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“Papilledema”: Neuroradiologic Evaluation of Optic Disk Protrusion with Dynamic Orbital CT

John R. Jenkins¹

Current-generation CT scanners enable the visualization in vivo of structures and substructures that were previously unobservable. Certainly the orbit and optic nerve/sheath complex have demonstrated a great number of pathologic and normal anatomic variations. It has been found in patients with elevated intracranial pressure that what was previously thought to be simple papilledema in fact masks a surprisingly large component of optic papilla protrusion. There may be a variable amount of increased intercellular/axonal fluid within the optic disk in patients with increased intracranial pressure; however, a significant factor in the “swollen disk” is the simple transmission of pressure along the optic nerve sheath to the papilla, causing it to bulge. Further investigations with dynamic CT reveal that there is decreased perfusion of the optic disk in the active phase of severe increased intracranial pressure in patients with papilledema and/or protrusion as compared with normal control subjects. This depressed flow pattern seems to originate subacutely and appears to resolve in certain patients after normalization of the elevated pressure. These findings apparently indicate that clinical intervention in cases of intracranial hypertension to restore the hemodynamic status of the optic disk would be timely, and thereby avert irreversible damage. This suggests and supports the theory that increased intracranial pressure may lead to rapid vision loss by the mechanical mechanism of pressure projected directly to the junction of the optic nerve and optic nerve head, leading to decreased perfusion, ischemia, axonal flow stasis, and resultant optic nerve atrophy.

The medical definitions of papilledema range from the abbreviated “edema of the optic disc,” [1] to the definitive, “edema of the optic disc . . . most commonly due to increased intracranial pressure, malignant hypertension, or thrombosis of the central retinal vein” [2]. Almost all definitions, however, stress a dominant or sole component of real edema in the optic nerve head accounting for the swelling without regard to differing pathologic processes [3, 4]. This study was undertaken to evaluate both the in vivo structure of the optic nerve/sheath complex at its junction at the optic nerve head in patients with clinical papilledema caused by intracranial hypertension as well as to study the perfusion characteristics of the optic papilla in patients with increased intracranial pressure (ICP) from various causes.

Subjects and Methods

The scanner used in this study was the GE 9800 with dynamic capability. Unenhanced static scans were obtained with 5-mm-thick axial sections through the orbit followed by 3-mm sections through the optic nerve head subsequent to IV administration of contrast material.

The patient population encompassed a total of 20 subjects with increased ICP caused by various processes including trauma, premature craniosynostosis, hydrocephalus, true tumor, primary pseudotumor, and pseudotumor secondary to cerebral venous sinus thrombosis. In addition, a control group of eight patients scanned for reasons other than increased ICP completed the study (Table 1).

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TABLE 1: Papilledema: Summary of Cases

Case No.	Age	Gender	History	Diagnosis	Clinical Findings	Visual Acuity	CT of Optic Disk	
							Static	Dynamic
1	1	M	Orbital swelling	Contralateral retinoblastoma	Contralateral orbital mass	Unknown	Flat disk	Not performed
2	21	M	Headaches	Nonorganic headaches	Normal	20/20	Flat disks	Not performed
3	30	F	R facial weakness	Facial nerve neuropathy, ?etiology	Normal	20/20	Flat disks	Not performed
4	22	F	Diplopia	Temporal meningioma	R III & VI cranial nerve palsy ?Retinitis	Normal	Flat disks	Normal disk perfusion
5	31	M	Chronic headache	Nonorganic headaches	Normal	Normal	Flat disk	Normal disk perfusion
6	10	M	Orbital swelling	Contralateral lymphangioma	Contralateral orbital mass with hemorrhage	Unknown	Flat disk	Normal disk perfusion
7	33	M	Orbital swelling	Contralateral hemangioma	Contralateral orbital mass	20/20	Flat disk	Normal disk perfusion
8	18	F	Headaches	Nonorganic headaches	Normal	20/20	Flat disks	Normal disk perfusion
9	19	F	Headache, nausea, vomiting, decreased vision	Pseudotumor secondary to cerebral venous sinus thrombosis	Papilledema: resolved to optic atrophy	Blind	Initial: optic sheath dilatation; late: flat disks	Normal disk perfusion after resolution of ICP
10	25	F	Headache, nausea, vomiting, decreased vision	Pseudotumor secondary to cerebral venous sinus thrombosis	Papilledema: resolved to optic atrophy	OD: 20/60, OS: 20/50	Initial: optic sheath dilatation; late: flat disks	Normal disk perfusion after resolution of ICP
11	4	F	Headache, vision loss	Premature craniosynostosis	Optic atrophy, papilledema	Blind	Optic sheath dilatation, papilla protrusion	Not performed
12	46	M	Headache, meningismus	Bacterial meningitis, hydrocephalus	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Not performed
13	20	F	Headache, vomiting	Primary pseudotumor	Papilledema	OD: 20/40, OS: 20/50	Optic sheath dilatation, papilla protrusion	Not performed
14	3	F	Headache	TB meningitis, hydrocephalus	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Not performed
15	9	F	Nausea, vomiting, decreased vision	Thalamic astrocytoma and hydrocephalus	Papilledema	OD: 20/30, OS: 20/30	Optic sheath dilatation, papilla protrusion	Not performed
16	23	F	Headache, decreased vision, R-sided weakness	Traumatic subdural hematoma	Papilledema	OD: 20/30, OS: 20/30	Optic sheath dilatation, papilla protrusion	Not performed
17	31	F	Headache, nausea, vomiting	Primary pseudotumor	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Not performed
18	14	M	Headache, nausea, vomiting	Pineal germinoma, hydrocephalus	Optic atrophy	OD: 20/30, OS: 20/70	Optic sheath dilatation, papilla protrusion	Not performed
19	2	M	Progressive weakness, enlarging head	Midbrain glioma, hydrocephalus	Papilledema, retinal hemorrhages	Unknown	Optic sheath dilatation, papilla protrusion	Not performed
20	1	M	Progressively enlarging head & CHF	Arteriovenous fistula, vein of Galen aneurysm, hydrocephalus	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Not performed

