 days if high risk), and a steep decline in function in renal artery stenosis (check creatinine in 1 week or in 4 and 10 days if high risk). Re-checks should be undertaken after substantial increases in dose, or if loop diuretics are added or much increased. Note that only a 20-30% rise in creatinine should be regarded as significant; a small rise is normal. UK CKD Guidelines recommend a cautious 20% (15% reduction in eGFR). See *website – GP Info: 'How to start ACE inhibitors'*.

**Heparin** – low molecular weight heparins are renally excreted and should not usually be used above prophylactic doses. See Anticoagulation.

## PROTEINURIA IN RENAL DISEASE

Heavy proteinuria is characteristic of glomerular diseases. 'Nephrotic range' proteinuria (>3.0g/24h on pbD definition) is always glomerular in the absence of urinary infection. Proteinuria is also an important prognostic indicator for the progression of chronic renal damage.

Relating protein excretion to excretion of creatinine corrects for variations in urine concentration, and measurements of this ratio on single samples can in most circumstances substitute for 24h collections. The conversions shown here are approximate. Those with lower creatinine production will have a higher ratio for a given rate of protein excretion. This conversion assumes a daily creatinine excretion of approx 10 mmol – on the low side for muscular men.

<b>Protein excretion g/24h</b>	<b>Protein/creatinine mg/mmol</b>
<0.15	<15
1	100
3.5	350
10	1000

### Normal ranges for microalbuminuria.

Microalbuminuria estimation is of little value in patients known to have renal disease, but may be useful for new patients.

<b>An albumin excretion rate of</b>	<b>is the normal value for</b>
< 30mg/24h	24h collection
< 2.5mg/mmol (m)	Albumin/creatinine ratio
<3.5mg/mmol (f)	

Note that up to 150mg/24h is normal and about 30mg of this is albumin. A concentration of about 150mg/l corresponds to 'trace' on a urine dipstick, and 300mg/l to 1+, but this is obviously affected by urine concentration/dilution.

As proteinuria rises, albumin forms a relatively larger proportion; about 50% at 300mg/d and 70% at 1g/d. So the referral threshold of 100mg/mmol is not very different for albumin and it may be simplest to quote the same.

## **RADIOLOGY**

### **Dialysis patients/osteodystrophy**

There is no routine screening. Where hyperparathyroidism is being quantitated, a hand XR alone will usually suffice (request 'hand XR for hyperparathyroidism'). Views of the pelvis may be justified in some circumstances – explain these on the request form if so.

### **Renal arteriography and angioplasty**

MR Angiography has greatly reduced use of conventional angiography.

However concerns about Gadolinium-containing MR contrast media and Nephrogenic Systemic Fibrosis (Nephrogenic Sclerosing Dermopathy) mean that MRA be used very cautiously if at all in dialysis patients and patients with severe renal failure. For conventional angiography, patients admitted on day of procedure, earlier if less fit. Overnight stay not routinely required for diagnostic angiograms, usually suitable for day case unit. Should stay overnight for interventions.

### **Pre-procedure**

Warfarin - stop 3 days in advance and check clotting on morning of procedure

Non-steroidals other than aspirin: stop on day of procedure and for 48h

Metformin - omit on day of procedure and withhold for 48 hours, restart if function OK

All other medications including anti-hypertensive and anti-anginal to continue  
*Fluids only* for 2 hours prior to procedure (can have light breakfast if late am procedure)

### **Investigations – recent results to be available for**

FBC (Hb must be >80g/l, Pts >100)

U&E (K should be <5, if not give IV 10% dextrose 20mls/hr and 5mg Salbutamol neb)

Coagulation screen - only if on anticoagulant or abnormality likely

ECG if history of IHD, glucose if diabetic.

### **Observations**

Record BP – postpone only if very high

Assess and document peripheral pulses

### **Fluid management**

Ensure patient well hydrated and good urine output prior to contrast (if pre-dialysis) - if in doubt put up 6hrly 500mls N Saline. Do not fluid overload dialysis patients. Avoid diuretics. Beware that after stenting/angioplasty, some patients may become polyuric.

### **Consent**

Should be done in OPD for diagnostic studies. Radiologist should obtain consent for interventional studies, need to warn of risks of contrast, and catheter-related complications including embolism, arterial occlusion, bleeding from puncture site, loss of renal function, occasional need for

surgery after intervention.

### Post-procedure

Diagnostic studies - mobilise at 4 hours if no complications

Interventions – mobilise at 6 hours, and overnight stay

Pulse and BP: hourly for 4 hours, then 6 hourly overnight if IP

Urine output: beware polyuria post-angioplasty

Assess pain, wound, haematuria

Check U &E following morning if kept in

Warfarin can be restarted the following day

Consider Aspirin 150mg if angioplasty/stent – ask radiologist if not clear.

### Myeloma skeletal survey

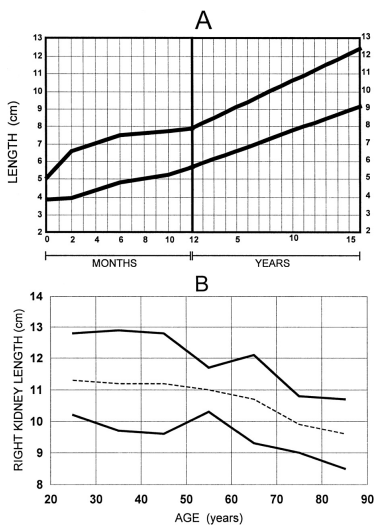
(Request in these words) - includes CXR, lateral skull, lumbar spine, pelvis, upper femur; plus 2 views of any symptomatic region.

### MR and CT angiography

Undertaken only after discussion with radiologist.

