Usually the premonitory symptoms precede an acute attack and are as follows: rapid impairment of accommodation, with increasing presbyopia; rainbow-like rings around the gas or candle flame, a cloudy condition of sight which the patient will liken to looking through a fog or mist and momentary loss of vision. Should an acute attack now supervene we should find the following chain of symptoms: increased tension of the eye-ball, rapidly increasing presbyopia, congestion of the anterior ciliary veins (venous hyperæmia), marked arterial pulsation, cloudiness of the aqueous and vitreous humors, with opacities; pupil dilated, of oval shape and quite irresponsive to light, dimness of sight, contraction of the field of vision, shallowness of the anterior chamber, anæsthesia of the cornea and diminution of its lustre, giving it a steamy look, chemosis, epiphora, discoloration of the iris, intense pain, cupping of the disc, nausea and vomiting. These symptoms may pass off, leaving the eye with some defects of central vision, increased tension, and a sluggish and slightly dilated pupil; or they may return again and again, leaving the sight more impaired with each attack, until the vision is completely destroyed. These attacks have frequently been mistaken for neuralgia or stomachic trouble, and only discovered when too late to save the sight.

Acute inflammatory glaucoma usually comes on at night. Occasionally these symptoms come on without any premonitory stage and with such violence as to destroy the sight completely in a few hours. This form is known as glaucoma fulminans, so named by Von Graefe. This form requires prompt treatment.

Again, we have it coming on as chronic inflammatory glaucoma, in which the inflammatory process is similar to that of the acute, only less severe and slower in progress. It is often caused by excitement, mental anxiety or fatigue, excessive exercise or a full meal. It may come on insidiously, as chronic inflammation, recurring frequently, and gradually destroy the sight, or it may be ushered in as an acute attack, which gradually subsides into a chronic form. Acute attacks may develop at any time during the course of the chronic form, and keep on returning at intervals until an iridectomy has to be done for the preservation of sight and relief of pain. Chronic inflammatory glaucoma may lead to complete loss of sight without any severe inflammation or acute pain, and this is the main characteristic between it and the acute form.

Secondary glaucoma is consequent upon some other disease already existing in the eye, such as intra-ocular tumors, staphyloma of the cornea or sclerotic, serous iritis, swelling of an injured lens, or dislocation of the same.

Hæmorrhagic glaucoma is produced by effusions of blood into the retina or optic nerve, and sometimes into the vitreous. These hæmorrhages are usually followed in a few weeks by increased intra ocular tension and the development of acute or sub-acute glaucoma. The chief point of difference between this form and the ordinary, is that loss of vision precedes by some time the actual onset of the inflammatory process. The symptoms are not constant, but subject to great variety. Usually only one eye is affected. There are no premonitory signs. The first symptom is sudden loss of sight. Noyes is of the opinion that in some cases at least, the cause is due to embolism of some of the retinal arteries, with consecutive hæmorrhages in consequence of an atheromatous condition of the vessels, rendering them unable to adjust themselves to the disturbances in the circulation.

This disease may be diagnosed from plastic cyclitis by the following symptoms: In plastic cyclitis we have a deep anterior chamber, deeper than normal and no increase of tension, while the eye is very sensitive to pressure over the ciliary region. From serous cyclitis by the increased depth of the anterior chamber, absence of pain, unless the tension is increased by hypersecretion of aqueous humor, when an examination of the field of vision will tell the tale, and by the appearance of a dust-like opacity in the anterior portion of the vitreous humor. From purulent cyclitis by the appearance of membraneous opacities in the vitreous and hypopion in the anterior chamber. From iritis by the pericorneal injection, occlusion of the pupil, the presence of an inflammatory exudation in the iris, pupil and posterior surface of the cornea, posterior synechia, and increased depth of the anterior chamber, as seen in serous iritis, in which case the pupil may be dilated. The history of the case must be borne in mind as it is frequently due to rheumatism, gout or syphilis, and often is consequent