Acute visual loss

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Table 12-1

CAUSES OF EPISODIC VISUAL LOSS

Adolescence and young adulthood

- Migraine
- Optic neuritis
- Papilledema
- Antiphospholipid antibody syndrome and systemic lupus erythematosus
- Early tumor compression of the optic nerve
- Takayasu aortic arteritis
- Viral neuroretinitis
- Idiopathic

Adulthood

- Carotid stenosis or dissection
- Embolism to the retina
- Intrinsic central retinal artery atherosclerotic disease
- Temporal arteritis (generally over age 55)
- Glaucoma
- Papilledema

RISK FACTORS OF CRAO

- 50 to 70 years old,
- vascular risk factors
- history of collagen vascular disease
- vasculitis
- cardiac valvular abnormality
- sickle cell disease
- increased orbital pressure
- acute glaucoma
- retrobulbar hemorrhage
- endocrine exophthalmos
- hypercoaguable states, including anti-phospholipid antibody syndrome
- Giant cell arteritis
- carotid or aortic athero- sclerosis

CRAO
SUDDEN PAINLESS BLINDNESS

1. non-arteritic and permanent (constituting over two-thirds of all CRAO cases), such as an ischemic stroke of the retina
2. non-arteritic and transient, which may be seen with a transient ischemic attack [TIA] of the retina related to vasospasm
3. arteritic due to a systemic inflammatory condition like temporal arteritis