### Ultrasound

The most requested and most frequently useful investigation, but observer dependent – speak to the operator if any question. Renal length averages 11 cm in adults, but there is some variability in measurement, and differences of up to 1cm in repeated measurements are common. Figures showing 95<sup>th</sup> centiles are from O'Neill, *Am.J.Kid.Dis.* 35:1021-38 (2000).



## Preventing contrast nephropathy

This is rare in the absence of its chief risk factors:

- Under-hydration/hypovolaemia
- Renal impairment
- Myeloma and possibly diabetes

The use of lower doses and less hyper-osmolar contrast media reduces risk.

Testing of several putative protective treatments has shown them to be harmful – this includes mannitol, diuretics, and dopamine. The use of N-Acetyl Cysteine (NAC) prophylactically probably does no serious harm, but the evidence that it prevents severe acute renal failure is weak.

If a patient is at increased risk, does this alter the balance of risk for doing/not doing the rest? If not:

- Warn the patient
- Administer fluid and withhold diuretics
- Consider NAC (but it cannot be regarded as essential)

### Protocols for prevention of contrast-associated nephropathy

- **Keep contrast dose low**
- **Administer fluids and omit diuretics** - see below for amounts used in trials
- **N-Acetyl Cysteine (NAC)** is of uncertain additional value

#### IV saline/bicarbonate

0.9% saline @ 1ml/kg/h from 2-12h pre procedure, continuing to 24h

One trial found NaHCO<sub>3</sub> to be superior to saline; requires confirmation:

1.28% NaHCO<sub>3</sub> @3ml/kg/h for 1h pre-, then 1ml/kg/h for a further 6h

#### Oral NAC protocol (in addition to fluid regimen)

Can be given orally but it is very bitter. Give 600mg twice daily for 48h starting the night before the procedure. 3mls of 200mg/ml intravenous NAC solution may be diluted up to 12ml with orange juice or cola. It may cause nausea or vomiting and rarely hypersensitivity reactions.

#### Immediate Saline/ IV NAC regimen

NAC 150mg/kg in 500ml N saline over 30 mins pre-procedure, then

50mg/kg in 500ml N saline over 4h post-procedure.

This is a much higher dose than the oral regimen and may cause side effects.

## RENAL BIOPSY

Patients admitted on day of procedure, or earlier if less fit. Must be arranged in advance via ultrasound sec or by discussion with radiologist who has a list. If deemed suitable for day care, can be assessed by Clinical Nurse Practitioner. If admitted to general wards to be assessed by medical staff.

### Pre-procedure:

- Aspirin/clopidogrel stop 1 week in advance; other NSAIDs omitted on the day
- warfarin stop AT LEAST 2 days in advance if indication permits. Some patients will need iv heparin until day of biopsy.
- all medications including anti-hypertensives and anti-anginals to continue (usually patients' own supply)
- pathology request form must be filled in by renal team and **attached to front of notes**. Do same with consent form if obtained in advance.

Fluids only for 2 hours prior to procedure but no need to fast.

### Investigations and observations

Ensure no major change in condition or therapy (seek advice if there is)

- BP should be <160/90

Recent results for the following must be available:

- FBC (Hb must be > 80g/l, Pts > 100x10<sup>12</sup>/l)
- coag screen (PT and APTT must be within 3 seconds of control value)
- Group and save
- U & E in patients on dialysis, or if uraemic

if any values are outside these ranges the Registrar and the Radiologist should be informed

**Consent:** may be obtained in OPD. Should be attached to front of notes, with pathology request form. Inform of small risk of bleeding, very small risk of requiring intervention (including surgery). See Patient Info, below.

### Post-procedure:

- bed rest 6hrs: first 2h lying flat
- pulse and BP: half-hourly for 2 hours  
then hourly for 2 hours  
at 6h, then 6-hourly to 24h if still an inpatient
- advise patient to drink plenty
- pain relief: paracetamol is adequate in most instances
- assess and document:
  - biopsy site
  - presence/ absence haematuria (macroscopically only)
  - dialysis or uraemic patients: check [K] the following day
- aspirin, warfarin can be restarted the following day if uncomplicated. Discuss reintroduction of heparin if patient at high risk of thrombosis.

**Discharge** at the agreed time if all the above satisfactory. Minimum is 6h if suitable for day-case biopsy, see below. In all cases

- patient must have passed urine
- 6h or final BP and pulse should have been recorded
- patient must be given a number to call if problems (renal ward)
- results usually given at an outpatient appointment in near future
- returning to work and other activities: a day or two off work is usually enough. Heavy manual activities should be avoided for a few days. No other special precautions are required.

### **Day case biopsies**

These are suitable if the following conditions can be met:

*Inclusion criteria:* Low risk  
Suitable responsible person at home  
Can arrange own transport for discharge in evening

*Exclusions:* Anticoagulation  
Creatinine > 250 micromol/l  
Serious comorbid disease

Diabetes mellitus does not automatically exclude day case biopsy as there is no requirement to fast.

Patients must have an appropriately early biopsy.

**Urgent biopsies** – warn renal pathologist

**Out of hours procedures** – contact the consultant pathologist *first*

### **Further info on the web:**

Information for patients about renal biopsy

## STONE DISEASE

Recurrent stone formation is common, but people who have frequent early recurrences should be screened for risk factors. Check:

Blood	Renal function Ca and PO <sub>4</sub> Uric acid HCO <sub>3</sub> PTH
Urine	Infection Request 24h urine for 'stone screen', (plain bottle) to check volume, calcium, oxalate, Na, urate, cystine. Note that creatinine and protein need to be requested separately.
Stone	Don't forget to analyze the stone itself
Family history	hypercalciuria, medullary sponge kidney, distal RTA, Dent's disease
Drug history	occasionally stones formed from drugs (including ephedrine)
Dietary assessment	important. See section on Diet.

