One sweeps by, attended by an immense train,  
All emblematic of peace — not a soldier or menial  
among them.

One sweeps by, old, with black eyes, and profuse white hair,  
He has the simple magnificence of health and strength,  
His face strikes as with flashes of lightning whoever it turns toward.

Three old men slowly pass, followed by three others,  
and they by three others,  
They are beautiful — the one in the middle of each group holds his companions by the hand,  
As they walk, they give out perfume wherever they walk.
Because patients with chronic kidney disease (CKD) are more likely to die than to reach end-stage renal disease (ESRD), incident ESRD patients—who have survived CKD—are unique. And it should not be surprising that they are affected by an increasing level of comorbidity.

Since diabetes is the leading cause of ESRD, we assessed the total burden of the disease at the initiation of ESRD therapy. As either a primary or secondary cause of ESRD, the occurrence of diabetes in new patients has grown from 49.4 percent in 1995 to 55.7 percent in 2004. At initiation, 27–35 percent of patients have evidence of congestive heart failure, and 16–30 percent have ischemic heart disease; rates of cardiovascular disease are consistently higher in diabetics. And chronic obstructive pulmonary disease in new patients is increasing, particularly in those with diabetes.

We look here also at prescription drug therapy used to treat some of these diseases. Since prescription drug use is highly influenced by the patient’s insurance coverage, we look at patients in employer group health plans who are on dialysis. More patients are using angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs), particularly patients with diabetes, congestive heart failure, or hypertension. Use of beta blockers and diuretics is also growing.

Over the last decade there has been a slow but steady increase in the mean hemoglobin level at initiation of ESRD treatment; the percent of patients receiving erythropoietin treatment prior to initiation has grown as well, but is now relatively stable. As noted in previous ADRs, African-American patients entering treatment have lower hemoglobins, but it appears that the disparity between whites and blacks is narrowing. Interestingly, patients who enter ESRD with secondary glomerulonephritis have hemoglobin levels almost 0.5 g/dl lower than those with ESRD due to primary glomerular diseases, but in spite of these low levels, EPO use before initiation is actually lower in those with the lowest hemoglobins. These data suggest that pre-ESRD anemia management needs improvement based on National Kidney Foundation guidelines.

This year we present biochemical data by body mass index (BMI). As expected, the lowest BUNs and serum creatinine levels occur in patients with the lowest BMIs; this is consistent with malnutrition, low muscle mass, and low protein intake. The combination of a low BMI and a low serum albumin, indicative of both malnutrition and inflammatory load, is associated with higher BUN/creatinine ratios. And the increasing prevalence of obesity in both ESRD patients and the general population is shown by the increasing odds of having a high BMI.

Lower BUN and creatinine levels at the initiation of dialysis also suggest that patients are starting dialysis earlier in their course of progression to ESRD. Using the Modification of Diet in Renal Disease (MDRD) formula, which includes serum creatinine, age, gender, and
race, we look here at estimated glomerular filtration rates (eGFR). At initiation, eGFRs have increased from 7.4 to 10 ml/min/1.73 m² over the last ten years, and similar trends are noted across all racial and ethnic groups. While these estimates may be subject to variation due to differences in racial and ethnic groups (such as the Asian and Hispanic populations, in which the MDRD formula has not been validated) and the lack of standardization in serum creatinine measurements, the increase is clear.

Data also show that the higher the BUN/creatinine ratio, the higher the eGFR; this is consistent with pre-renal azotemia and congestive heart failure, for which the BUN/creatinine ratio has been historically used as an indicator along with volume depletion, liver disease, and nephrotic syndrome.

There appears to be little difference between eGFRs in the diabetic and non-diabetic populations, based on overall cardiovascular disease and single cardiovascular conditions. There are differences, however, based on the number of co-morbid conditions, particularly in non-diabetics.

Individuals with the greatest number of co-morbidities have the highest eGFRs, consistent with the idea that patients are initiating ESRD treatment earlier because of the increasing complexity of their co-morbid conditions, and that congestive heart failure, fluid overload, hyperkalemia, acidosis, and uremic symptoms are indicating the need for therapy.

After adjustments for age, higher eGFRs are more likely in individuals with atherosclerotic heart disease, other cardiac diseases, congestive heart failure, and chronic obstructive pulmonary disease. These trends are similar across racial and ethnic groups.