21	2	F	Irritability, delayed development	Aqueductal stenosis, hydrocephalus	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Not performed
22	45	M	24 hr onset: headache, coma	AVM, hemorrhage, hydrocephalus	Papilledema	Unknown	Optic sheath dilatation, papilla protrusion	Normal disk perfusion
23	29	F	Headache, nausea, vomiting decreased vision	Primary pseudotumor	Papilledema	OD: 20/50, OS: 20/100	Optic sheath dilatation, papilla protrusion	Depressed perfusion of optic disk
24	15	F	Headache, vomiting	Primary pseudotumor	Papilledema, retinal hemorrhages	Unknown	Optic sheath dilatation, papilla protrusion	Depressed perfusion of disks
25	50	F	Headache, nausea, vomiting, decreased vision	Lung cancer metastases with hydrocephalus	Papilledema, retinal hemorrhages	Unknown	Optic sheath dilatation, papilla protrusion	Depressed perfusion of optic disk
26	63	F	Decreased vision, weakness, dementia	Cerebellopontine angle arachnoid cyst, pituitary adenoma, hydrocephalus	Optic atrophy, papilledema	Long-term blindness	Optic sheath dilatation, papilla protrusion	Depressed perfusion of optic disk
27	23	M	Headache, nausea, vomiting	Pseudotumor secondary to cerebral venous sinus thrombosis	Papilledema, retinal hemorrhages	OD: 20/40, OS: 20/200	Optic sheath dilatation, papilla protrusion	Depressed perfusion of optic disk
28	23	M	Headache, decreased vision	Pseudotumor secondary to venous sinus thrombosis	Papilledema	"Decreased"	Optic sheath dilatation papilla protrusion	Depressed perfusion of optic disk, extravasation of contrast material

Note.—R = right; OS = left eye; ICP = intracranial pressure; TB = tuberculosis; CHF = congestive heart failure; AVM = arteriovenous malformation.

The dynamic injection format was standardized at 4 ml/sec of Renografin-76 for a total of 60 ml introduced with a power injector in an antecubital vein through a short, 20-gauge venous catheter. Simultaneously, the scan sequence began with one initial scan followed by an 8-sec arm-to-head circulation delay, ending after 12 scans in rapid sequence (about 4.5 sec/scan); the entire sequence lasted 1 min. The dynamic scan thickness was 5 mm and the axial angle was through a plane bisecting the orbital apex. From the group of 28 patients, a complement of seven of the patients with clinical papilledema, two with resolved papilledema, and five "normal" subjects were scanned with this dynamic technique.

Results

Regardless of the cause of cranial hypertension, certain features were noted repeatedly in the study group of patients with papilledema as compared with normal subjects (Fig. 1). These features included (1) a dilated optic nerve sheath that was seen in all 20 patients on IV-contrast thin-section orbital CT (Fig. 2), as well as on metrizamide cisternography in one patient; (2) a "bulging" of the terminal optic sheath subarachnoid space into the posterior aspect of the globe at the optic nerve head in 18 of 20 patients with clinical papilledema (Figs. 2-4); and (3) depressed perfusion of the optic disk/nerve junction as measured by dynamic CT at the time of papilledema diagnosis in all six patients with chronic symptomatology (greater than 4-weeks duration) (Figs. 5 and 6). This aberrant perfusion was seen in primary pseudotumor (two cases), pseudotumor secondary to cerebral venous sinus thrombosis (two cases), and obstructive hydrocephalus caused by posterior fossa arachnoid cyst (one case) and metastases (one case).

With regard to this depressed perfusion, evaluations were based on reference to both normal subjects as well as to the choroid adjacent to the protruding optic disk. The latter seems a valid standard, as the choroid may receive its blood supply from a source other than the central retinal artery, namely, the ciliary arteries. Importantly, those ciliary arteries feeding the peripheral choroid surrounding the swollen disk are not subjected to the influences of the increased pressure within the optic nerve sheath. However, this pressure may directly affect both the ciliary vessels supplying the protruding optic papilla as well as the central retinal arterial and venous structures traversing the optic nerve itself [5-8].

In an attempt to follow this aberrant flow state, two other patients not scanned with dynamic techniques during the acute phase of cranial hypertension were later scanned after resolution of the papilledema [9]; normal perfusion patterns were seen, despite blindness in one individual and moderate visual impairment in the other (Fig. 7).

Discussion

Papilledema may result from many causes, including developmental changes, drug medication, metabolic diseases, true tumors, primary or secondary pseudotumor cerebri, trauma, hemorrhage, vascular accidents, inflammatory processes, or any one of many other acquired disease processes of the orbit or brain. The interest in papilledema lies not only