As for Protein, urinary calcium can be measured as a ratio with creatinine, instead of a 24h clearance:

Ca/creat ratio	Comment
<0.6	Normal
0.6 – 0.8	Equivocal
>0.8	High

### Management principles

Important principles are common to most stones

- Maintain high urine volume, especially at night
- Restrict dietary sodium
- Maintain good *dietary* calcium intake
- Consider thiazide for hypercalciuria (avoid loop diuretics)
- High protein diet is associated with stones - reduce

For management of individual metabolic abnormalities, seek specific information.

Further info on the web: Patient information

## SURGERY IN PATIENTS WITH ESRD

In order to prevent problems arising please consider the following:

- Patients being admitted for surgery should usually be admitted on the morning of the day prior to surgery. This will enable adequate time for the SHOs to admit the patient, and identify any problems which require to be addressed prior to surgery. This will also mean that the anaesthetist will be able to see the patient and be aware of any potential anaesthetic problems. Patients should not be advised to come for surgery on the afternoon or evening of the day prior to surgery
- If we think there is a case for day-case surgery, then this should be discussed with the anaesthetist and a clear protocol arranged
- If on dialysis, then some reorganisation of the dialysis schedule may well be required. For haemodialysis, arrangements should be made for the patient to have dialysis on the day before surgery. The plan should be put in writing with copies to the Dialysis Unit and Ward SHOs (if to be an in-patient)
- Urea and electrolytes, creatinine, and a full blood count should be done on the morning of admission so that results are available for the anaesthetist when they assess the patient. In addition, for HD patients (or APD or CAPD if dialysis has been interrupted) urea and electrolytes should be done urgently, as early as possible, on the morning of surgery and these results should be sent with the patient to theatre
- If hyperkalaemia is anticipated, and since patients may be fasted overnight, there is a reasonable case for giving a slow dextrose infusion overnight and for some patients a single dose of calcium resonium on the evening prior to surgery. If hyperkalaemia is anticipated, proper planning avoids a crisis on the morning of surgery: SEE NEXT PAGE
- In patients with ESRF, veins are precious and we should avoid siting i.v. cannulae in veins which may be used for future vascular access - this applies particularly to the cephalic vein in the forearm, see 'Veins and vascular access'. Clearly if a patient is going for vascular access procedure, then cannulae should not be placed in the arms being used for access
- Postoperatively, remember problems with prescribing of analgesics, especially NSAIDs and opiates, in different patient groups. There is a specific protocol for management of epidurals and patient-controlled analgesia (PCA, using fentanyl) in renal failure

## **Peri-operative management of potassium**

### **Pre-operatively**

The objective is to ensure that  $[K^+]$  is  $\leq 5\text{mmol/l}$ . Post-dialysis  $[K^+]$  should be checked at least 5 minutes after the end of dialysis. It should be well below 5.0 if possible (but in the normal range). This may necessitate arranging dialysis two days running, in patients who are frequently hyperkalaemic.

#### **If 5.0 – 5.5:**

This may be too high for some types of surgery – eg prolonged, or likely to involve too much blood loss. If acceptable (discuss with anaesthetist), use the following maintenance regimen to prevent a further rise

- infuse 10% dextrose at 40ml/h (without insulin in non-diabetic patients)
- give nebulised salbutamol 5mg 6-hourly

If there is much delay, recheck  $[K^+]$

#### **If 5.5 – 6.5:**

This is likely to indicate a need for further dialysis pre-operatively – and should have been avoided. If surgery is to go ahead,

- give 50mls 50% dextrose with 5u Actrapid over 15 minutes
- follow with maintenance regimen above

Such decisions will normally be made at a senior level.

#### **If > 6.5:**

Dialysis is indicated except in an emergency. The relative risks then have to be judged.

### **Post-operatively**

Potassium should be checked after the patient returns. This may not be necessary if potassium was under 5.0 pre-operatively, and the patient has had superficial surgery carried out under local anaesthesia, with insignificant blood loss.

## SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)

### Acute severe disease

Patients with impaired and usually deteriorating renal function with diffuse proliferative nephritis + crescents, or critical disease affecting other organs have been treated with cyclophosphamide-based regimens for many years, and these remain the standard therapy today. Daily cyclophosphamide may still be used in very severe disease, but pulsed regimens have largely substituted in other circumstances. Alternatives to cyclophosphamide are being increasingly explored.

**Oral cyclophosphamide regimen.** Prednisolone at 1mg/kg/day and cyclophosphamide at 2mg/kg/day, rounded down to the nearest 50mg. This is usually 100 or 150mg/day. (2.5mg/kg/day almost invariably results in leucopenia after 2-3 weeks treatment followed by stop-start treatment). Use less in over-55s. Cyclophosphamide is arbitrarily given for 12 weeks followed by a change to azathioprine at 2mg/kg/day (usual start dose = cyclophosphamide dose) or to MMF. Prednisolone is tapered but generally more slowly than in vasculitis once below 20-25mg/day.

**Pulsed cyclophosphamide regimens** are described in detail below. These reduce toxicity and cumulative dose. They also prolong the patient's exposure to cyclophosphamide, possibly an advantage in chronic/relapsing disease. Note that doses should be adjusted to achieve lowering of wbc in severe disease. Dose interval may also be reduced.

**Mycophenolate Mofetil (MMF)** is being increasingly used and early trials have not shown that it is inferior to cyclophosphamide. However SLE is a disease with long evolution. MMF has the advantage of avoiding gonadal toxicity and probably has fewer severe side effects overall. The dose used in trials has been 3g/day, which may not be well tolerated. In subacute setting usually start at 500mg BD and increase at intervals of a few days.

**Plasma exchange** may be used in patients with very severe disease, especially if apparently not responding to drug treatment. Possible indications might be dialysis-dependent disease, encephalopathy.