In conclusion, the incident population is becoming older and more complicated in its co-morbidity, and appears to be starting ESRD therapy with higher glomerular filtration rates—a reflection of both the increasing disease burden and, in those with low BMIs, malnutrition as well. Hemoglobin levels at initiation have improved, but interventions with erythropoietin seem to have peaked in 2002, with only a third of patients now receiving this treatment. Almost 70 percent of patients starting ESRD therapy have hemoglobin levels less than 11 g/dl, the lower end of the target suggested by the National Kidney Foundation’s K/DOQI guidelines.

Although the population appears to be initiating treatment with higher eGFRs, the level of co-morbidity seems to be stabilizing, also consistent with the flattening of incident rates and a decline in certain populations.

### Chapter Highlights

**Figure 3.4** Seven percent of white patients are reported to have cancer at the time of ESRD initiation, compared to 4 percent of blacks and 3 percent of patients of other races. **Figure 3.14** Mean hemoglobin levels among all patients initiating ESRD therapy increased nearly 1 g/dl between May 1995 and May 2004, from 9.22 to 10.15 g/dl. **Figures 3.23–25** Levels of blood urea nitrogen in patients initiating ESRD therapy decreased steadily between 1995 and 2004, falling by 10.6 mg/dl overall and between 10–13 mg/dl depending on BMI index. **Figure 3.34** Since 1995, estimated glomerular filtration rates have risen steadily, adding credibility to the theory that patients are beginning ESRD therapy at an earlier stage of chronic kidney disease.
Since 1995, the year in which information on comorbidity was added to the Medical Evidence form, the number of patients who begin ESRD therapy with a listed diagnosis of congestive heart failure (CHF) has remained relatively stable (Figure 3.2). Among whites, 34–35 percent—and 41 percent of those with diabetes—have CHF at initiation; the disease is identified in 27–28 percent of patients of other races. Racial differences are wider for ischemic heart disease (ISHD), with 30 percent of white patients having the diagnosis at initiation, but only 16 percent of blacks, and the proportion of new patients with ISHD has grown slightly since 1995. Peripheral vascular disease is noted on the Medical Evidence forms of approximately 16 percent of whites, 9 percent of blacks, and 11 percent of patients of other races; in the non-diabetic population it is twice as common in whites. Across diagnoses, diabetic patients are more likely than their non-diabetic counterparts to begin therapy with a listed diagnosis of cardiovascular disease.

Similar patterns by race are seen in the reporting of chronic obstructive pulmonary disease (COPD) (Figure 3.3). Ten percent of white patients initiate ESRD therapy with a listed diagnosis of COPD compared to 5 and 3 percent, respectively, of blacks and patients of other races. The diagnosis occurs at a slightly higher rate in white patients who do not have an accompanying diagnosis of diabetes. With the exception of these latter patients, rates of reported COPD among whites and blacks have increased slightly since 1995.

Seven percent of white patients are reported to have cancer at the time of ESRD initiation, compared to 4 percent of blacks and 3 percent of patients of other races (Figure 3.4). The diagnosis is least common in diabetics, and occurs most often in whites without diabetes—since 2001, 10 percent of these patients have had cancer at the time of initiation. After slight increases in the late 1990s, however, rates do seem to be stabilizing.

In Figures 3.5–7 we examine the same comorbidities, but identify them at a different time and with a different cohort and method, using hospital admissions in the one year following the first 90 days of ESRD therapy. While Figures 3.2–4 identify comorbidities reported at initiation,
The cohort is also restricted to patients who survive and have Medicare as primary payor for the entire first year, to ensure complete claims. These data can be thought of as representing patients actively hospitalized with a diagnosis for the comorbidity, and may capture those who develop the comorbidity during the first year of ESRD therapy.

Data for the year following the first 90 days of ESRD therapy show that hospitalization rates for almost all examined comorbidities (the exception is cancer in diabetics) are consistently higher in white patients. They also identify slightly greater rates of cardiovascular hospitalization in diabetic patients, and lower cancer hospitalization rates in this same population.

While hospitalization rates tend to be higher for whites, growth in the rates since 1991 has generally been similar by race (Figure 3.5). First-year hospitalizations for COPD, for example, are rising, and in 2002 occurred in 16.2 percent of white patients, 11.2 percent of blacks, and 7.4 percent of patients of other races (Figure 3.6).

