mate of the so-called slip correction, which arises from the fact that molecules of a gas flowing through a capillary tube do not have zero tangential velocity at the walls.

We here report data on the viscosity of argon at high temperatures. [Values for the viscosity of argon below room temperature have been reported (*5*).] The capillary-flow technique was used with provision for varying the mean pressure of the gas in the capillary from 10 to 100 cm of mercury. In this pressure range the correction for slip is of the form (*6*)

$$\eta = \eta_0 (1 + 4\kappa\lambda/r) \quad (1)$$

where η is the corrected viscosity, η_0 is the uncorrected viscosity, λ is the mean free path, r is the radius of the capillary tube, and κ is a constant. Thus the slip correction depends inversely on pressure. Therefore, extrapolation of the apparent viscosities of the gas to the limit of pressure $1/p \rightarrow 0$ permits the effects of slip to be eliminated from the calculation without the introduction of theoretically estimated correction factors. The latter are unreliable, especially at high temperatures, as it is not possible to gauge the extent to which the collisions of molecules with the wall of the capillary are specular (that is, the angle of incidence is equal to the angle of reflection). A number of different helical capillaries made of platinum, platinum-rhodium alloy, and silica were used. These were placed in furnaces which were regulated to an accuracy of ± 1°K. Apart from the slip effect, the results were corrected for curved pipe flow (*6*) and gas imperfection. The viscosity of argon from 300° to 1600°K was determined relative to the viscosity of argon at 293°K with a precision of about 0.3 percent. These data were fitted, by the method of least squares, to give the equation

$$\log [\eta(T)/\eta(293)] = 0.63842 \log T - 6.9365/T - 3374.72/T^2 - 1.51196 \quad (2)$$

Data calculated from this equation are given in Table 1. The ratio of the viscosity of argon to that of nitrogen over this temperature range was measured with a precision of better than 0.2 percent.

The results for argon are compared with those of other workers in Fig. 1. The widely accepted results of Trautz and Zink (*7*) and Vasilesco (*8*), though mutually consistent, are too low at temperatures above 500°K (*9*). The discrepancy between these workers and the results reported here reaches 6 percent at 1500°K. Only part of this discrepancy would appear to be attributable to errors in their slip corrections. Our data lie about 0.5 to 1 percent below those of Kestin and Whitelaw (*10*) and DiPippo (*11*) from 300° to 600°K and are about 2 percent below the results of Guevara *et al.* (*12*) from 1100° to 1500°K. However, because these results (*11, 12*) are subject to variations of ± 1 percent, we are unable to confirm that this difference is significant, particularly since the accuracy of our results is probably about ± 1 percent.

Table 1. Viscosity of argon. Smoothed values of viscosity are derived from Eq. 2 based on the standard η (293°K) = 222.8 × 10^{-6} poise. The original measurements are illustrated in Fig. 1.

Temperature (°K)	Viscosity ($\eta \times 10^6$ poise)
(293	222.8)
300	227.4
500	340.1
700	432.0
900	513.0
1100	586.9
1300	655.6
1500	720.3

The viscosities calculated from thermal conductivity data (*13*) (Fig. 1) are in broad agreement with our data. The new data are more consistent with intermolecular potential functions calculated with measurements based on molecular beam experiments and second virial coefficients.

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Antigenic Streptococcal Components in Acute Glomerulonephritis

Abstract. *Fluorescein-labeled immunoglobulin G fractions from serums of patients with acute glomerulonephritis and from many normal serums stained the glomerular basement membrane and mesangium of renal tissue from patients with early acute glomerulonephritis; these serums did not stain the corresponding tissues from patients with any other kidney disease. Previous absorption of the serum fraction with frozen and thawed nephritogenic beta hemolytic streptococci abolished all staining. Other bacteria studied did not abolish the staining. Only the plasma membrane of the streptococcus absorbed the immunoglobulin G fraction; such absorption eliminated staining. Fluorescein-labeled antiserums against streptococcal plasma membrane had staining properties similar to patients' serums.*

The fact that immunoglobulin (IgG) and complement (β_{1c}-β_{1a}) labeled with fluorescein isothiocyanate become localized on the glomeruli of patients with acute glomerulonephritis suggests a complement-consuming antigen-antibody reaction (*1, 2*). A causative relation between β-hemolytic streptococcal infections and acute glomerulonephritis can be established in most instances, but the presence of streptococcal components as antigens and their specific antibodies on the glomeruli have not been demonstrated consistently (*2, 3*).

We studied 16 children with acute poststreptococcal glomerulonephritis, as manifested by hematuria, proteinuria, low serum complement activity, and elevated antistreptolysin O titers. Renal tissue was obtained by biopsy within 3 days after clinical onset of the disease. As judged by light microscopy, such tissue had the typical glomerular changes. The glomerular basement membrane and, to a moderate extent, the mesangium stained in a beaded fashion characteristic of the early phase of the disease when antiserums against human IgG and complement (β_{1c}) were used. Serums from these patients and from normal individuals were obtained; IgG fractions were prepared (*4*) and labeled with fluorescein isothiocyanate (*5*).