**Pulse methylprednisolone** – we hardly ever use this, as there is no evidence that it adds anything to the above regimens, which all include prednisolone 60mg/kg/d, and there are added risks of infection, effects on bone, and sometimes severely exacerbated hypertension.

### Maintenance after acute severe disease

In general if patient goes into remission and extrarenal manifestations are controlled, prednisolone is gradually tapered to 10mg/day by 6-9 months, and 5-7.5mg by 1 year. Azathioprine is generally substituted for cyclophosphamide (as for systemic vasculitis) at 3 months; MMF is now an alternative, but the minimum dose should be 1g BD.

We generally taper and stop steroids and then aza/MMF at 18-24 months except in patients with continuing active disease, or who have had several or particularly severe flares of disease. Any deterioration in renal function, especially if baseline function is good, is usually managed after repeat biopsy. Minor or extra-renal flares are often treated with increased steroids followed by slower reduction.

## Pulsed cyclophosphamide therapy

750mg/m<sup>2</sup> is given with Mesna at monthly intervals for 6 months; 500mg/m<sup>2</sup> if elderly or at increased risk of leucopaenia. If WBC nadir (10-14 days post dose) is much >5,000, next dose is increased by 250mg/m<sup>2</sup> to max 1g/m<sup>2</sup>. Consider reducing by 10-15% if WBC <3,500 or neutrophils <1,500. Delay subsequent doses if neutropenia persists (neutrophils <2,000). Reductions for low GFR: see below. Shorten dose interval in severe disease.

Prednisolone is usually increased at the start of this treatment and tapered as above. Three 2-monthly (or two 3-monthly) doses are given after the first 6 pulses. At one year most patients are converted to azathioprine/ MMF and then managed as above. Conversion can be delayed until later, and cyclophosphamide pulses continued for longer in patients believed to be at high risk from recurrent disease.

Haemorrhagic cystitis is very uncommon when cyclophosphamide is used in this way. Mesna may cause fixed drug eruptions.

### Protocol for IV pulsed cyclophosphamide

#### First pulse

Time	
0	500ml N saline over 2h plus 500ml oral fluid.
1.5h	Ondansetron 8mg oral or granisetron 1mg oral, and dexamethasone 10mg orally if patient not taking prednisolone (unlikely).
2.0h	Cyclophosphamide in 250mls N saline over 1h, 750 mg/m <sup>2</sup> Mesna (20% of the cyclophosphamide dose) added to the same bag.
3.0h	Mesna (40%) orally at 4 and 8h after end of infusion (400mg tablets). Ondansetron 8mg orally 12 hourly for 1-3 days AND Domperidone 20mg orally 6h prn for 3 days. Advise oral intake of 2.5-3 litres over next 24h.
10-14d	Check wbc (outpatients or GP).

For IV cyclophosphamide/Mesna, contact Pharmacy at least one day in advance so that it will be ready when the patient arrives. A detailed protocol will come from Pharmacy with the drug.

Patients should be cautioned to seek help if they become febrile or otherwise unwell during cytotoxic therapy of this type.

#### Subsequent pulses

- Ensure WBC recovering before administering next dose (check 1-7d before).
- Adjust dose as described above.
- Review antiemetics. If acute vomiting, (1) change oral premed to IV, (2) add second dose of ondansetron/granisetron on day 1, and/or (3) add haloperidol 1.5mg BD orally on day 1.

### Oral pulsed cyclophosphamide therapy

Is possible and effective, and usually taken at home. The cyclophosphamide dose is the same as for IV therapy but spread over 2-3 days. The need for monitoring of blood tests, and adjustment of doses according to results,

applies regardless of the route of administration (see discussion of IV therapy above). Patient info sheet available on the web:

<b>Oral pulse cyclophosphamide treatment</b>
<p><b>Each day</b></p> <ul style="list-style-type: none"> <li>• Drink 2-3 litres (preferably 3) every day during this treatment.</li> <li>• Start treatment in the morning, so that you can drink plenty during the day.</li> <li>• Take an <b>ondansetron</b> tablet (8mg) just before or 1-2h before your cyclophosphamide tablets.</li> <li>• Then take the <b>cyclophosphamide</b> tablets with plenty of fluid. <i>[The total dose is as for IV pulses, but divided into 2-3 daily doses]</i></li> <li>• Take <b>Mesna</b> tablets, one 400mg tablet three times each day on every day when you are taking cyclophosphamide. One should be taken last thing at night.</li> </ul>
Cyclophosphamide and Mesna are usually continued for <b>2 or 3 days</b>
Ondansetron should be continued (8mg 12 hourly) for one or two days after that (the first time, for two or three days), to a total of <b>3 to 6 days</b> . Some people need a higher dose than this, or alternatives to ondansetron (e.g. see above)
Other drugs (e.g. prednisolone, hypotensive agents) should be taken as usual during cyclophosphamide treatment.

A blood count is needed 10-14 days after starting each pulse to catch the nadir wbc. Blood tests should be repeated a few days before the next pulse to confirm recovery, at least in the first treatment cycles. Antiemetic therapy may need to be reviewed, though symptoms are rarely severe with this protocol.

### **Cyclophosphamide dose reductions in renal impairment**

Note that these are conservative recommendations (see Vasculitis) and they should be individualised in severe disease, paying close attention to 10 day wbc, or there may be a significant risk of undertreatment.

GFR 15-50mls/min, or on CVVH	75% of dose
GFR <15mls/min, on IHD/CAPD	50% of dose

### **Gonadal toxicity of cyclophosphamide**

#### **MALES**

Risks of oligospermia increase above a cumulative dose of 100mg/kg in mature males. This amount is approximately equal to a single three month course of daily cyclophosphamide, or about 8 intravenous pulses.

Cryopreservation of sperm should be offered to all males embarking on non-emergency treatment with cyclophosphamide. Where pulse therapy is being undertaken, cryopreservation should be undertaken before the first pulse. Where a 3-month period of daily treatment is being used, and prior cryopreservation is not feasible, it is reasonable to undertake it well after the end of the course if it is likely that further cyclophosphamide may be required in the future.