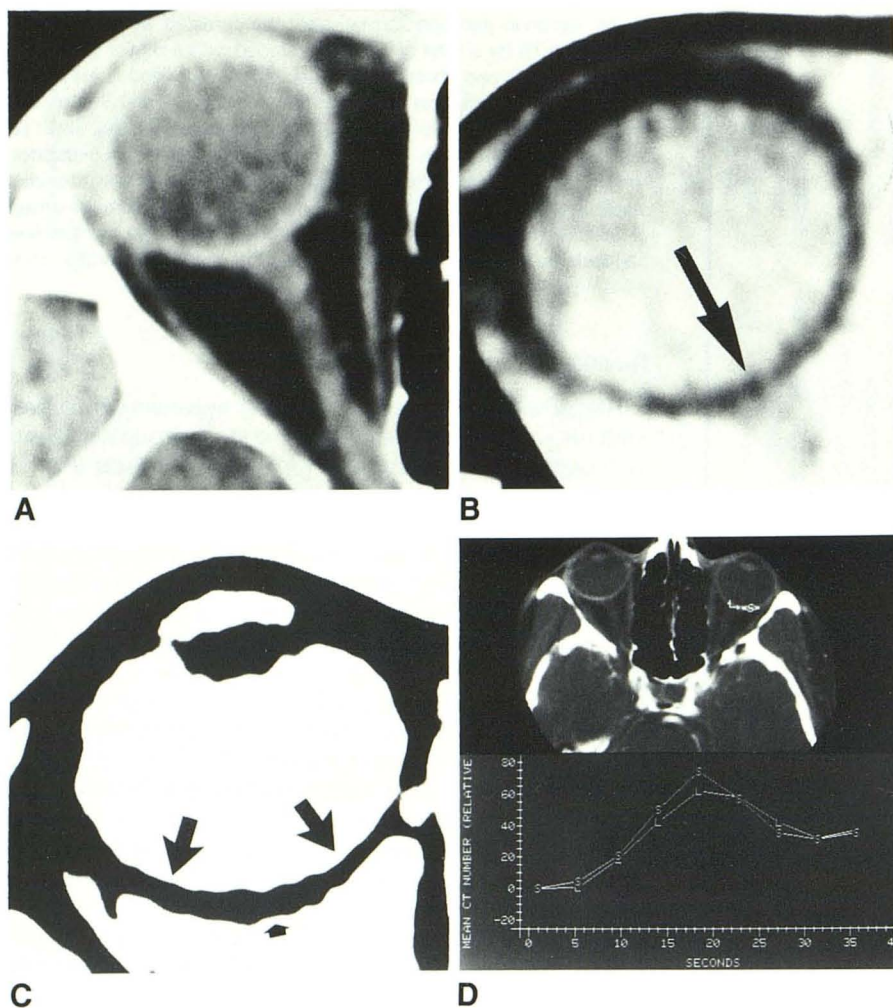


Fig. 1.—Case 8.

A, Magnified view of right orbit shows normal appearance of optic nerve/sheath complex.

B, Video-reverse magnified view of right globe shows normal, flat appearance of optic disk (arrow).

C, Enlarged video-reverse view of globe during dynamic perfusion peak in normal subject shows complete rim of "choroidal" enhancement (large arrows) with equal enhancement of optic nerve head (small arrow).

D, Dynamic perfusion curves centered over orbits show almost identical perfusion patterns of left optic nerve head (L) as compared with adjacent choroid of left globe (S).

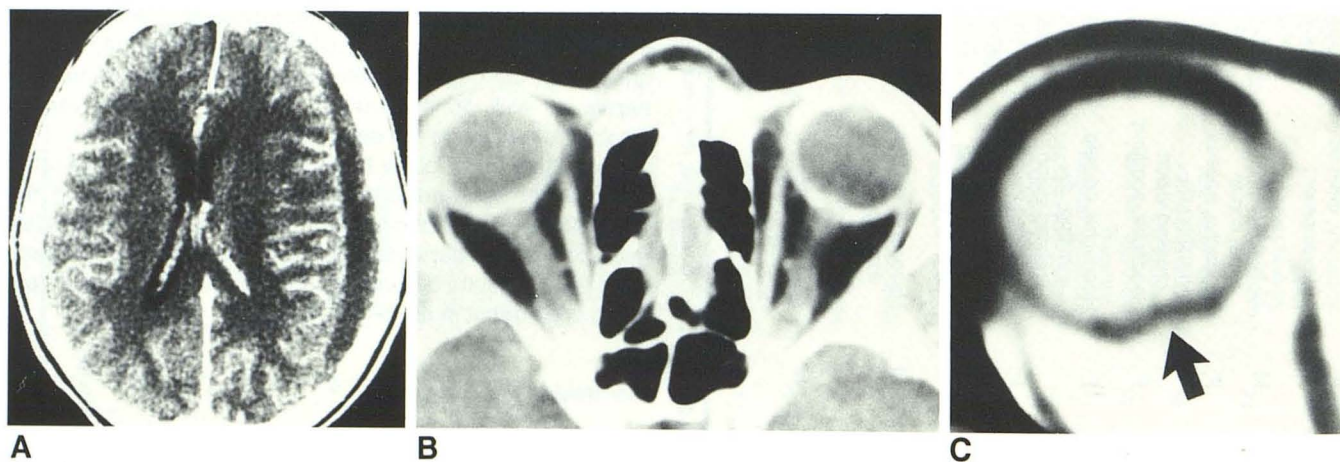


Fig. 2.—Case 16.

A, Axial section shows left hemispheric subdural hematoma.

