Pars planitis may occur as a consequence of systemic disease (for example, sarcoidosis or multiple sclerosis or cat scratch or Lyme disease) or it may be idiopathic. Our experience suggests that, at least in a tertiary referral practice, 50% of the cases are idiopathic. Our philosophy regarding a steroid-sparing step ladder algorithm approach to treating pars planitis to accomplish a goal of complete abolition of all active inflammation, regardless of whether or not vision has yet been affected, is associated with significantly less cataract development over the natural history of the patient's disease, and with a better visual outcome because of the prevention of permanent structural damage to macula, with fixed cysts, epiretinal membrane formation, etc. Our step ladder algorithm for the treatment of patients with idiopathic pars planitis differs slightly from that described previously on this web site for the treatment of patients with recurrent anterior non-granulomatous uveitis. Our approach is as follows:

We do not use steroid drops instead, the first step on our therapeutic step ladder in the care of patients with idiopathic pars planitis is regional steroid injection therapy. We prefer to inject through the pre-orbital septum, through the lower lid, in a manner similar to the administration of the peri bulbar injection for anesthesia, the difference being that the steroid (40 mg of Kenalog) is administered through a short 30 gauge needle. Results of studies in our clinic suggest that this approach is equal in efficacy to posterior sub-Tenon's injection technique, is associated with considerably less intraocular pressure elevation, and is much more acceptable to the patient.

If, after a series of six transseptal steroid injections separated by at least two weeks, the patient's pars plana inflammation recurs or continues to recur, we add a systemic non-steroidal anti-inflammatory drug (for example, Naprosyn, 500 mg PO BID).

Pars plana cryopexy follows, if the patient's inflammation continues to recur despite the use of the systemic non-steroidal anti-inflammatory agent.

The choice between systemic immunosuppressive chemotherapy, for example with low dose Cyclosporin or with once a week Methotrexate, or pars plana vitrectomy depends greatly on the individual circumstance, based on the patient's age, sex, other medical disease, and whether or not the patient is phakic or aphakic. If we choose an immunosuppressive chemotherapeutic agent, we will usually begin with either once a week Methotrexate or with systemic Cyclosporin (low dose) again, based on the patients age, sex, and past medical history (eg a history of liver problems or with renal problems).

For reasons that are not absolutely clear, pars plana vitrectomy can have a substantial ameliorating effect on the likelihood that inflammation at the pars plana will continue to recur in patients with recurrent idiopathic pars planitis. Some have argued that the immunologic characteristics of the eye are significantly changed by producing a "unicameral" eye rather than a bicameral one; others have argued one rids the eye of long lived "memory" immunologically competent cells from the vitreous matrix, cells which are at
least partially responsible for a continued or recurrent out-pouring of inflammatory cytokines. Regardless of the mechanism or explanation, we agree with others that this therapeutic technique is in fact effective, and therefore we include, it in our therapeutic armamentarium in the care of patients with recurrent pars planitis.

In summary, pars planitis is a significant, vision-robbing inflammatory disease that probably should be treated more aggressively than it usually is. Our step ladder approach in such an aggressive approach is outlined above.