### **For cryopreservation of sperm**

Contact Reproductive Medicine Laboratory to arrange sample storage.

Samples should be stored before **ANY** exposure to cyclophosphamide (either as pulse IV or oral protocol).

## **FEMALES**

Thresholds for gonadal toxicity are believed to be higher in mature women (200-300mg/kg) and in prepubertal girls (400mg/kg). The consequences – premature menopause – may only become apparent years or decades later.

Experimentally, a programme of ovarian cryopreservation has been set up jointly between Oncology at the RHSC and the Dept. Gynaecology. The programme was set up for children receiving much larger doses of cyclophosphamide than we normally prescribe, but it should be considered when very young women are likely to require substantial cumulative doses of cyclophosphamide. As above, it is best undertaken before patients have received any cyclophosphamide.

### **For ovarian cryopreservation in young women**

(This programme is experimental)

Prof Richard Anderson (Gynaecology RIE) bleep #6841 Sec 22444

Dr Hamish Wallace (Oncology RHSC) 20426

## **Newer treatments for SLE**

Several biological agents are currently being tested for their promise in SLE. Contact Dr David Kluth about most of these.

**Rituximab** – anti-CD20 antibody with proven effects in some autoantibody-associated conditions. However efficacy probably not due solely to reduction in antibody titres. Given as IV infusion of 1000mg on two occasions two weeks apart. Before starting treatment check CD19 count (B cell marker; haematology at WGH) and immunoglobulins. During infusion the patient's vital signs (bp, pulse, respiration and temperature) should be monitored every 15 minutes for the first hour, and then if stable, hourly until infusion stops. **First infusion:** 50mg/h for first 30 min, escalate in 50mg/hr increments every 30 min to max of 400mg/h. **Subsequent infusions:** Initial rate 100mg/h for first 30 min, escalated by 100mg/h every 30 min to max 400mg/hr.

- Elderly patients may require a slower infusion rate.
- If a patient develops severe cytokine release syndrome the infusion should be stopped. On resolution, the infusion can be resumed at not more than one-half the previous rate. Mild to moderate infusion-related reactions usually respond to a reduction in infusion rate.
- Anaphylactic reactions can occur to this humanised mouse antibody. CD19 count is checked before second infusion and at 2 weekly intervals for next 6-8 weeks.

## SYSTEMIC VASCULITIS AND RPGN

Vasculitis is diagnosed by clinical and pathological criteria, not by ANCA alone. Most patients have acute disease, for which the following guidelines are predominantly intended.

### Treatment of severe acute disease

Induction treatment is given to those with severe disease that needs to be controlled soon. Most receive cyclophosphamide and prednisolone. Those with the most severe disease receive plasma exchange in addition. There is controlled trial evidence for the benefit of plasma exchange in patients who have serum creatinine >600 micromol/l or who require dialysis. Other indications would include life threatening pulmonary haemorrhage or worsening disease despite conventional therapy.

#### Cyclophosphamide

2mg/kg ideal body weight, rounded down to nearest 50mg. This is usually 150 or 100mg. Use less in over-55s. This dose is continued for three months unless (i) rapid fall in total WCC from 10 to less than 4.5 or (ii) WCC falls below 3.5. The neutrophil count rather than the lymphocyte count determines the risk of infection. Closer monitoring rather than substantially lower doses is usually appropriate in severe renal impairment - these patients often have more to gain.

Pulses of cyclophosphamide can be used as described above under SLE. We generally use this for less severe, or sub-acute 'grumbling' disease.

#### Prednisolone

Corticosteroids are a vital part of therapy, but the cumulative dose of steroids is also a major determinant of the risk of infection. The usual starting dose is 1mg/kg/day, reducing approximately in line with the following scheme:

Standard prednisolone regimen		
60 mg OD	7 days	(or 1mg/kg)
45 mg OD	7 days	reduction in proportion
30 mg OD	7 days	to starting dose
25 mg OD	7 days	
20 mg OD	7 days	may be varied
17.5 mg OD	14 days	depending on
15 mg OD	until 12 weeks	clinical condition

#### Plasma Exchange

See above for indications. Daily 1 volume plasma exchanges for albumin x 5(-10). Prevent depletion of clotting factors by monitoring closely with guidance from haematologists; either giving fresh frozen plasma 2 units at the end of exchange for all patients at risk of bleeding. Additional therapy required for patients with pulmonary haemorrhage or other active bleeding.

#### Methyl prednisolone

We rarely use this unless plasma exchange is contraindicated or other pressing reason, as it may confer a substantial additional risk of infection and cause delayed avascular necrosis. 500mg once daily x 3.

## Maintenance treatment

Almost all patients with idiopathic RPGN and systemic vasculitis need maintenance immunosuppression. The need for maintenance treatment in patients with vasculitis is reviewed after one year, but after three months most patients require:

- **Azathioprine:** 2-3 mg/kg/day to be started immediately after stopping cyclophosphamide
- **MMF** is an alternative to azathioprine (see below)
- **Prednisolone:** 10-15 mg OD depending on clinical condition (and usually reducing slowly)

**Cotrimoxazole** may reduce relapse rate in Wegener's; some reserve it for chronic sinus or lung disease. Not with methotrexate (2 folate antagonists).

## Additional or alternative agents

Discussion is required before initiating any of these agents.

**Mycophenolate mofetil (MMF)** is being tested as an alternative induction agent to cyclophosphamide. It is widely accepted to be an effective maintenance agent as an alternative to azathioprine. Minimum dose should be 1g BD; in active disease, higher doses are standard.