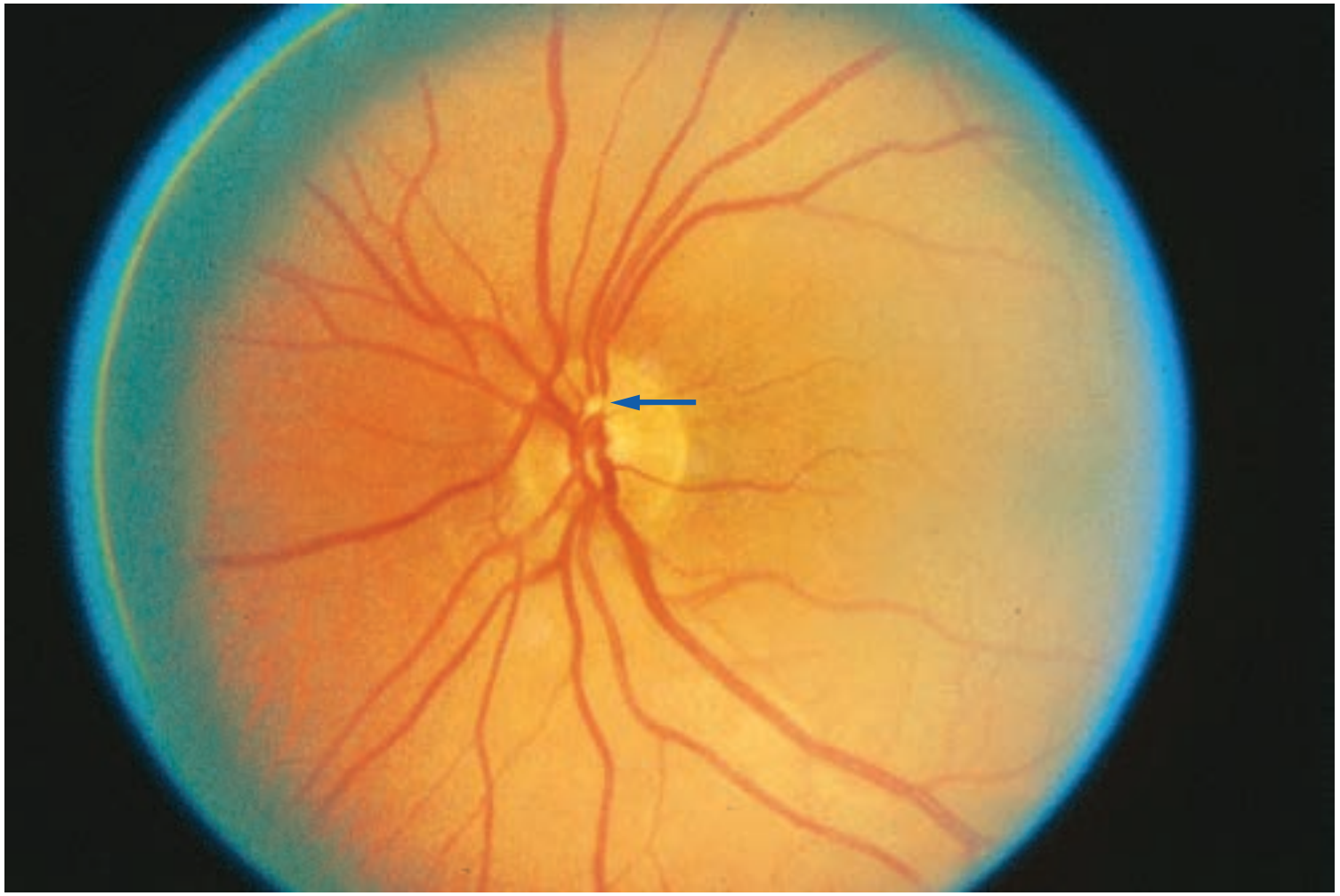
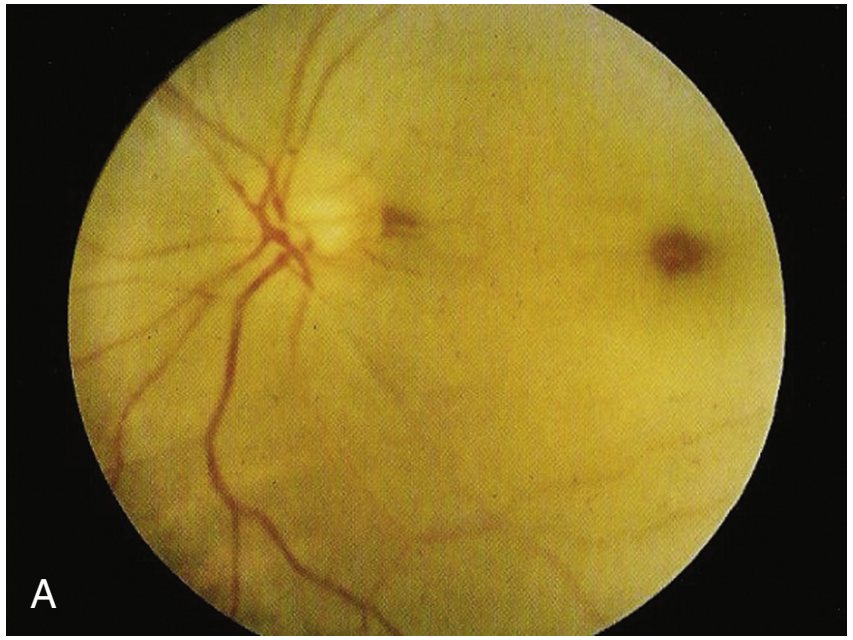


Figure 12-9. Glistening "Hollenhorst plaque" occlusion of a superior retinal artery branch (*arrow*). These occlusions represent atheromatous particles or, less often, platelet-fibrin emboli. Some are asymptomatic and others are associated with segmental visual loss or are seen after central retinal artery occlusion. (Courtesy of Dr. Shirley Wray.)