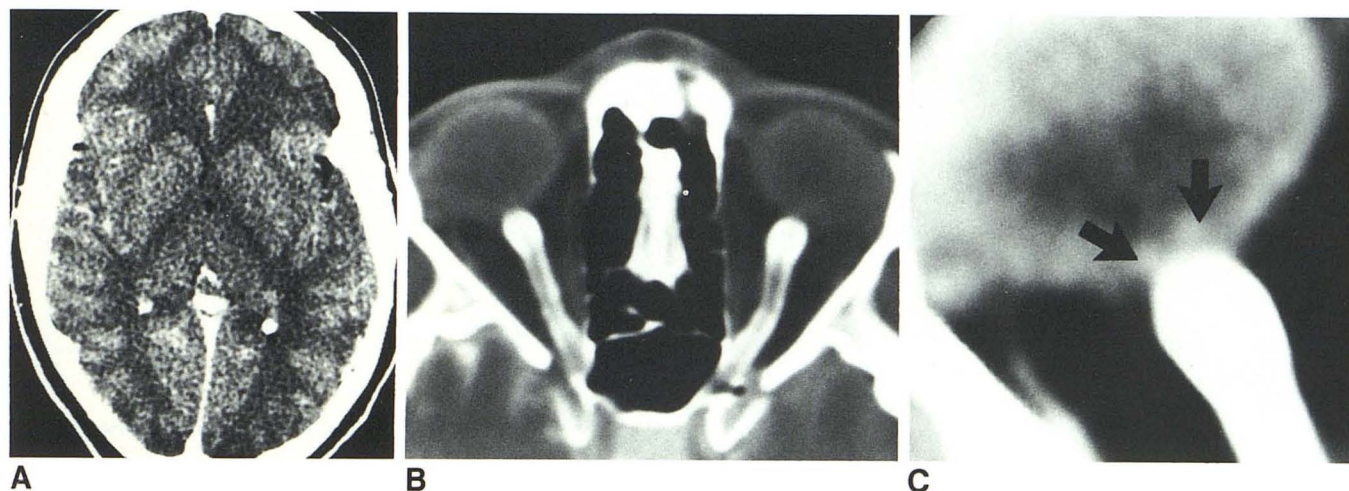
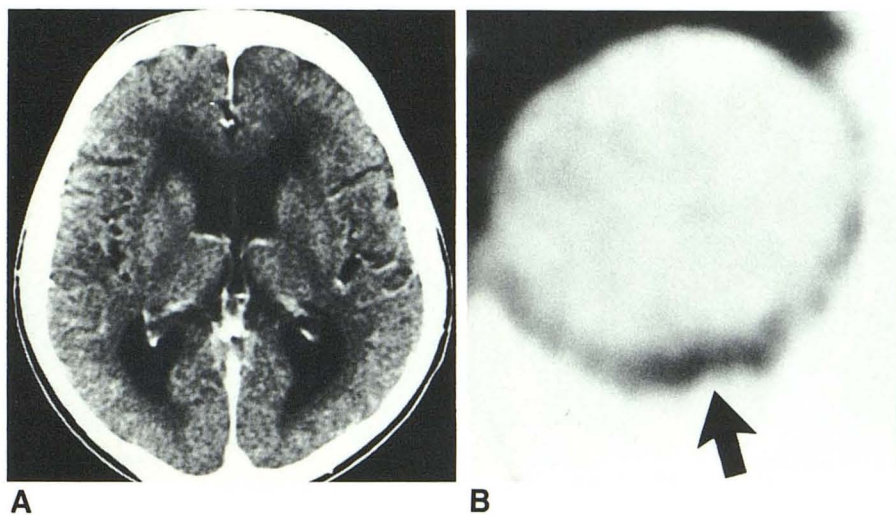
B, Magnified view over orbits shows dilated optic nerve sheaths bilaterally.

C, Video-reverse magnified image of right globe after IV enhancement shows optic papilla protruding into posterior aspect of globe (arrow).

Fig. 3.—Case 26.

A, Axial CT section at level of thalami shows hydrocephalus and periventricular edema secondary to posterior fossa arachnoid cyst causing obstruction to CSF flow at fourth ventricular level.

B, Magnified contrast-enhanced view of right globe on video-reverse imaging shows optic papilla protruding into posterior aspect of globe (arrow).

**Fig. 4.—Case 13: primary pseudotumor cerebri.**

A, Axial cranial CT after contrast administration shows compressed lateral ventricular system.

B, Metrizamide cisternogram centered over orbits outlines markedly dilated optic nerve sheaths extending to globes, traversed by low-density optic nerves.

C, Magnified view of right orbit shows contrast-filled, ballooned terminal optic-sheath subarachnoid space protruding into posterior aspect of globe at optic nerve head (arrows).

in its diagnostic potential as a measure of orbital or cerebral pathology, but also in the secondary effects of disk swelling leading to rapidly progressive blindness in certain cases [10–14].

To understand papilledema in the present context, it is perhaps best to isolate two major categories of swollen disks. The specific pathologic etiology of “optic disk swelling” covers a spectrum extending from true edema due to many acquired causes to the type of swelling observed in increased ICP [4]. It is the latter that is of concern here and that seems to involve in part the simple dilatation of the sheath surrounding the optic nerve terminating in a “ballooning” of the optic nerve head, causing it to protrude into the globe [4, 15–17]. In addition to this protrusion, there may be a variable amount of

associated intercellular fluid (true edema) accumulation within the optic papilla due to one or more factors: primary tissue ischemia [18, 19], an increased pressure gradient between the subarachnoid space and the vitreous of the globe with resultant transarachnoid transudation of fluid, or quite likely an extravascular extravasation of fluid secondary to this pressure gradient (Fig. 8) [18, 20, 21]. In agreement with the latter theory was the evidence of extravascular accumulation of contrast material in the optic disk due to a breakdown in the “blood-nerve barrier” in a patient in the current study who had cranial hypertension secondary to extensive dural venous sinus thrombosis (Fig. 6C). Nevertheless, a significant factor in early disk swelling or papilledema is simple papillary protrusion (Fig. 9).

