Glycemic control was classified into two
20 MAR test. The mean macular thickness
Snellen chart test and 1.26 in the Log-
fuse associated form to cystoid form
edema (DME). More than half of those
had diabetic retinopathy (DR) and one-
investigators wrote.

of the macula.
within one disc diameter of the center
larger) in size, any part of which was

nonproliferative, moderate proliferative,
The results were then classified as mild
macular edema by improved con-
prevention of diabetic macular ede-
American Diabetes Association categories (3.35 mmol/L or high-

research, no lipid parameters were asso-
ciated with the progression of diabetic retinopathy or with proliferative diabetic retinopathy after adjustment for gly-
cated hemoglobin and other risk factors, the investigators explained.

The presence of macroangiopathy. For this, one or more of the following had to be present: symptoms of angina pectoris, history of myocardial infarction, coro-
ary artery bypass grafting, percutaneous transluminal coronary angioplasty, symp-
toms of or operation for intermittent claudication, history of amputation, tran-
sient ischemic attack, and stroke.

The authors maintained that this relation-
ship between macronodopathy and DME "may be explained, in part, by the increased incidence of macular edema with increased levels of lipids, which was strongly associated with the development of macroangiopathies in previous studies (Br. J. Ophthalmol. 2002;86:84-90; Ophthalmology 2002;109:1225-34)."

The presence of arteriolar hypertension,
dimmers of sypholic and clavationary, and director of the hypertension unit at the University of Chicago. "These data suggest that at similar levels of blood pressure control, the longer-acting, higher-binding telmisartan may confer relatively greater protection against the development of ESRD (end-stage renal disease), though that hypothesis must be tested prospectively." Dr. Bakris and associates randomized 860 patients with type 2 diabetes mellitus, hypertension (defined as blood pressure greater than 130/80 mm Hg), and overt nephropathy to either telmisartan 40 mg or losartan 50 mg for 2 weeks, and then titr-
ed to 80 mg and 100 mg, respectively. If blood pressure was not controlled, con-
comitant antihypertensives were allowed, except ARBs, angiotensin-converting en-
yzme inhibitors, and direct vasodilators.

At admission, the average systolic/dia-
tolic blood pressure was 143/80 mm Hg in both groups; mean urinary protein:creati-
in ratio was 1.971 mg/gCr in the telmis-
artan group vs. 2.010 mg/gCr in the losar-
tan group, and the mean serum creatinine was 1.54 mg/dL in the telmisartan group vs. 1.53 mg/dL in the losartan group. In all, 827 patients were available for analysis. After 1 year of treatment, the mean change in morning spot urinary pro-
in:creatinine—the study’s primary end point—was 0.71 for telmisartan and 0.80 for losartan. This translated to a 29% re-
duction from baseline for telmisartan and a 20% reduction for losartan. Systolic and diastolic BP reductions were not signifi-
cient between groups (−4.8/−3.2 mm Hg vs. −2.7/−2.9 mm Hg, respectively).

Among secondary end points, telmisar-
tan produced superior reductions in urinary albumin:creatinine, and prolonged the time to first cardiovascular event. There were no significant differences between the drugs in urinary sodium:creatinine, glomerular fil-
ltration rate, serum aldosterone, or high-
sensitivity C-reactive protein. Adverse events were not different between groups.

Dr. Bakris disclosed that he is a consul-
tant and speaker for Boehringer Ingelheim, which sponsored the study, and he has re-
ceived research support from the firm.