The labeled IgG fractions from patients with acute nephritis stained, in a segmental pattern, the glomerular basement membrane and the mesangium

obtained at biopsy of those patients and of all other patients with early acute glomerulonephritis (Fig. 1). Serums taken during the first few days after onset stained material from biopsy obtained early in the disease less intensely, compared to serums obtained 2 weeks later. Blocking tests with the patient's unlabeled γ-globulin eliminated the fluorescent staining. The labeled γ-globulin fractions from patients with acute glomerulonephritis did not stain normal renal tissue or that from patients with subacute glomerulonephritis, chronic glomerulonephritis, systemic lupus erythematosus, pure nephrosis, diabetic nephropathy, discoid lupus, and pyelonephritis.

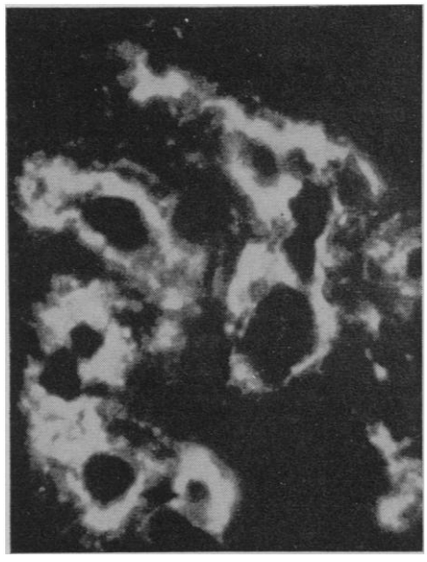

Fig. 1. Part of glomerulus from a patient with acute poststreptococcal glomerulonephritis stained with his own fluorescein-labeled IgG. Day 4 of clinical disease. Oil immersion (× 750).

Fig. 2. Part of glomerulus from the same patient as Fig. 1; stained with fluorescein-labeled antiserum to streptococcal plasma membrane. Oil immersion (× 750).

Tissues taken from two patients 3 months after onset of disease continued to stain with antiserums to human IgG and complement; however, they no longer stained with the patient's own labeled IgG or with any of the other serums from patients with acute nephritis. Serums obtained at time of this later biopsy, however, continued to stain renal tissues of patients in the early phase of the acute glomerulonephritis.

Renal tissue of patients in the early stage of acute glomerulonephritis was stained positively, but less intensely, by labeled IgG fractions from 11 out of 15 serums obtained from "normal" adults.

To investigate the nature of the factor combining with the IgG component —probably a circulating antibody—we used various bacteria for absorption of the labeled serums. When the labeled serums were absorbed with uniform amounts (5 mg) of sediments from repeatedly frozen and thawed *Streptococcus mitis, Streptococcus fecalis, Staphylococcus aureus, Staphylococcus epidermidis,* and *Escherichia coli,* their staining capacity was not abolished. However, when the serums were absorbed by three identically treated nephritogenic strains of β-hemolytic streptococci (Lancefield group A) the staining was markedly reduced or completely abolished.

The nephritogenic streptococci were fractionated into bacterial wall (*6*), M protein (*7*), and plasma membrane (*8*). The fluorescein-labeled serums of the nephritic patients and certain normal individuals first absorbed with the plasma membrane did not stain. Previous absorption of the serums with streptococcal cell wall and M protein reduced the staining capacity slightly, but all our preparations still contained some plasma membrane constituents. Antiserums against β-hemolytic streptococcal plasma membrane were produced in the rabbit and labeled with fluorescein isothiocyanate. These labeled serums stained the glomeruli of patients with early acute glomerulonephritis just as they stained the serums from patients with glomerulonephritis (Fig. 2).

Our results indicate that streptococcal components and, more specifically, plasma membrane constituents are present in the glomeruli of patients with acute poststreptococcal glomerulonephritis, but apparently these can be detected only during the early phase of the disease when not all antigenic sites are fully saturated and when only insufficient amounts of antibody are available. The possibility that antigen is removed after the initial insult appears improbable because antibody and complement that are bound in the original process can still be demonstrated after antigen can no longer be stained. The constituent in the IgG fraction is directed against a specific component of β-hemolytic streptococci, as the absorption studies suggest. All patients with acute glomerulonephritis examined so far seem to share the same antibody, and many normal individuals also have it. The presence of the antibody in "normal" individuals may be due to previous experience of these adults with the specific streptococcus resulting in a long-lasting antibody level.

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Cerumen Types in Choctaw Indians

Abstract. *Cerumen from 432 Choctaw Indians from Mississippi was classified as sticky or dry. The frequency of the autosomal recessive dry type was intermediate between that for Indians of western North America and that for Mayan groups of southern Mexico.*

Human ear cerumen, which occurs in two distinct forms, wet and dry, is under the control of a single gene pair in which the allele for the wet trait is dominant over that for the dry (*1*). Thus cerumen types are inherited as a simple Mendelian trait. Homozygous