features of lupus glomerulonephritis with azathioprine-prednisone therapy. In addition, following suppression of the active proliferative lesion a new finding of membranous transformation was found.

Split products of fibrin in renal disease. ROSEMARY ANANIA and GEORGE A. RICHARD. *Univ. Fla. Coll. Med., Gainesville, Fla.* (Intr. by G. L. Schiebler).

Split Products of Fibrin (SPF) were determined by the tanned red-cell hemagglutination inhibition assay in 331 patients with various types of renal disease. The study included 77 sex-age matched controls. The mean value for SPF in the control group was 4.55 ± 2.46 S. D.

SPF were elevated in the Hemolytic-Uremic Syndrome (1/1), Lupus Erythematosis (SLE) (8/10), Steroid Resistant Nephrosis (SRN) (7/13), Acute Glomerulonephritis (AGN) (13/26), Steroid Dependent Nephrosis (SDN) (8/32), Renal Vein Thrombosis (3/3), Hereditary Nephritis (4/15), Active Anaphylactoid Purpura (2/3), and Hypocomplementaremic Nephritis (1/3).

To a lesser degree, the SPF were elevated in Steroid Sensitive Nephrosis (1/14), Idiopathic Hematuria (3/23), Post Acute Anaphylactoid Purpura (1/14), Post Acute Glomerulonephritis (2/20), Acute Urinary Tract Infection (2/18), and Post Urinary Tract Infection (2/81).

No elevation in the SPF was noted in Orthostatic Proteinuria (16), Idiopathic-Hypertension (15), Single Kidney (15), Hydronephrosis (9), and Chronic Glomerulonephritis without Azotemia (14).

A depressed Beta 1 C/Beta 1 A was closely correlated with the SPF in SLE (9/10) and AGN (24/26). Positive immunofluorescent staining of renal biopsy tissue for at least one immunoglobin was correlated with an elevated SPF in SRN (6), SLE (5), AGN (5), and SDN (3). Immunoflourescent stains for fibrinogen were seldom positive. These data indicate that SPF are not diagnostic, and may be elevated in several types of renal disease.

Antigen identification in Goodpasture's syndrome. RAWLE M. MCINTOSH and WILLIAM R. GRISWOLD (Intr. by Fred G. Smith). *UCLA, Los Angeles, Calif.*

Although there is unquestionable evidence that an anti-basement membrane antibody is involved in the development of the renal lesion in Goodpasture's syndrome the specific antigen has not been clearly identified. In an attempt to clarify this problem serum and antibody eluted from a patient with Goodpasture's syndrome was studied by fixation to normal human glomerular basement membrane (GBM) before and after treatment with collagenase, neuraminidase and 8 M urea. Controls for buffer, pH and temperature were employed. Absorption studies and spleen cell and lymphocyte transformation studies using GBM glycoproteins prepared by method of Kefalides were also performed.

Double layer immunofluorescent studies demonstrated that both serum and eluate fixed to the GBM of untreated normal human kidney and normal human kidney treated with 8 M urea, and neuraminidase. However they did not fix to collagenase treated kidney. This suggests that the collagen like glycoprotein rather than the non-collagenous or sialic acid rich glycoprotein is the antigen which induces autoimmune nephritis in Goodpasture's syndrome. Although cell transformation studies were inconclusive this finding was confirmed by absorption studies.

Nature of kidney-bound antibody in the renal disease of bacterial endocarditis. ROBERT L. LEVY and RICHARD HONG. *Univ. of Wisconsin Med. Ctr., Madison, Wis.*

It is thought that the renal disease which sometimes accompanies bacterial endocarditis is due to antigen-antibody complex mechanisms because of the demonstration of immunoglobulin and complement in histologic specimens; however, the specificity of the antibody is not known. We have attempted to further define the pathogenetic mechanism by elution of kidney-bound protein and investigation of its properties. A patient with no previous renal disease who died of bacterial endocarditis and renal failure was studied. He presented with bacterial endocarditis accompanied by elevations of serum immunoglobulins and ultimately developed a monoclonal IgM. Light microscope examination of the kidney revealed segmental nephritis; fluorescent staining showed deposits of the 3 major classes of immunoglobulins and complement along the glomerular basement membrane (GBM) in a "lumpy-bumpy" distribution. Protein was eluted from the *post-mortem* kidney and radiolabeled with I^{125}. Radioautographic studies showed the presence of IgG and IgA. The eluted antibody was shown to selectively combine with bacteria (formalin fixed) cultured from the patient *ante-mortem*. There was also evidence for anti-GBM antibody activity of the eluate. The eluted antibody was not anti-antibody (i.e. did not selectively combine with IgG coated human red blood cells). The demonstration in the kidney eluate of antibacterial antibody specific for the organism cultured from the patient strongly supports the concept of antigen-antibody complex disease in the pathogenesis of the nephritis which accompanies SBE.

Tamm-Horsfall glycoproteinuria: An early index of human renal allograft rejection. ROBERT H. SCHWARTZ, ALLYN G. MAY, ERIC A. SCHENK, RICHARD B. FREEMAN, MICHAEL F. BRYSON, and JAN VAN ESS. *Univ. of Rochester Sch. of Med., Rochester, N. Y.* (Intr. by Douglas Johnstone).

Urinary glycoprotein of Tamm and Horsfall (T & H) is a renal substance found in the ascending limb of the loop of Henle and the distal convoluted tubule. Excretion can be measured by 0.58 M NaCl precipitation. Normal excretion = (1.7–2.1 mg/hr/1.73 sq. m. body surface area). Increased excretion occurs with dichromate-induced renal tubular damage in rats. This observation suggested that monitoring T & H excretion might provide an early index of kidney damage in human allograft rejection. Eight patients (4 males, 4 females. Age 9–49 years) were studied during the first 37–120 days post-transplantation. Seven rejection episodes in 5 patients were diagnosed clinically. In each instance T & H excretion exceeded 25 mg/24 hours (½ upper limit of normal adult excretion = normal excretion of one kidney) prior to the onset of clinical rejection. The interval between onset of increased excretion and clinical rejection was between 4 and 14 days (mean = 9 days). Peak excretion rates up to 110 mg/24 hours occurred. High excretion rates in chronic rejection (1 patient) and in glomerulonephritis of the transplanted kidney (1 patient) were also observed. In these instances T & H was primarily in the form of insoluble urinary casts. Since anti-rejection measures are likely to be more effective when the diagnosis of a rejection episode is early, T & H measurement has a practical clinical value.

The "swan-neck" lesion in childhood cystinosis. CHARLES P. MAHONEY, GARY E. STRIKER, and GEORGE H. FETTERMAN. *Univ. Wash. Med. Sch., Seattle; Children's Hosp., Pittsburgh, Pa.* (Intr. by Robert P. Igo).