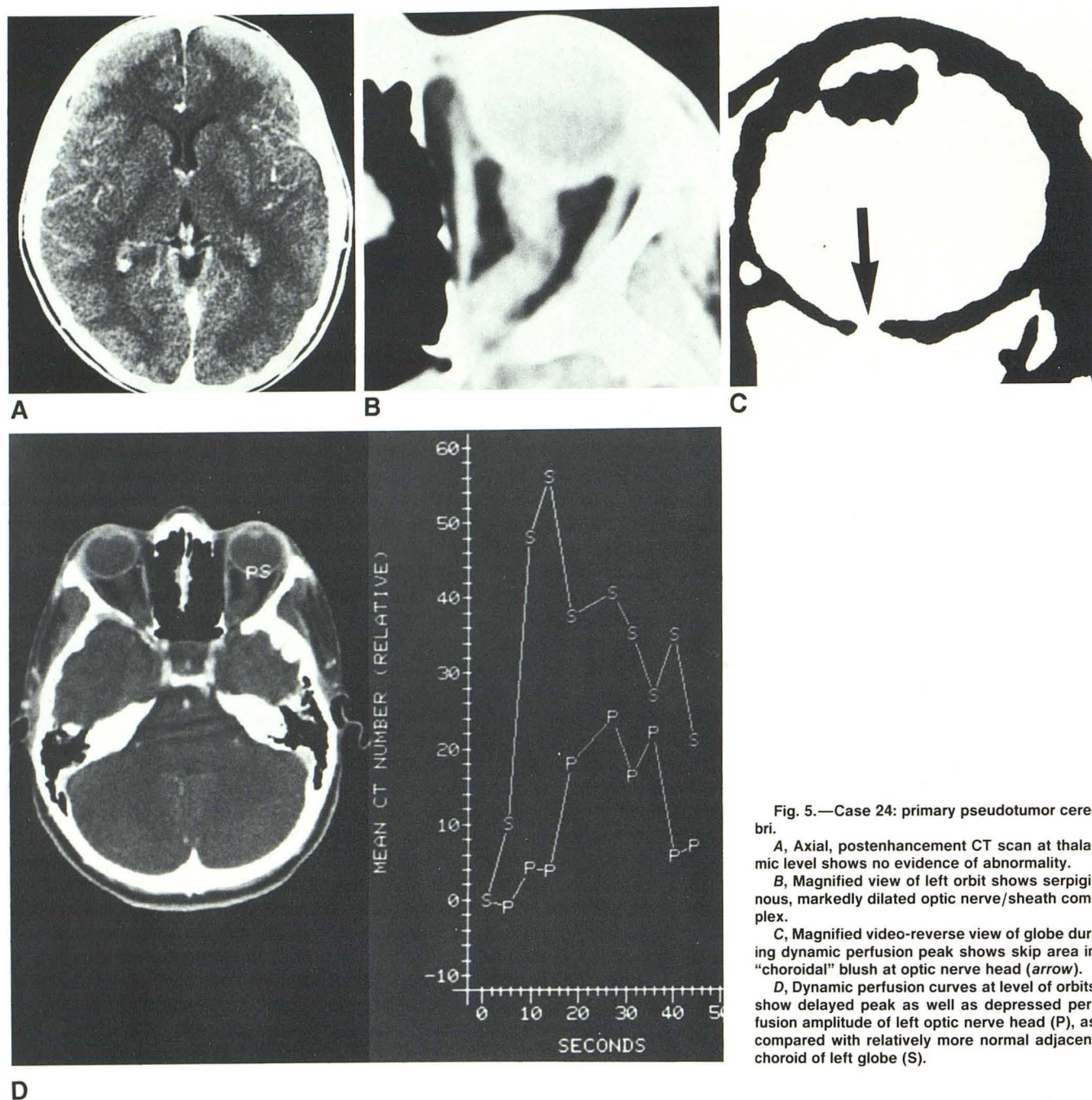


Fig. 5.—Case 24: primary pseudotumor cerebri.

A, Axial, postenhancement CT scan at thalamic level shows no evidence of abnormality.

B, Magnified view of left orbit shows serpiginous, markedly dilated optic nerve/sheath complex.

C, Magnified video-reverse view of globe during dynamic perfusion peak shows skip area in "choroidal" blush at optic nerve head (arrow).

D, Dynamic perfusion curves at level of orbits show delayed peak as well as depressed perfusion amplitude of left optic nerve head (P), as compared with relatively more normal adjacent choroid of left globe (S).

The more serious consideration previously alluded to is the progressive blindness that accompanies papilledema in some cases. Table 1 points out that this decrease of visual acuity is not strictly limited to patients with "benign" cranial hypertension, nor is it bilaterally symmetric. Many theories have been proposed as to the cause of this vision loss; most of them hinge on abnormal pressure relationships and ischemia [16, 22-24]. A most intriguing mystery is why the optic nerve is unusual in its vulnerability to damage from increased ICP. One line of investigation in this regard is to examine why the optic nerve is basically different from the other cranial nerves.

Simplistically, the first cranial nerve is the only one to have its origin at a point still bordering on the subarachnoid space: the optic nerve head. It would seem then that pressure changes in the intracranial space could be transmitted easily to the optic nerve head along the subarachnoid space accompanying the optic nerve sheath. Should this pressure be sufficiently severe, adverse effects would be manifest by degeneration of the optic nerve itself (Fig. 7C).