Fewer patients are hospitalized for cancer in their first year of ESRD treatment, and rates are slightly higher in non-diabetics (Figure 3.7). In 2002, for example, 4.9 percent of non-diabetic white patients were hospitalized for cancer, compared to 2.2 percent of those with diabetes.
In Chapter One we evaluate medication use in Stage 1–4 chronic kidney disease (CKD) patients age 60 and older, using NHANES III and 1999–2002 populations; these analyses look only at medication use within a month of the NHANES survey. In this chapter, in contrast, we assess prescription drug therapy at or just after the initiation of dialysis, using the Medstat Employer Group Health Plan (EGHP) database. This analysis encompasses the cumulative use of various prescriptions in patients age 20–63 during the one year after initiation. A patient receiving one prescription in a particular drug category in the year following initiation is counted in the same way as a patient who receives multiple prescriptions within the same drug category over the year. Each patient, then, provides one count in that drug category.

Prescription of angiotensin-converting enzyme inhibitors (ACE-Is) and angiotensin receptor blockers (ARBs) increased substantially from 2000 to 2003 (Figure 3.8). Sixty percent of all new dialysis patients, and 62, 57, and 64 percent of new dialysis patients with diabetes, congestive heart failure, and hypertension, respectively, received at least one prescription for ACE-Is and ARBs in 2003. This trend may continue in light of the newly released National Kidney Foundation (NKF) Clinical Practice Guidelines on Hypertension and Antihypertensive Agents in Chronic Kidney Disease, which list ACE-Is and ARBs as the preferred agents in CKD Stage 1–4 patients with diabetes, whether or not it is accompanied by hypertension.

The prescription of beta blockers has grown even more than that of ACE-Is/ARBs (Figure 3.9). Similar to the trends seen in patients with CKD of Stages 1–4 (Chapter One), the use of beta blockers rose, between 2000 and 2003, from 36 to 63 percent, 36 to 74 percent, and 31 to 56 percent in new dialysis patients with diabetes, congestive heart failure, and hypertension, respectively. This most likely reflects lower blood pressure goals, and the fact that beta blockers were advocated as first-line therapy for reaching new blood pressure goals by the Joint National Committee on Prevention, Detection, Evaluation and Treatment 6 (JNC 6) guidelines in 1997. In addition, American Heart Association (AHA) and Amer-
American College of Cardiology (ACC) guidelines published in 2001 recommend the use of beta blockers after myocardial infarction and in congestive heart failure, conditions both common in patients with ESRD.

The use of dihydropyridine calcium channel blockers (CCBs: amlodipine, felodipine, isradipine, nicardipine, nifedipine, nimodipine, nisoldipine) has declined slightly overall, but particularly in new dialysis patients with congestive heart failure, in whom the percentage dropped from 46 to 37 (Figure 3.10). These trends are in line with the most recent AHA and ACC guidelines (2001), which discourage the use of any CCB in patients with CHF. Conversely, the use of nondihydropyridine CCBs (diltiazem and verapamil) has increased slightly, for reasons which are unclear. These drugs may also be used as antiarrhythmic agents, and perhaps are being used to prevent sudden death in some patients.

The use of loop diuretics (bumetanide, furosemide, and torsemide) in incident dialysis patients remained stable between 2000 and 2003, except in hypertensive patients, in whom the percent prescribed these medications grew from 21 to 29 (Figure 3.11). The same trend is seen with the use of thiazide diuretics, although their use is much less common in new ESRD patients. This trend is probably reflective of JNC 6 and JNC 7 guidelines that advocate diuretics (particularly thiazide diuretics) as first-line antihypertensive agents in the general population. Loop diuretics become the predominant diuretic as the glomerular filtration rate declines and thiazide diuretics lose effectiveness, except when used in combination with loop diuretics.

The use of lipid-lowering agents has increased in new patients receiving dialysis (Figure 3.12). Statin use has grown from 35 to 39 percent in all incident dialysis patients, but rose from 37 to 44 percent and from 37 to 41 percent in patients with diabetes and cardiovascular disease, respectively. The negative results from the 4D study, conducted in Germany and evaluating atorvastatin use in hemodialysis patients with Type 2 diabetes, may dampen enthusiasm for statin use in ESRD patients. There has not, however, been a prospective study conducted in patients with Stage 3–4 CKD to determine whether early and continued use of statins into ESRD is beneficial in these patients. Until further research is available, it seems reasonable to continue statin use when indicated as patients transition from CKD Stage 4 to Stage 5. Non-statin use (e.g., bile acid sequestrants, fibrate acid derivatives, and niacin) has increased as well, although the use of these agents is quite low compared to that of statins (Figure 3.12). Overall, only 11 percent of incident EGHP dialysis patients received a non-statin in 2003.