CRAO
SUDDEN PAINLESS BLINDNESS

1. The retina becomes opaque
2. gray-yellow appearance
3. the arterioles are narrowed segmentation of columns of blood
4. A “cherry-red spot” appears in the fovea



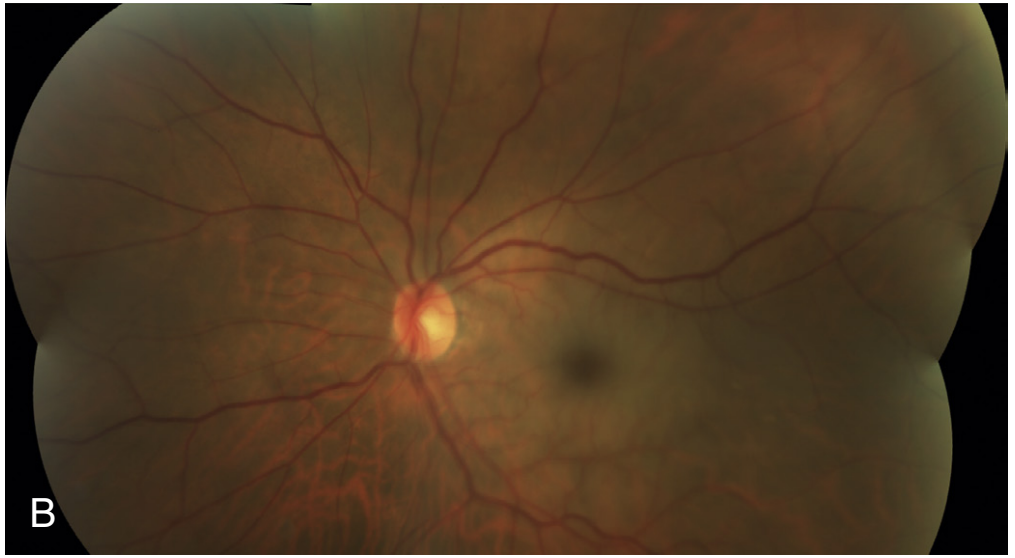


Fig. 57.28 Central retinal artery occlusion (CRAO). (A) Note the cherry-red spot representing the fovea. (B) Note whitening of the retina, with a less prominent cherry-red spot. (B) (Courtesy Jeffrey Lee, MD, University of California San Diego.)