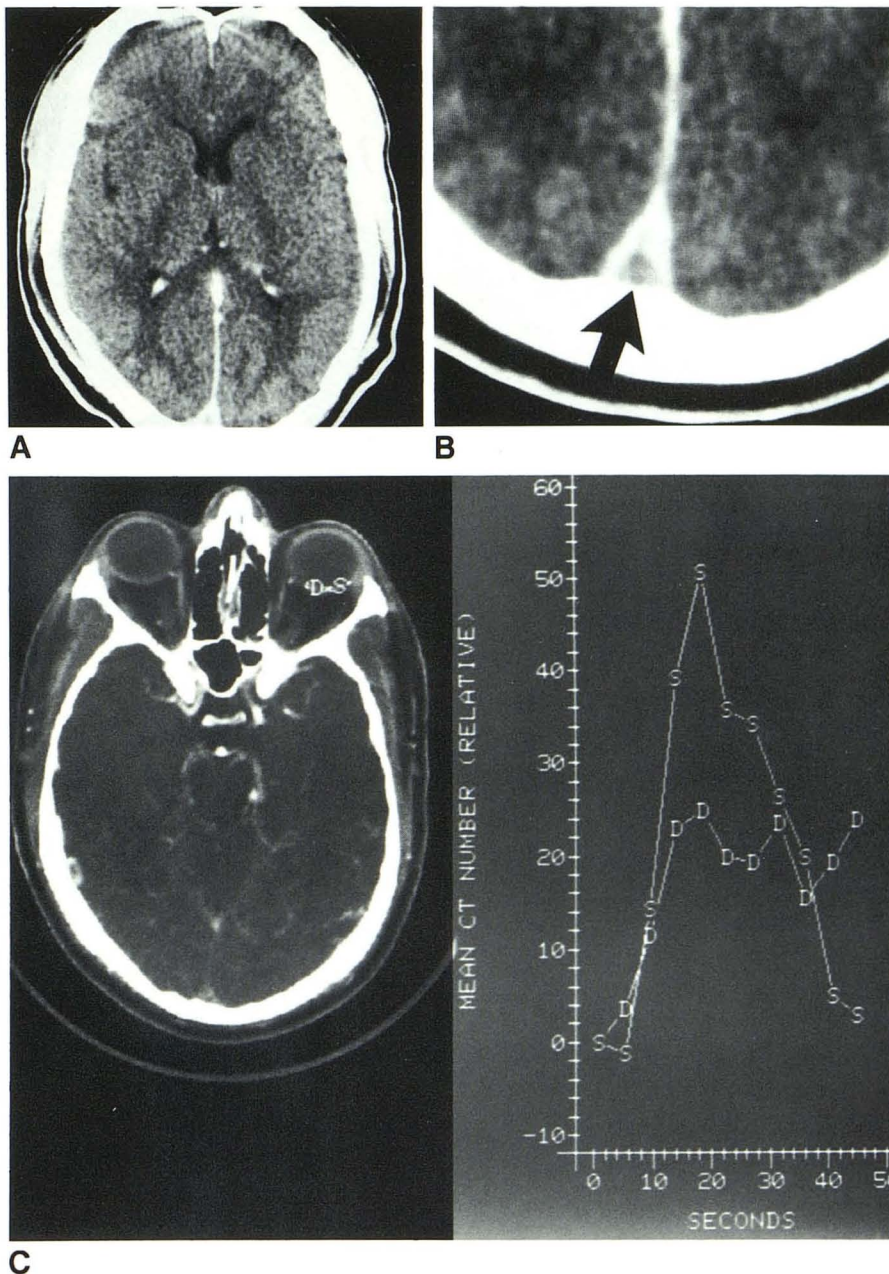
In regard to the sequence of events leading to this projected ischemia, research by others has demonstrated that disk swelling (protrusion) precedes any obvious arterial abnormal-

Fig. 6.—Case 28: secondary pseudotumor cerebri.

A, Postenhancement axial CT shows no evidence of parenchymal nor ventricular abnormality at thalamic level.

B, Magnified view in occipital region shows "delta" sign of sagittal sinus thrombosis (arrow).

C, Dynamic perfusion curve shows depressed perfusion peak followed by elevated plateau phase (D) indicating hypoperfusion of disk and extravascular accumulation of contrast medium due to "blood-nerve barrier" breakdown, respectively, as compared with "normal" adjacent choro-



ity in the optic disk, and that this change (pressure gradient) manifests itself rapidly, appearing within the first 24 hr of increased ICP [21, 25, 26]. In fact, the single instance in the present series of papilledema that did not demonstrate depressed disk perfusion was in a patient harboring a large arteriovenous malformation with acute hydrocephalus and coma of less than 24-hr duration. A postulated aberrant flow state, then, must begin subacutely after an undetermined period of elevated ICP and papillary protrusion [18, 21, 25].

It is necessary, however, to review the circulation of the terminal optic nerve in order to translate this experimental

information into an anatomic explanation. Four main anastomosing sources of perfusion are available to the optic nerve/head junction: the central retinal artery, the short ciliary arteries, the pial vasculature, and the choroidal vessels fed from the ciliary arteries (Fig. 10A) [5, 6, 14]. Despite this interlocking vascular network, the retrolaminar region of the optic nerve is in fact believed by some to be a watershed area and therefore prone to ischemic insult [27-29]. In addition, light microscope studies have demonstrated early abnormal interstitial fluid accumulation from vascular sources in this same region in experimental papilledema, which supports the hy-