The occurrence of diabetes as the primary or secondary cause of ESRD in new dialysis patients grew from 49.4 percent in 1995 to 55.7 percent in 2004. The cumulative percent of new dialysis patients with diabetes receiving insulin and thiazolidinediones (TZDs: pioglitazone, rosiglitazone, and troglitazone, which was taken off the market in 2000) also increased from 2000 to 2003, perhaps reflecting patients with more severe diabetic disease or receiving more aggressive treatment (Figure 3.13). Sixty-four percent of diabetic patients received insulin therapy in 2003, up from 56 percent in 2000. The use of TZDs rose from 10 to 14 percent, which is similar to the percent of CKD patients who received TZDs in the NHANES 1999–2002 cohort (see Chapter One). Metformin use occurred in 0.6 percent of patients in 2003, demonstrating the continued need to educate health care professionals that metformin is contraindicated in patients with serum creatinine values exceeding the normal level.

(Figures 3.8–13) incident EGHP dialysis patients in the Medstat database, age 20–65.
Mean hemoglobin levels among all patients initiating ESRD therapy increased nearly 1 g/dl between May 1995 and May 2004, from 9.22 to 10.15 g/dl (Figure 3.14). The increase has been slightly higher among patients on peritoneal dialysis. Hemoglobin levels remain lowest in patients who initiate on hemodialysis—not surprising, as the sickest patients are usually started on this modality—while they are highest in those who receive a preemptive transplant.

In 2003 and 2004, the mean hemoglobin level among patients receiving EPO at initiation was up to 0.45 g/dl greater than that of patients not on the therapy (Figure 3.15). Since 2002, nearly one in three new patients is receiving EPO therapy at initiation; this number has, however, remained relatively stable—a cause for concern—as has the mean hemoglobin level for EPO-treated patients.
For patients initiating ESRD therapy, the National Kidney Foundation’s Kidney Disease Outcomes Quality Initiative (K/DOQI) sets a target hemoglobin of 11 g/dl or higher. At least 30 percent of new patients now meet this target, almost double the level seen in 1995 (Figure 3.16). Nearly half of new patients, however, still begin therapy with a hemoglobin less than 10 g/dl.

Since 1998, mean hemoglobin levels at the initiation of therapy have risen 0.5 g/dl for both male and female patients, and levels continue to rise steadily with age (Figure 3.17). The youngest patients continue, however, to have the lowest hemoglobin levels at initiation. Nearly 40 percent of pediatric patients—both boys and girls—are receiving EPO at the start of ESRD, up from 36 and 33 percent, respectively, in 1998. During the five-year period EPO use overall increased 7–8 percentage points.

Initial mean hemoglobin levels are highest in white patients and lowest in blacks, at 10.1 and 9.8 g/dl (Figure 3.18). The increase between 1998 and 2003 ranged from 0.42 g/dl in white males to 0.58–0.6 g/dl in black and Native American patients of both genders. EPO use prior to the start of therapy continues to be most common in Asian patients, at 34 and 37 percent of men and women, respectively. Use of this treatment has, however, grown dramatically in the Native American population—from only 4 and 8 percent of male and female patients in 1998 to 29 and 35 percent in 2003. EPO therapy is now given least often in black and Hispanic males.

Figures 3.19–22 present data on anemia and EPO therapy in patients whose ESRD is caused by one of three major diagnoses. Mean hemoglobin levels at initiation are greatest in patients with primary glomerulonephritis, at 10.1 g/dl in 2004, and lowest for those with secondary GN, at 9.7 (Figure 3.19). Patients with primary GN have also seen the greatest increase in hemoglobin levels—0.9 g/dl since 1995, compared to 0.7 g/dl among patients with secondary GN or cancer. Patients with primary GN are somewhat more likely to receive EPO prior to ESRD—36 percent of patients in 2004, compared to 34 percent of those with cancer and 32 percent of those with secondary GN.

Since 2003, an average of 31 percent of patients with primary GN have begun therapy with a hemoglobin that meets the K/DOQI target of 11 g/dl or above (Figure 3.20). Among those whose ESRD is caused by secondary GN or cancer, however, only 21–23 percent meet this target; 38 and 32 percent percent of these patients, respectively, start treatment with a hemoglobin less than 9 g/dl (Figures 3.21–22).