**Methotrexate** can be used to maintain remission in patients with Wegener's, and is effective in other types of vasculitis and inflammatory arthritis. It is renally excreted and should be avoided if GFR <30, and used with caution in moderate renal impairment. Its onset of action is slow and its place is probably in chronic, grumbling disease but it can be used as induction therapy in extrarenal disease that is not life-threatening. Start at 7.5mg *once weekly* followed 48h later by folic acid 5mg. Start lower in renal impairment. Maintenance dose is usually 10-20mg once weekly.

**Anti-TNF therapies** – Anti-TNF therapy is currently reserved for those with evidence of persisting disease activity despite standard immunosuppression. Current drugs available are infliximab, an anti-TNF antibody and etanercept, a soluble p75 TNF receptor. We currently use infliximab 5mg/kg as an IV infusion given on weeks 0, 2, 6 and 10. Infusion is given over 2 hours with monitoring of vital signs (pulse, bp, temp and respiratory rate) every 15 minutes. If patient responds they can continue with monthly infliximab injections long term. Before commencing therapy patients should be assessed for presence of latent tuberculosis by chest x-ray and tuberculin test. Infection is the main adverse event and infusions should not be given if infection considered likely.

**Rituximab** – see SLE

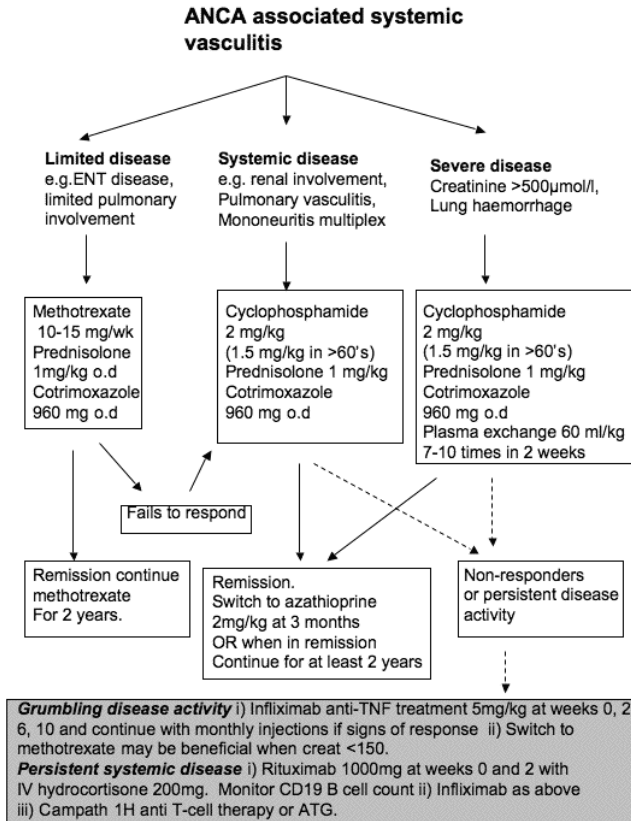
**Anti-T cell antibodies** – reserved for severe disease unresponsive to other treatments. The risks when used after other failed therapies may be very high; discuss with local experts first.

**Campath 1H (alemtuzumab)** (anti-CD52) is a humanised lymphocyte-depleting monoclonal antibody. Peripheral blood lymphocyte numbers return to normal but patients have very prolonged depletion of CD4 cells. It is given as an infusion with a total dose of 40-50mg over 2-5 days.

**Anti-thymocyte globulin (ATG)** is a rabbit polyclonal antibody against activated lymphocytes. This is administered as a 10 day regimen as in renal

transplantation (see detailed protocol there), with discontinuation of other cytotoxics or biological agents.

## Treatment algorithm



## Goodpasture's (anti-GBM) disease

Is mentioned here because of its similarity in presentation and treatment to small vessel vasculitis. Treatment should be cyclophosphamide-based and plasma exchange should be used aggressively – daily for 10-14 days or until antibodies are suppressed. Discuss no active treatment if renal damage is irreversible and there is no lung haemorrhage.

Obtaining research samples before treatment may be extremely valuable – contact ANT/RGP or the research lab.

## THROMBOSIS PROPHYLAXIS

An assessment of the requirement for thromboprophylaxis should be made on all patients at the time of admission.

LOW RISK	MEDIUM RISK	HIGH RISK
Minor illness at any age	Medical Patients with any additional risk factor	Acute illness causing lower limb paralysis
Early mobilisation	s/c enoxaparin 20mg daily	s/c enoxaparin 20mg daily +/- TED stockings

### Additional Patient Risk Factors

Patients with a history of DVT/PE thrombophilia move up one category

Age >40 years	Heart failure
Pregnancy	Recent myocardial infarction
Immobility	Nephrotic syndrome
Malignancy (esp. pelvic, abdo, metastatic)	Inflammatory bowel disease
Severe infection	Polycythaemia
Marked obesity	Certain other conditions: eg Paroxysmal nocturnal haemoglobinuria, Behcet's disease, Homocystinaemia, Paraproteinaemia with hyperviscosity
Paralysis of lower limb(s)	
High dose oestrogens	

### Prescribing notes

Use enoxaparin with great caution (or not at all; or consider unfractionated heparin) in patients with acute renal failure or other condition that is not fully diagnosed or understood. This protocol recommends lower doses in high risk patients with renal impairment because of increased half life and possibly exacerbated effects in patients with renal failure.

Thromboprophylaxis should be stopped 1 full day before renal biopsy (i.e., no dose within 24h).

*Note that the risk of heparin may outweigh the benefits in some moderate and high-risk patients*

### Contraindications to enoxaparin

- Within 12 hours of invasive procedures where there is a danger of significant bleeding complications e.g., epidural/spinal anaesthesia, or surgery
- Active peptic ulceration, recent intracranial haemorrhage or other excessive risk from bleeding
- Coagulopathies and thrombocytopenia
- Severe liver disease

## TRIALS AND STUDIES

We have local Ethical Committee permission to enter patients into the following ongoing trials or studies; but others will be initiated:

### 1. **Causes of Nephritis (local)**

Gives us permission to take samples from patients with any renal disease and from relatives and other normal individuals, for research purposes, in order to study risk factors, aetiology, or prognosis.