CENTRAL RETINAL VEIN OCCLUSION CRVO

- dilated and tortuous veins
- retinal hemorrhages
- disk edema

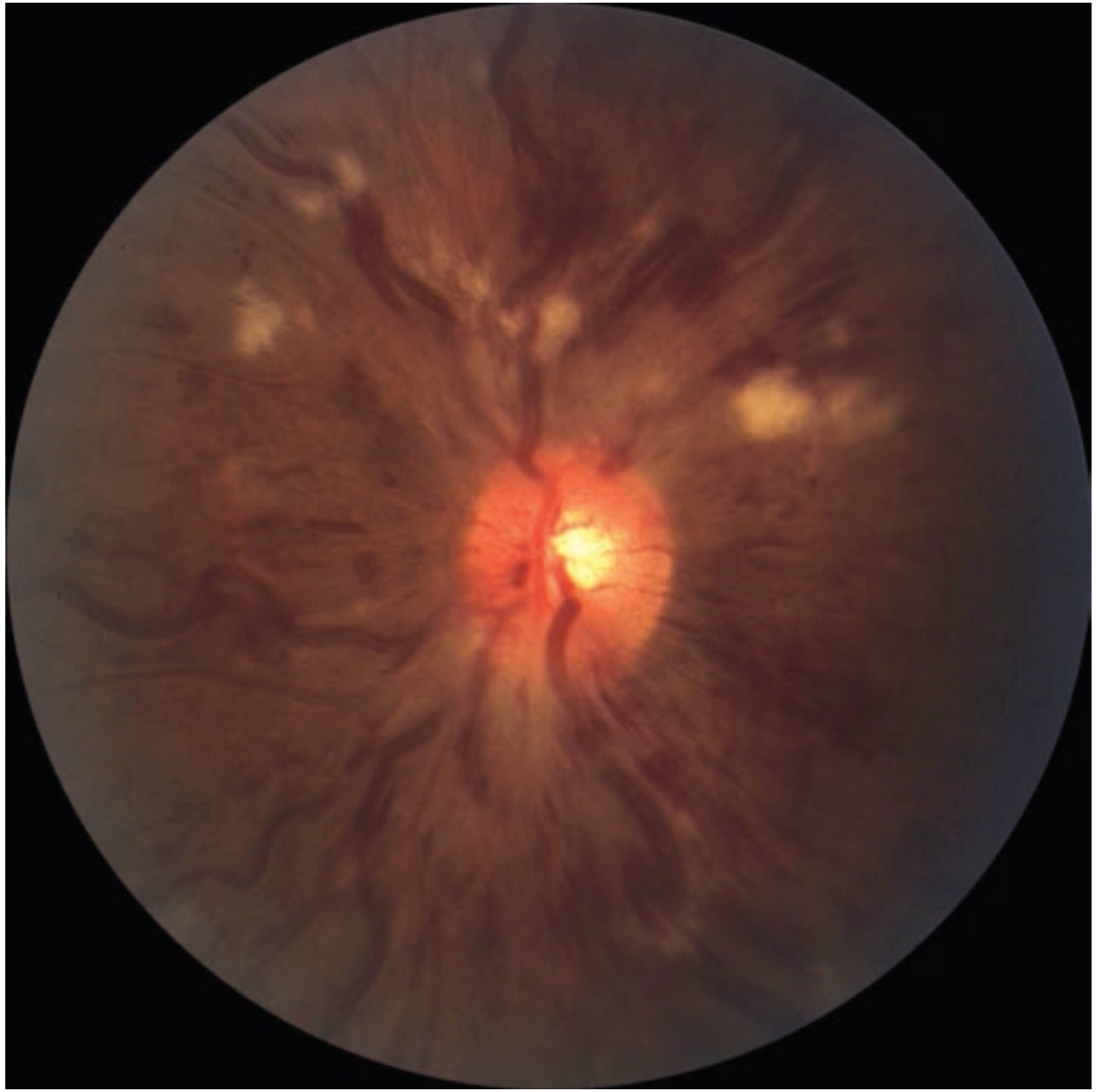


Figure 12-10. Occlusion of the central retinal vein with suffusion of the veins, swelling of the disc, and florid retinal hemorrhages. (Courtesy of Dr. Shirley Wray.)

CENTRAL RETINAL VEIN OCCLUSION CRVO

- Neovascular glaucoma and macular edema
- a non-ischemic CRVO is associated with dilatation of retinal vessels and edema only
- ischemic CRVO presents with the sudden onset of painless vision loss in one eye