(Figures 3.14–22) incident ESRD patients with a first service date between May 1995 & June 2004; data from Medical Evidence form.
Levels of blood urea nitrogen (BUN) in patients initiating ESRD therapy are shown in Figures 3.23–25. These levels decreased steadily between 1995 and 2004, falling by 10.6 mg/dl overall and between 10–13 mg/dl depending on BMI index. Asians exhibit the highest levels of BUN at initiation, while the lowest levels are found in blacks and Native Americans. No consistent patterns are evident when comparing BMI index and BUN level. In whites and Asians, however, there are definite upward trends in BUN level as BMI increases.

Serum creatinine levels at initiation, shown in Figures 3.26–28, exhibit downward patterns similar to those of BUN levels. Overall creatinine levels in 2004 were 1.8 mg/dl lower than those found in 1995. Blacks have noticeably higher creatinine levels at initiation compared to patients of other racial and ethnic groups, while the lowest levels exist in the white population. Except in Asians, BMI index does not appear to influence serum creatinine levels, and in most instances the highest creatinines appear to be associated with mid-range BMI indices.

Figures 3.29–31 show trends in the BUN/creatinine ratio at initiation. This marker can be used to predict the existence of catabolic states in patients. Since 1995, there has been a steady rise in the BUN/creatinine ratio for all ranges of BMI. In 2004, the highest ratios were associated with whites and the lowest with blacks, with a difference of 3.5 mg/g between the two groups. The influence of BMI on the BUN/creatinine ratio appears only in whites, where there exists a noticeable rise in ratios with each increase of BMI.

It appears that neither albumin nor BMI has a direct impact on BUN/creatinine ratios (Figures 3.32–33). Between 1995 and 2004, for example, ratios rose steadily in both nutrition-compromised and normal patients of all BMIs. Levels of increase are 0.9–2.7 and 1.6–2.4 mg/g in patients below and above the albumin test’s lower limit, respectively.

The odds of having a BMI of 30 kg/m² or above—the definition of obesity—have increased considerably (Table 3.1). Among incident Hispanic patients, for example, the odds in 2002–2004 are 55 percent greater than in 1996–1998; for whites they
are 82 percent higher, and for blacks who have doubled. Among Asians, women are only 20 percent more likely than men to be obese; among blacks, in contrast, the odds for women are 78 percent greater.
Since 1995, estimated glomerular filtration rates (eGFR) have risen steadily, adding credence to the theory that patients are beginning ESRD therapy at an earlier stage of chronic kidney disease. In 2004, eGFRs in all patients increased 2.5 ml/min/1.73 m2 over 1995 levels, and ranged from 2.5–2.9 depending on BMI (Figure 3.34). Differences in rates of increase were consistent for all BMI categories. The higher eGFRs found in patients with BMIs lower than 18.5 may be secondary to low muscle mass caused by severe protein malnutrition.

Rates of increase in eGFR also show consistent differences between racial and ethnic groups (Figure 3.35). Whites continue to have the highest eGFRs and Asians the lowest, with rates in 2004 being 10.3 and 9.1 ml/min/1.73 m2, respectively.

Except in Asians, no discernable patterns emerge when assessing the impact of BMI on eGFR. In 2003, rates of eGFR in the Asian population show a distinct decrease as the BMI index increases.

Figures 3.37–39 depict eGFR levels in patients with low albumins. Increases over time by BMI index and race/ethnicity are similar to those found in all patients.

BMI, BUN/creatinine ratio, and albumin as predictors of eGFR are illustrated in Figures 3.40–41. Regression analyses and an $r^2$ of 0.0016 show that the BMI index is not a good predictor of eGFR. Predictability is somewhat improved using the BUN/creatinine ratio, with an $r^2$ of 0.30.

Estimated glomerular filtration rates based on cardiac comorbidity and number of comorbidities increased in a similar fashion between 1995 and 2003 in both diabetic and non-diabetic patients (Figures 3.42–43). Diabetic status, rather than type of cardiac comorbidity, seems to influence eGFR to a greater degree, with eGFRs on average 6–7 percent higher in diabetics than in non-diabetics. The number of comorbidities also appears to be associated with eGFR. Patients with 4–6 listed comorbidities at initiation have the highest eGFRs, regardless of diabetic status, suggesting that these patients may be entering ESRD treatment at an earlier stage of chronic kidney disease. Differences in eGFR by the number of comorbidities are more apparent in non-diabetics; compared to those of patients with only one listed condition, for example, rates were 31 percent (2.6 ml/min/1.73 m2) higher in patients with 4–6 comorbidities.

