In the two preceding chapters on general and specific signs and symptoms of intracranial aneurysms there were brief discussions of ophthalmic signs and symptoms. The preponderance of these latter findings in patients and the volume of literature on the subject (1-152) necessitates a separate chapter. Most patients with either ruptured aneurysms or unruptured large aneurysms will have some alterations of cranial nerves (CN) 2, 3, 4, and 5 and/or of the ocular fundus. These alterations often are diagnostic and even localizing but are frequently missed without a detailed neuroophthalmologic study.

This chapter will not make detailed distinctions between small and large aneurysms, partly because the authors quoted did not provide the size and partly because a small aneurysm close to a cranial nerve may give findings similar to those of a large aneurysm whose origin is some distance from the cranial nerve. Emphasis is placed on the prominent role ophthalmology plays in assessing the patient with an intracranial aneurysm. For example, Brenner and Pendl (14) studied 491 aneurysm cases and found that 24% had signs and symptoms related to fundal changes, eye motor activity, or ocular sensation. Ten percent (52 aneurysms) had no SAH but presented with neuroophthalmologic signs. Figure 7.1 illustrates the site of these 52 aneurysms and their relation to cranial nerves. The largest number (22) were in the cavernous sinus. Walsh and Hoyt (147) believed that “ocular signs are the most important focal neurologic manifestations of unruptured intracranial aneurysms.” In 94 autopsied cases of ruptured aneurysm Hyland and Barnett (69) found 40% had cranial nerve involvement—one-half due to direct contact by the aneurysm and one-half due to secondary effects of the bleed upon the brain.

Cranial Nerves 3, 4, 5, and 6

Trigeminal Nerve (CN-5)

The fifth, or trigeminal, nerve (ophthalmic, maxillary, and mandibular divisions in decreasing order of frequency of involvement) is rarely compromised except by giant aneurysms or by ic/cslICA (see below). True tic douloureux pain is rare in aneurysm patients (see Chapter 6). Forehead or retroorbital pain may occur from tentorial irritation (referred ophthalmic division pain). Trigeminal pain may be seen with an aneurysm adjacent to CN-5. Suspicion should be aroused in the presence of a depressed corneal reflex or sensory deficit.

Oculomotor Nerve (CN-3)

A number of authors have demonstrated that the oculomotor nerve is the most commonly compromised nerve of extraocular movements (9, 12-14, 18-20, 25, 27, 29, 35, 48, 56, 60, 61, 63-65, 67-69, 89, 100, 110, 112, 116, 125, 129, 147, 152). The large majority of these cases had an ICAA, especially in the cavernous sinus or at the posterior communicating artery take-off. The first author to demonstrate a CN-3 palsy from a pcICA was France (52) in 1846.

Exact percentages are quite variable among different studies. Charamis et al. (25) found a 20% incidence of oculomotor nerve paresis in
Signs and Symptoms

Fig. 7.1. Diagram of sites of 52 aneurysms causing neuroophthalmologic signs without SAH. Arabic numbers, number of aneurysms. Roman numbers, cranial nerves. Oral direction to the left. Foreground, Gasserian ganglion. Background, sella turcica. From Brenner H, Pendl G: ref. 14.

102 intracranial aneurysm cases with ophthalmic findings. Dailey et al. (29) studied 65 cases of intracranial aneurysm associated with ophthalmic findings; 42% had an isolated CN-3 palsy and 21% had CN-3 plus CN-4, -5, or -6 deficits. In Zielinski's (152) 83 intracranial aneurysm cases, 28% had evidence of CN-3 weakness. Holmes (67) found a 12% incidence of CN-3 palsy in 120 cases of intracranial aneurysm. Brenner and Pendl (14) discovered that 54% of 52 unruptured symptomatic intracranial aneurysms had a CN-3 palsy. Riise (116) examined 100 cases of ICAA; 39% had developed CN-3 palsy. Thirty-four percent of 174 pcaICAA cases had oculomotor palsy by Soni's count (129).

On the other hand, Green et al. (58) investigated 130 cases of CN-3 palsy of various etiologies and listed the following percentages caused by disease processes: 30%, parasellar aneurysm; 20% diabetes; 10%, trauma; 9%, syphilis; 4%, tumor; 27%, other. Rucker (120,121), in two studies of CN-3, -4, and -6 palsies from all causes, found the following percentages (combined statistics of both studies) due to intracranial aneurysms: 609 cases of CN-3 palsy, 19%; 151 cases of CN-4 palsies, 0%; 924 cases of CN-6 palsies, 3%; 88 cases of CN-3 and -4 palsies, 23%; 105 cases of CN-3 and -6 palsies, 10%; 122 cases of CN-3, -4, and -6 palsies, 9%.

The signs of a CN-3 palsy or paresis from a nearby ruptured aneurysm include the following features (29,61,147): firstly, the pupil dilates; secondly, the lid droops; thirdly, extraocular motion becomes limited. Pupillary dilation is slight at first, achieving an increasing degree of dilation as the eyelid becomes ptotic. Finally, the pupillary light reflex is totally lost; the pupil is widely dilated; the eyelid is closed; and paresis of elevation, depression, and adduction of the ipsilateral eye increases. The closed eyelid prevents the subjective diplopia, but invariably there is a localizing headache or retroorbital pain from irritation of the tentorium (referred ophthalmic division pain).

In 40 cases of pcaICAA with oculomotor palsy, Harris and Udvarhelyi (61) found that the CN-3 paresis first appeared from 2 to 7 weeks after the SAH (average, 3 weeks) and took 2.5 days to become complete. In cases with frontal pain (SAH not diagnosed) the palsy appeared at about the second week and usually took 6 days to become complete. Only rarely is the pupil not involved (80,120,121). Rucker (120,121) had four cases out of 114 with no pupillary dilation. The pupil is small in Raeder's paratrigeminal syndrome (miosis, ptosis, ophthalmic division pain, absence of anhidrosis) in the rare case caused by an internal carotid aneurysm (85). Simultaneous CN-3 palsy and sympathetic plexus disruption will result in a fixed, midzone pupil.

France's (52) first report of an aneurysm causing CN-3 palsy in 1846 had dilation of the pupil. Walsh and Hoyt (147) stated, "absence of pupillary involvement almost invariably excludes a diagnosis of aneurysm." They went on to opine, "isolated pupillary paralysis without involvement of extraocular muscles innervated by the third nerve virtually excludes the possibility of intracranial carotid aneurysm." The latter statement would not apply to early cases, for this author has occasionally seen early pupil-