SPECIAL ARTICLE

Ocular Inflammatory Disease in the New Millennium

Annabelle A. Okada, MD; John V. Forrester, MD

As we enter the year 2000, we are asked to consider what advances may occur in the field of ocular inflammatory disease in the next 50 to 100 years. This is a daunting and probably unwise endeavor, but an extremely interesting one.

The diagnosis and treatment of ocular inflammatory disease began as a scientific discipline in the 17th century, with the recognition of infectious diseases, such as gonorrhea, that can affect the eye.1 Thereafter, for the better part of the next 2 centuries, most all ocular inflammatory diseases were considered infectious in nature, with syphilis and tuberculosis believed to be the primary culprits. As such, demonstration of the efficacy of antibiotics in treating such infections was a major medical milestone of the 20th century. However, not all cases of ocular inflammation responded to antibiotics, and theories began to appear suggesting that “autoimmunity” may also be playing a role in some forms of ocular inflammation. For example, experiments in as early as the first decade of this century showed that lens antigens could induce anaphylaxis in guinea pigs,2 and that ocular tissues injected into rabbits and guinea pigs could cause endophthalmitis.3

Accordingly, current practice emphasizes the importance of differentiating between infectious and noninfectious intraocular inflammation.4 However, in many cases this is difficult and since the exact etiology or pathogenesis of these immune-