Table 3.3 shows the odds ratio of having an eGFR at initiation higher than the gender-specific mean. Compared to those of the 1996–1998 period, the odds have increased 53 percent for Hispanic patients, and 81 percent for white and Asian patients. The odds are 20–29 percent lower in women than in men and, by age, are lowest in patients age 45–64.
PATIENT CHARACTERISTICS

2005 Annual Data Report

3.40 BMI (kg/m²) & albumin as predictors of eGFR

3.41 BUN/creatinine ratio as a predictor of eGFR

3.42 Estimated GFR, by diabetic status & cardiac morbidity

3.43 Estimated GFR, by diabetic status & number of comorbidities

3.44 Odds ratio of having an eGFR greater than the population’s gender-specific mean at initiation

[Figures 3.34–43] incident ESRD patients with a first service date between May 1995 & June 2004. The lower limit of albumins measured by brom cresol purple is 3.2 g/dl, & by brom cresol green is 3.5 g/dl. Figure 3.37 includes all comorbidities listed on the Medical Evidence form. (Table 3.b) incident ESRD patients with a first service date between January 1996 & July 2004. – – – (Figures 3.37–39 & Table 3.b) eGFR calculation for ages 0–18 from Schwartz et al., & for ages 19 & above from Levey et al.
**Figure 3.4** Seven percent of white patients are reported to have cancer at the time of ESRD initiation, compared to 4 percent of blacks and 3 percent of patients of other races. **Figure 3.5** While hospitalization rates tend to be higher for whites, growth in the rates since 1991 has generally been similar by race.

**Figures 3.8–9** The percent of incident dialysis patients using ACE inhibitors and ARBs increased from 35.4 in 2000 to 59.6 in 2003, while the percent using beta blockers grew from 35.3 to 57.

**Figure 3.14** Mean hemoglobin levels among all patients initiating ESRD therapy increased nearly 1 g/dl between May 1995 and May 2004, from 9.22 to 10.15 g/dl. **Figure 3.17** Since 1998, mean hemoglobin levels at the initiation of therapy have risen 0.5 g/dl for both male and female patients, and levels continue to rise steadily with age. **Figure 3.18** Initial mean hemoglobin levels are highest in white patients and lowest in blacks, at 10.1 and 9.8 g/dl.

**Comorbidity & diabetic status**

**Figures 3.21–25** Levels of blood urea nitrogen (BUN) in patients initiating ESRD therapy decreased steadily between 1995 and 2004, falling by 10.6 mg/dl overall and between 10–13 mg/dl depending on BMI index. **Figures 3.26–28** Serum creatinine levels at initiation exhibit downward patterns similar to those of BUN levels. **Figures 3.29–31** Since 1995, there has been a steady rise in the BUN/creatinine ratio for all ranges of BMI. **Table 3.a** The odds of having a BMI of 30 kg/m² or above have increased considerably.

**Prescription drug therapy at or near initiation**

**Figure 3.34** Since 1995, estimated glomerular filtration rates (eGFR) have risen steadily, adding credibility to the theory that patients are beginning ESRD therapy at an earlier stage of chronic kidney disease. In 2004, eGFRs in all patients increased 2.5 ml/min/1.73 m² over 1995 levels, and ranged from 2.5–2.9 depending on BMI. **Figure 3.35** Whites continue to have the highest eGFRs and Asians the lowest, with rates in 2004 being 10.3 and 9.1 ml/min/1.73 m², respectively. **Figure 3.43** Patients with 4–6 listed comorbidities at initiation have the highest eGFRs, regardless of diabetic status, suggesting that these patients may be entering ESRD treatment at an earlier stage of chronic kidney disease.

**Biochemical characteristics & BMI**

**EPO use & anemia at initiation**

**Figure 3.34** Since 1995, estimated glomerular filtration rates (eGFR) have risen steadily, adding credibility to the theory that patients are beginning ESRD therapy at an earlier stage of chronic kidney disease. In 2004, eGFRs in all patients increased 2.5 ml/min/1.73 m² over 1995 levels, and ranged from 2.5–2.9 depending on BMI. **Figure 3.35** Whites continue to have the highest eGFRs and Asians the lowest, with rates in 2004 being 10.3 and 9.1 ml/min/1.73 m², respectively. **Figure 3.43** Patients with 4–6 listed comorbidities at initiation have the highest eGFRs, regardless of diabetic status, suggesting that these patients may be entering ESRD treatment at an earlier stage of chronic kidney disease.