birth-related in the first month of life; AHT after the first month; and less commonly, nAHT.[137,155,156,169–171]

The pattern and distribution of the hemorrhages, presence of other retinal findings, clinical history, nonocular physical examination, and laboratory tests help to distinguish traumatic from nontraumatic causes of retinal hemorrhage. Vitreous hemorrhage may also be present in AHT but is less commonly seen than retinal hemorrhage. Retinoschisis and retinal folds are also less common but are highly specific for severe trauma, including AHT.[137] Apart from their diagnostic significance, retinoschisis and retinal folds are indictors of poor prognosis and associated with severe neurologic injury and mortality.[143,144] Retinal detachment is another less common sign and may signify a late presentation.[172]

Generally, increased severity of retinal hemorrhages correlates with increased severity of head trauma.[150,173] Extensive hemorrhages that are "too numerous to count," found at multiple layers, and extend to the peripheral ora serrata are highly specific for severe head trauma, including AHT and unambiguous severe nAHT.[40,174,175] However, the features of multilayered or peripheral RHs in isolation do not define a level of severity and, in and of themselves, are not diagnostic of AHT; the number and geographic distribution must also be considered.[158] Retinal hemorrhages are less common in AHT characterized only by blunt impact, similar to the low incidence observed in accidental falls.[137,151] Retinal hemorrhages occasionally occur from witnessed accidental "household" falls (ie, typically representing low-height or short-distance falls), but in this setting typically are sparse and not in the pattern more specific to AHT.[93,96,175] Retinal hemorrhages tend to occur more frequently in children who have died versus unimpaired survivors.[143,150,151,176,177]

A systematic review of 20 observational studies comprised ocular findings in 1948 children, including 242 children with nAHT and 973 children with AHT as determined by perpetrator confession, third-party witness, legal decision, autopsy findings, or multidisciplinary assessment.[137] This review found that intraocular hemorrhage was present in 44% to 100% of children with AHT but in less than 10% of children with nonabusive head injury. It should be noted that the children with accidental trauma in most comparative series typically have different types of intracranial injuries than do children with AHT, only infrequently having subdural hemorrhages. However, with this caveat in mind, the sensitivity of intraocular hemorrhages for AHT was 75%; the specificity for AHT was 94%. The specificity for AHT was highest for intraocular hemorrhage that was bilateral, multilayered (preretinal in addition to intraretinal), extended beyond the posterior pole, and of moderate to severe number/extent.

In addition to the multilayered, numerous in number, and extending to the periphery retinal hemorrhages, retinal folds and retinoschisis are also highly specific for severe head trauma, most commonly AHT.[178–180] They also have been reported after unequivocal severe accidental head injury, such as fatal motor vehicle crashes, head crush injury, and high-elevation (eg, 10 m) falls, and more rarely in association with certain established medical diagnoses, including a ruptured intracranial aneurysm and leukemia.[152,181] Thus, when macular retinoschisis or perimacular retinal folds are present in a child without a history of major accidental head trauma, the likelihood of AHT is very high. The presence of retinal folds typically implies severe injury that often results in death or neurologic sequelae, although some surviving patients may experience good visual recovery.[143,144]

ii) Increased intracranial pressure. Raised intracranial pressure causes specific patterns of retinal hemorrhages that are different from head trauma. Raised intracranial pressure is associated with superficial intraretinal hemorrhages on or adjacent to a clearly swollen optic nerve head and, therefore, are in a peripapillary distribution.[182] Current evidence and extensive clinical experience with patients with raised intracranial pressure unrelated to trauma does not support raised intracranial pressure as a cause of widespread or multilayered retinal hemorrhages.[183,184] A very rapid elevation of intracranial pressure resulting from intracranial hemorrhage can cause intraocular hemorrhage in a condition referred to as Terson syndrome.[185–190] The pattern of hemorrhages in Terson syndrome is mainly of vitreous and preretinal hemorrhage adjacent to the optic nerve head or in the posterior pole with no or few intraretinal hemorrhages. Terson syndrome is uncommon in infants and children and does not refer to a specific cause, such as a ruptured arteriovenous malformation.[151,191,192]

iii) Other systemic illness. Patients with severe coagulopathies may rarely be found to have retinal hemorrhages, which are usually few in number and confined to the posterior pole; although they may be more widespread in children with leukemia.[193] It is not clear to what degree coagulopathy might worsen the severity of retinal hemorrhage attributable to head trauma, but even coagulopathy is not known to cause spontaneous extensive retinal hemorrhages.[194] Sepsis, meningitis, and endocarditis may be associated with hemorrhages that are typically intraretinal and confined to the posterior pole but can also be more extensive.[195–197] In a prospective case series of 159 critically ill children without abusive head injury receiving ophthalmologic evaluation within 48 to 72 hours of admission, the prevalence of retinal hemorrhages was 15% (95% CI, 9.5%–21%).[193] Most of these children had fewer than 5 single-layered retinal hemorrhages, but 6 children (3.7%) were found to have more numerous or multilayered hemorrhages and had diagnoses of fatal nonabusive traumatic