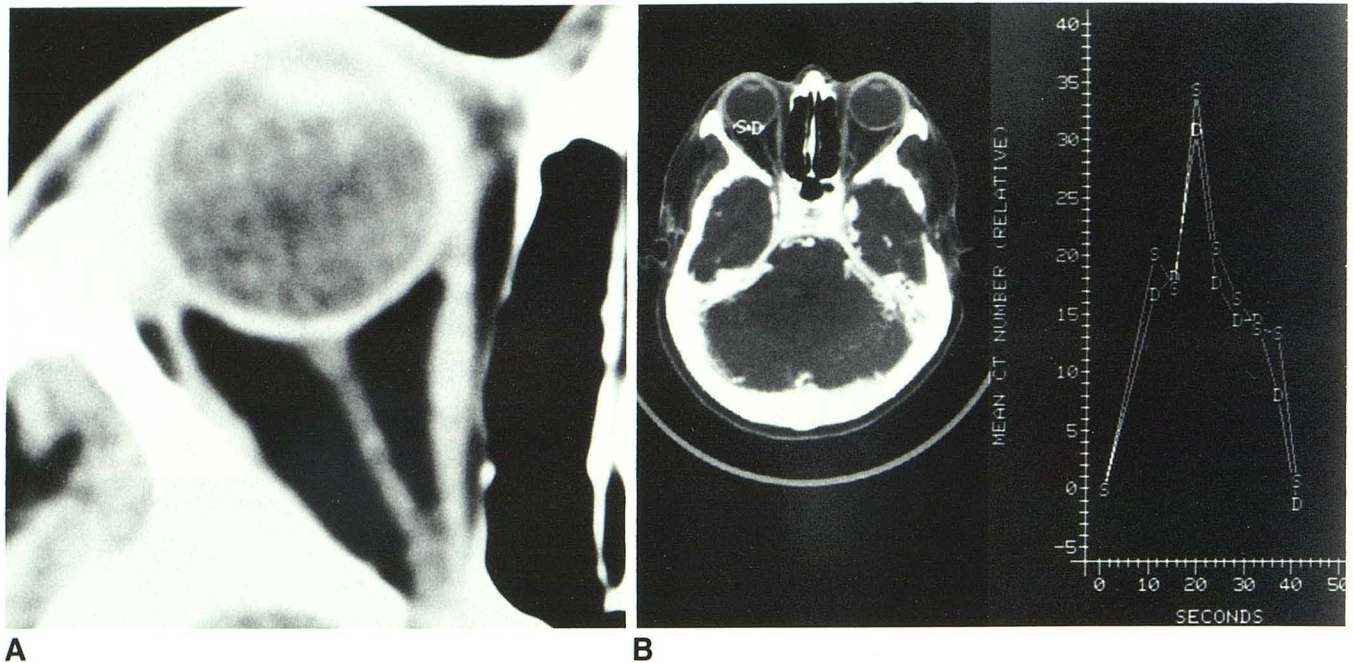


Fig. 7.—Case 9: about 1 year after acute venous sinus thrombosis and after resolution of cranial hypertension.

A, Magnified view of right orbit shows diminutive size of optic nerve/sheath complex, confirming return to normal of intracranial pressure.

B, Dynamic perfusion curve at level of orbits after resolution of papilledema shows identical perfusion curves of right optic nerve head (D) as compared with adjacent choroid (S). The patient had been subtotally blind for about 10 months, but perfusion patterns indicate return to normal state paralleling intracranial pressure.

C, Video-reverse, magnified, postenhancement section shows optic cupping and extreme thinning of disk indicating resultant severe optic atrophy (arrow).

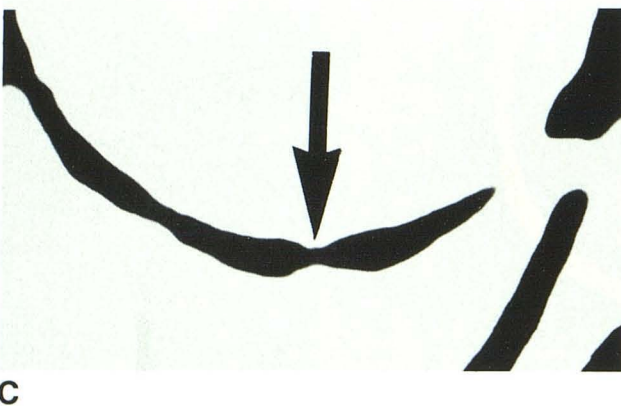


Fig. 8.—Case 19. Video-reverse, magnified postcontrast section shows intrinsic swelling of optic disk (arrows) equating with true papilledema in patient with long-standing hydrocephalus due to midbrain astrocytoma and secondary hydrocephalus.

pothesis of primary insult to this area [20, 30]. It follows that when severe dilatation of the distal optic subarachnoid space occurs, all of the regional neural as well as vascular structures are involved and are compressed and distorted to varying degrees. Applied to the sensitive retrolaminar area and contiguous disk, this mechanical action then presumably compromises flow emanating from all vascular sources as well as the venous drainage, thus leading to stasis, congestion, and the observed depressed dynamic perfusion curves (Fig. 10B) [16, 24, 31].

A third postulate of injury involving primary axonal disarrangement has been observed in vitro by other authors [19, 25] but is a nonspecific finding. The demonstrated axonal swelling might be a response to the increased pressure gradient and axoplasmic stasis, a result of histologic fixation or, alternatively, might be a secondary effect emanating from the preexisting ischemic state with associated axonal trans-