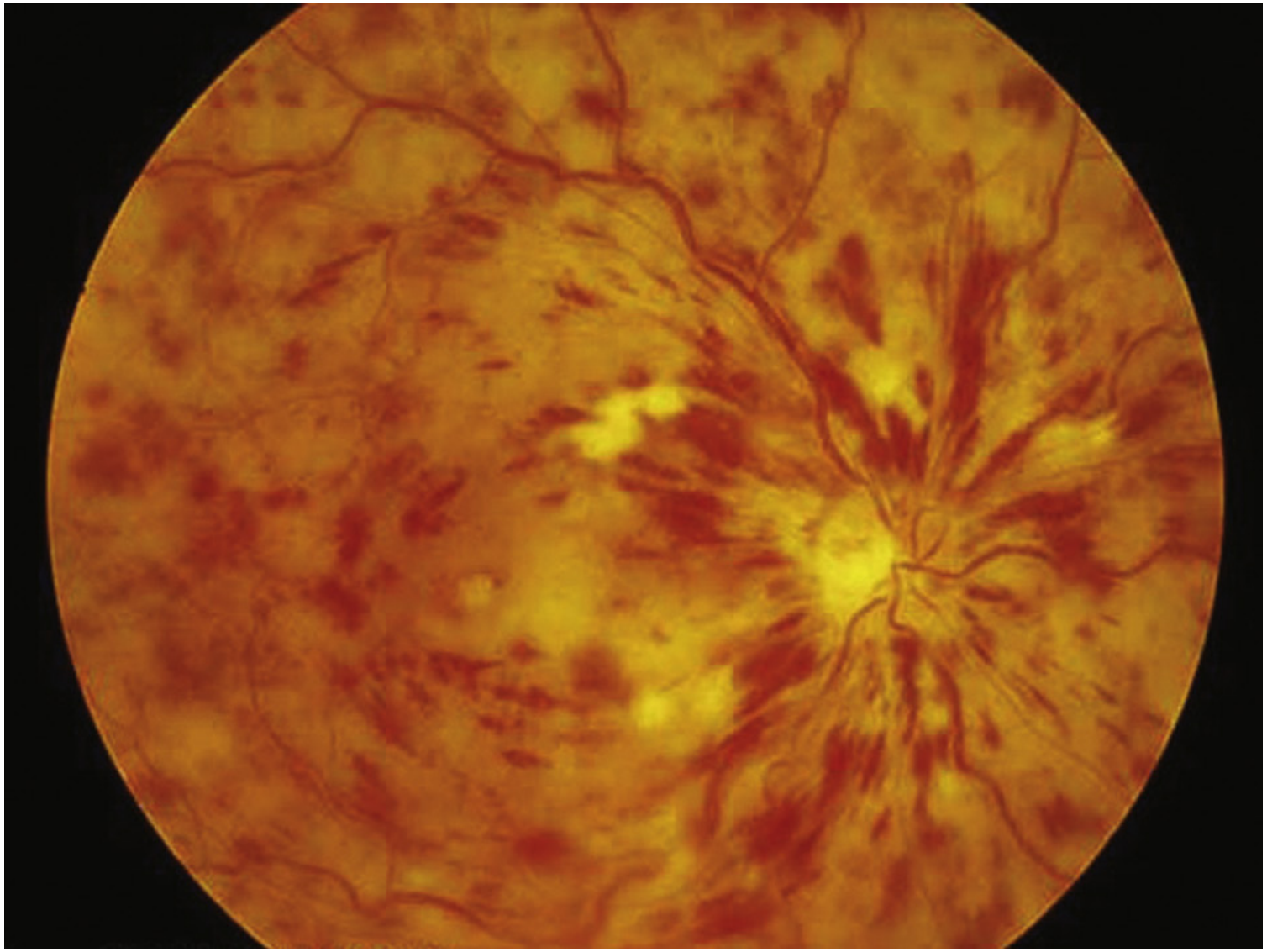


Fig. 57.29 Central Retinal Vein occlusion. Note the "blood and thunder" appearance. (Courtesy www.tedmontgomery.com.)

RISK FACTORS
CRVO

1. hypertension
2. hyperlipidemia
3. diabetes mellitus
4. vasculitides
5. hyperviscosity
6. smoking

TREATMENT

- Like acute ischemic stroke, CRAO is a time-sensitive process.
- direct digital pressure
- dilation of the artery to promote forward blood flow by increasing intra-arterial carbon dioxide level [pCO2] with an inhaled mixture of 95% oxygen and 5% carbon dioxide [carbogen]
- reduction of IOP through anterior chamber paracentesis to increase the perfusion gradient
- hyperbaric oxygen (HBO)
- thrombolytic agents

TREATMENT

- Treatment of CRVO includes treating the underlying etiology
- anti-vascular endothelial growth factor pharmacotherapies
- intravitreal injections of corticosteroids
- antiangiogenic monoclonal antibodies (ranibizumab, bevacizumab)
- retinal photocoagulation
- cyclocryotherapy
- low-molecular-weight heparin

TREATMENT

- ***Ischemic optic neuropathy***
- Admit patients for high-dose IV methylprednisolone

FUNCTIONAL VISION LOSS

1. rocking a mirror slowly back and forth in front of the patient
2. rapidly moving the examiner's hand toward the eye in question
3. checking for an APD(afferent pupillary defect) *Marcus Gunn pupil*
4. having the patient raise his or her arms and touch both index fin- gers together