Lead investigators: Dr R Phelps, Professor N Turner

### 2. **Immunopathogenesis of Goodpasture's disease (local)**

Investigation of lymphocyte responses to Goodpasture antigen – sampling only BEFORE TREATMENT is initiated if at all possible.

Contacts: Dr R Phelps, Dr L Henderson, or Prof N Turner

### 3. **Immunosuppression for membranous nephropathy (Multicentre: Professor Peter Mathieson, Bristol)**

For patients with deteriorating renal function over at least three months of observation and creatinine <300µmol/l.

Three limbs: chlorambucil/methylprednisolone; cyclosporin; supportive therapy only. Local lead investigator: Professor N Turner

### 4. **Genetics of Glomerulonephritis (Multicentre: Prof Andy Rees, Aberdeen)**

Local contact: Professor N Turner

### 5. **Intervention in renal artery stenosis (ASTRAL: Multicentre)**

Stenting versus medical management alone where the 'best' treatment is uncertain. Local contact: Dr Ian Gillespie (Dept. Radiology)

### 6. **Vasculitis Trials (ECYSVASTRIAL European Multicentre trials)**

Various trials cover most new patients presenting with ANCA + MPA/WG involving the kidneys.

Local contact: Dr D Kluth

### 7. **Lipid lowering in CRF (Multicentre: Dr Colin Baigent, Oxford)-SHARP**

Enrolment complete.

Local contact: Dr C Swainson

### 8. **Endothelin antagonists in CKD**

Role in reducing proteinuria and effect on haemodynamics.

Contacts: Dr N Dhaun and Dr J Goddard

### 9. Trials initiated after August 2006 not listed here.

# URINARY TRACT INFECTION

## Definitions

- Asymptomatic bacteriuria – treat only if patient is pregnant (+/- renal transplant recipient with stent in situ). If in doubt, discuss.
- Cystitis – may have alternate aetiology. Remember that lower urinary tract symptoms in women with 10,000-100,000 cfu/ml probably represents infection and should be treated
- Pyelonephritis – should always be treated, and will frequently require parenteral therapy
- Recurrent UTI -  $\geq$  three symptomatic infections per annum (provided  $\geq$  1 month interval; less suggests relapse). Review oral fluid intake, anatomical or bladder function problems, vaginal epithelium and consider long-term prophylaxis
- LUTS – many patients have symptoms of voiding dysfunction (urge incontinence, stress incontinence, incontinence, nocturia, prostatism). These may benefit from expert urological assessment
- Complicated vs. Uncomplicated – anatomical problems, stones, stents, transplants, pregnancy – should be treated more aggressively.

See the Unit website <http://www.edren.org> for more information – under Education, Undergraduate.

## VEINS AND VASCULAR ACCESS

### Veins

Native vein fistulas are the best permanent access for haemodialysis, and damaged veins make poor fistulas. Therefore, when inserting IV catheters:

- Avoid forearm veins – use hand.
- Do not use arm with working fistula
- Take blood on dialysis where possible (liaise with nurses)
- Preserve veins by limiting venepuncture to 1 arm if possible (preferably dominant arm).

### Fistulas

Fistulas are the gold standard of vascular access. They are end to side vascular anastomoses, usually radiocephalic, brachiocephalic or brachio basilic. They are created by either the vascular or transplant surgeons. May also use synthetic (PTFE/Gortex) grafts which are a conduit between artery and vein.

Remember to update the vascular access screen on Proton after creation.

### When to Organise

- Usually refer for fistula up to 1 year before required.
- Renal Association guidelines state 67% of people should have fistula if seen by nephrologists >4/12 prior to starting dialysis (good practice).

### How to Organise

- Fax (21208)/ email a 'vascular access referral form' to the vascular access coordinator who will either see the patient on the ward (inpatients), on dialysis, or in the nurse led clinic. The coordinator will organize duplex scan of limb vessels, add patient to the appropriate theatre list and organise admission.

### Fistula Creation

- Majority done under local anaesthetic, but some general anaesthetic cases (particularly transpositions).
- Local anaesthetic: Admission to day surgery unit on a non dialysis day.
- General anaesthetic: majority admitted to vascular ward the day before theatre. Ensure adequately dialysed pre-op, with other usual pre-operative assessment.
- Note – stop warfarin 48 hours prior to admission unless specific instructions from surgeons. No heparin, continue with aspirin. Seek advice from co-ordinator/surgeon re combination of aspirin and clopidogrel.
- Antibiotic prophylaxis required: see antimicrobial policy.
- Post operatively – ensure BP adequate. Document pulses and thrill. Restart any stopped drugs, including anticoagulation. Home with prophylactic antibiotics. Instruct patient to contact vascular access coordinator or ward if thrill disappears.

## Time to Use

- Time taken for fistula maturation is variable, average 8 weeks, but up to 6 months.
- Need to examine fistula prior to first use.
- First cannulation should be undertaken by an experienced nurse.

## Complications

- With all complications the vascular access co-ordinator and the surgeon who performed the procedure needs to be informed. Primary failure of fistulas occurs in 9-35% depending on the site. Risk factors for primary failure includes age, raised BMI, female gender, diabetes, peripheral vascular disease or cardiovascular disease.