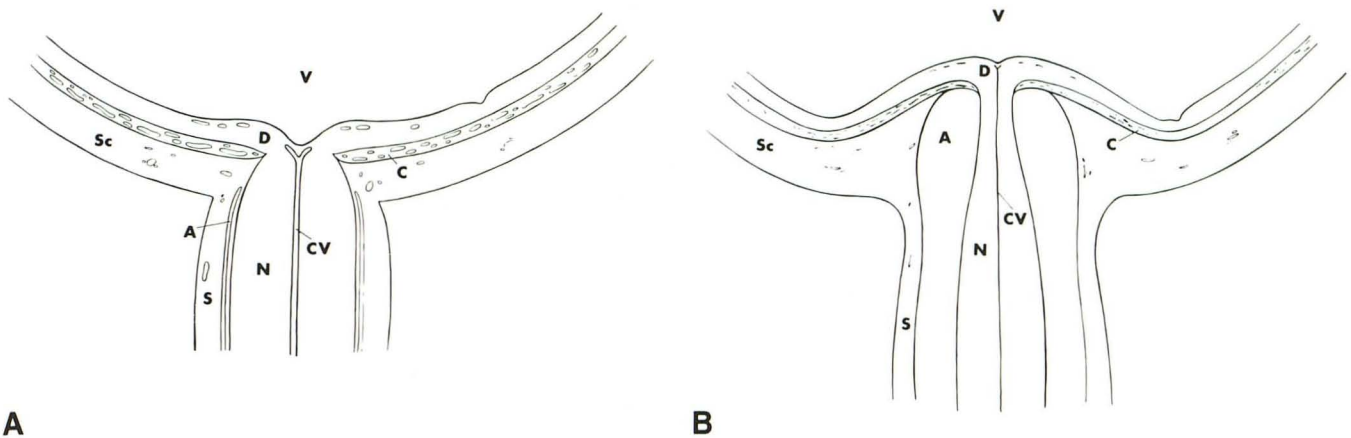


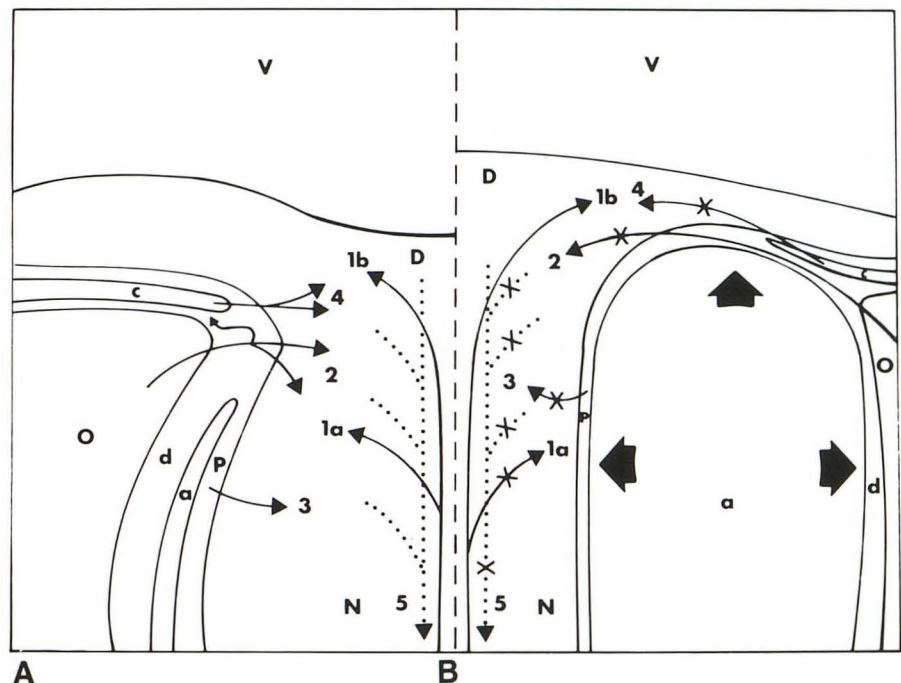
Fig. 9.—A, Normal distal optic nerve/sheath complex and optic nerve head in longitudinal section.

B, Depiction of patient with papilledema shows enlarged subarachnoid

space and bulging optic disk. A = subarachnoid space; C = choroid; CV = central retinal vessels; D = optic disk; N = optic nerve; S = optic sheath; Sc = sclera; V = vitreous of globe.

Fig. 10.—A, Normal vascular supply to left half of terminal optic nerve and head. Arteries (solid arrows), veins (dotted arrows). (Modified from [6].)

B, Configuration of right half of subarachnoid space and optic nerve/head in papilledema postulating compromised perfusion (crosses) due to elevated pressure gradient (short thick arrows) between subarachnoid space and surrounding orbit/vitreous of globe. 1a = central retinal artery in nerve; 1b = central retinal artery in disk; 2 = short ciliary arteries; 3 = pial arterioles; 4 = arterioles from choroid layer; 5 = central retinal vein; a = subarachnoid space; d = dura; D = optic disk; N = optic nerve; O = extravisual orbital contents; P = pia; V = vitreous.



port interference [18, 20, 21]. If indeed these changes are present in vivo, then swelling of the cellular structures as well as the intercellular spaces would further increase the tissue pressures in the optic disk and contiguous nerve, thereby accelerating the anoxic injury.

Regardless of these speculations, early pathologic evidence suggests that up to 35% of the optic nerve fibers may be obliterated in an eye with normal visual field testing, more than 50% with early visual defects, and 90% or more fibers may be nonfunctioning with severe field loss [32]. This has important implications in patients with abnormal pressure gradients at the optic nerve head of variable duration, due to

any cause, but with "normal" visual testing [33]. It indicates that injury to the optic nerve must already be significant by the time visual acuity begins to suffer, and therefore suggests that clinical intervention seems to be warranted as early as possible to correct the damaging aberrant pressure parameters before visual deterioration becomes apparent [10, 12, 14, 34–36].

In summary, patients with increased ICP of any origin often demonstrate optic disk swelling that initially is largely from transmitted pressure along the subarachnoid space accompanying the optic nerve sheath and terminating at the optic nerve head. This results in optic papilla protrusion with at-

tendant depressed perfusion of the optic nerve head, which may be temporary, depending on the degree of injury. The multifactorial insult of direct pressure effects, decreased perfusion, an increase in the inter- and possibly intracellular fluid content, and a disruption in axoplasmic flow/exchange all combine to produce insidiously progressive blindness, leading to optic nerve atrophy. It would seem imperative, therefore, particularly in cases of visually labile cranial hypertension, that the focus clinically should be the immediate reduction of the increased ICP to physiologic levels so that both the perfusion of the optic disk as well as the axonal transport mechanisms can be restored, thereby averting permanent injury to the optic nerve.

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