## Early

Complication	Associations	Action
§	Intravascular volume depletion Hypotension Hypercoagulability Metastatic calcification	Potentially reversible Give fluids D/W surgeon immediately
Bleeding		D/w surgeon immediately
Infection/abscess	Prosthetic grafts/MRSA	Septic screen inc swab Antibiotics-usually flucloxacillin or d/w med micro

## Late

Complication	Associations	Action
Bleeding	Infection	Compression. Urgent vascular referral.
Thrombosis	Intravascular volume depletion Hypotension Hypercoagulability Metastatic calcification	Potentially reversible D/W vascular surgeon
Infection/abscess	Prosthetic grafts/MRSA	Septic screen inc swab Antibiotics-usually

		flucloxacillin or d/w med micro
Stenosis/Poor flow/Developing abnormality/Not maturing	Inadequate dialysis	Inform vascular access co-ordinator, arrange duplex, d/w surgeons
Distal Ischaemia/Steal	Arterial insufficiency or venous HT, large fistulas	Inform vasc access coord/surgeon, arrange duplex, may require closure/revision
Aneurysm	True v's Pseudo	Inform vasc access coord – requires duplex and surgical revision
High output cardiac failure	Coexistent cardiac disease, large hypertrophied high flow fistulas	ECHO. Inform vasc access coord/surgeon – may req banding/revision.

- Duplex scans usually organised by vascular access coordinator but if unavailable then d/w radiologist.

## Tunneled Central Catheters (Permcaths)

Semi-permanent access utilised in the intermediate term. Used whilst awaiting fistula/graft placement or maturation. Also used in those with delayed recovery from ARF or those with no further options for native vascular access.

Remember to update the vascular access screen on Proton for insertion/removal

### When to Organise

- Fistula not created
- Fistula not mature
- Fistula problem meaning it cannot be used
- Prolonged ARF req dialysis with numerous temporary lines.

### How to Organise

Take completed radiology request card to the 'vascular labs' and discuss case with interventional radiologist. Permcaths placed under fluoroscopy.

### Pre Procedure:

- Bloods including FBC/U+E/Clotting/G+S required
- Consent form (completed in radiology)
- Prophylactic antibiotics: see prophylactic antibiotic regimens

### Post Procedure:

Permcath can be used immediately. No need for CXR to check position. Do not use for any purpose other than haemodialysis/CMH

### Complications

Problem	Action
Bleeding/haematoma post insertion	Apply pressure and dressing
Infection	Exit site swab, blood/line cultures. Empirical antibiotics – May req line removal
Blockage/Poor flow	Check line position May require urokinase/line stripping (see below)
Inadvertent bolus of heparin lock	Dialysis with no further heparin. If bleeding d/w Haem SpR

If permcath providing poor blood flows (<150 mls/min) or is blocked then:

1. Flush with 30ml boluses of normal saline. Remember permcaths are locked with 5000u/ml repair which must be removed before finishing.
2. Urokinase/Alteplase/Stripping – see below.

## Temporary Lines

Used in acute renal failure and as a temporary measure in patients with ESRF whose other access is not available (for example, malfunctioning fistula). Do not use for any purpose other than haemodialysis/CMH. Remember temporary lines are 'locked' with 5000u/ml Heparin and this must be removed first.

Inserted using sterile Seldinger technique under USS guidance to minimise complications. Use either double or triple lumen (IV fluid/drug administration). To prevent thrombus formation both lumens of catheter are instilled with heparin (5000u/ml), the amount required is clearly labelled, this limits systemic heparinisation.

### 1. Internal Jugular lines

- R sided easier to insert than L and get higher blood flows.
- 16cm line usually used.
- Allows measurement of CVP if triple lumen used
- Difficult to place in pulmonary oedema
- Complications include carotid artery puncture (minimised with USS) and pneumothorax (less risk R>L)
- Check CXR mandatory

### 2. Femoral Lines

- 19cm line usually used
- Complication rate of insertion lowest (femoral artery perforation minimised with USS, apply compression)
- Easier to place in pulmonary oedema
- Preferable for patients with respiratory disease/distress as avoids possibility of pneumothorax.
- Infection rate higher than other temporary lines.

### 3. Subclavian Lines

- Least preferred route
- Increased risk of stenoses/thromboses with consequent loss of ipsilateral arm for future HD access
- Check CXR mandatory

## Unblocking catheters

INDICATION FOR UROKINASE/ ALTEPLASE – clearing of clotted dual lumen catheters and those giving insufficient blood flow rate (<150ml/min) where flushing with boluses of 30ml saline has been ineffective.

### Protocol for urokinase

Dilute dry-powdered Urokinase, 5000 units/vial, with normal saline. The added volume of saline should be the total volume of both lumens. The volume of each lumen is clearly marked on the end pieces of each. Instill the reconstituted Urokinase into each lumen as a bolus and clamp the line.

After 30-40 min aspirate the Urokinase lock and attempt dialysis. If unsuccessful repeat the above procedure up to a maximum of 3 times. If patency cannot be restored the patient may need a temporary line and catheter-ogram and/or stripping of fibrin sheath arranged with radiology.

### Protocol for Alteplase

Alteplase can replace urokinase for this indication. Using this protocol, very little alteplase reaches the circulation, therefore usual contra-indications (where patients at high risk of haemorrhage) do not apply and side-effects should not occur.

- Instill 1mg/ml solution made up from 10mg vial, using positive pressure technique to fill internal lumen of arterial and venous sides of catheter (approx 2ml)
- After 30 mins aspirate alteplase solution from catheter
- Start haemodialysis immediately if lumen patent
- If catheter still blocked repeat alteplase instillation and aspiration up to two more times
- The solution is stable for up to 24h if kept in a refrigerator (0-4°)
- Monitor for local bleeding

### Catheter stripping

Fibrin sheaths can be removed mechanically from semi-permanent lines. A snare is inserted via another route (usually femoral vein). Discuss with interventional radiologists.

### Anticoagulation

Controlled trial evidence has suggested that anticoagulation for vascular access protection is more likely to cause serious bleeding than to save access. There may be individual circumstances where the balance of risk is different.

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