

Glaucoma After Intraocular Bleed

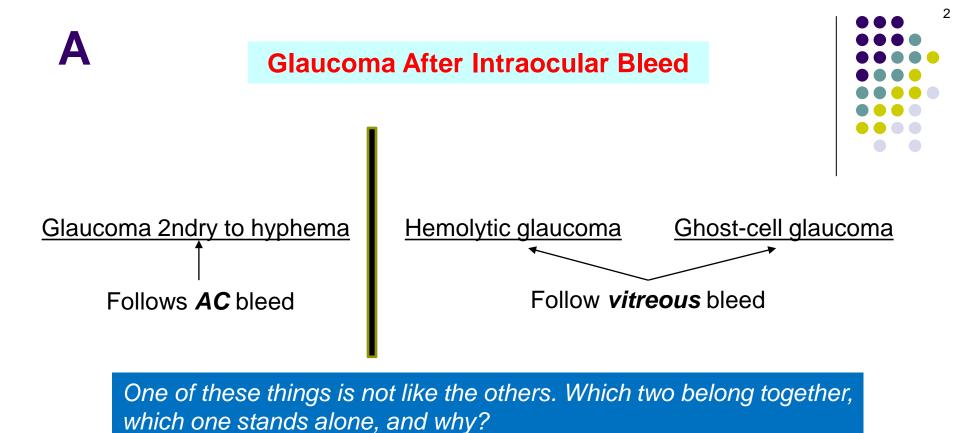


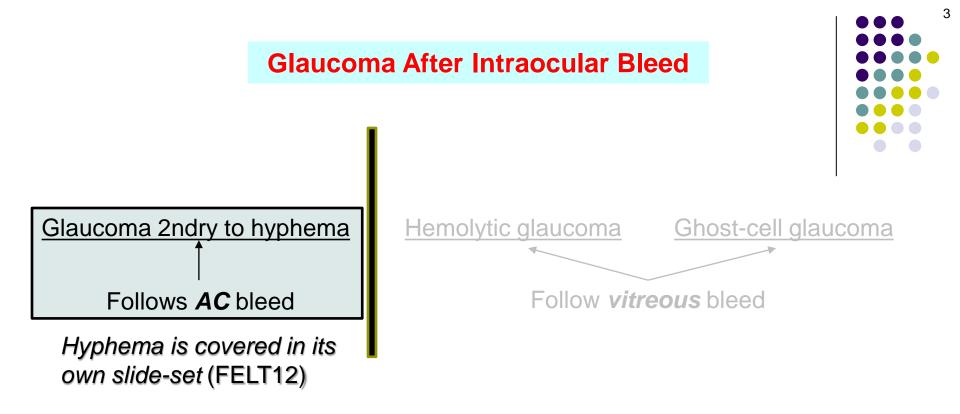
Glaucoma 2ndry to hyphema

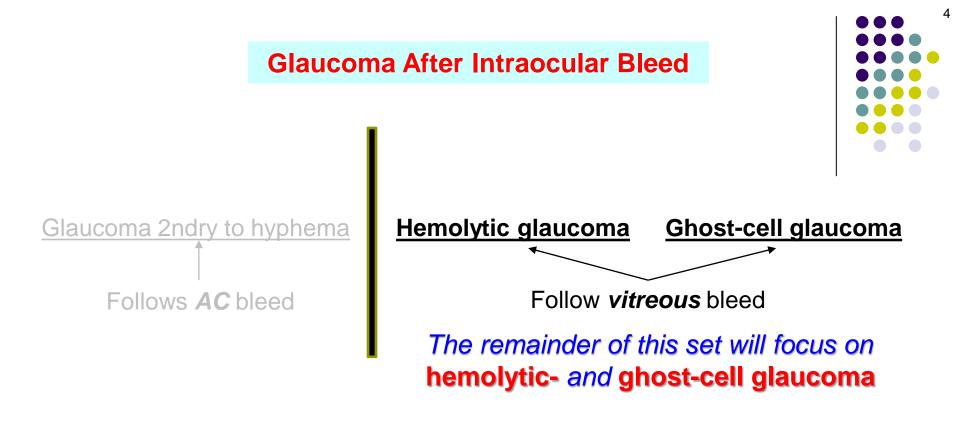
Hemolytic glaucoma

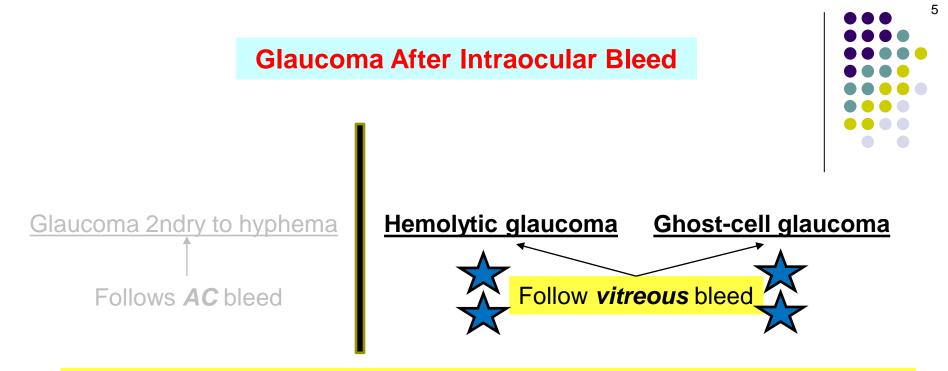
Ghost-cell glaucoma

One of these things is not like the others. Which two belong together, which one stands alone, and why?

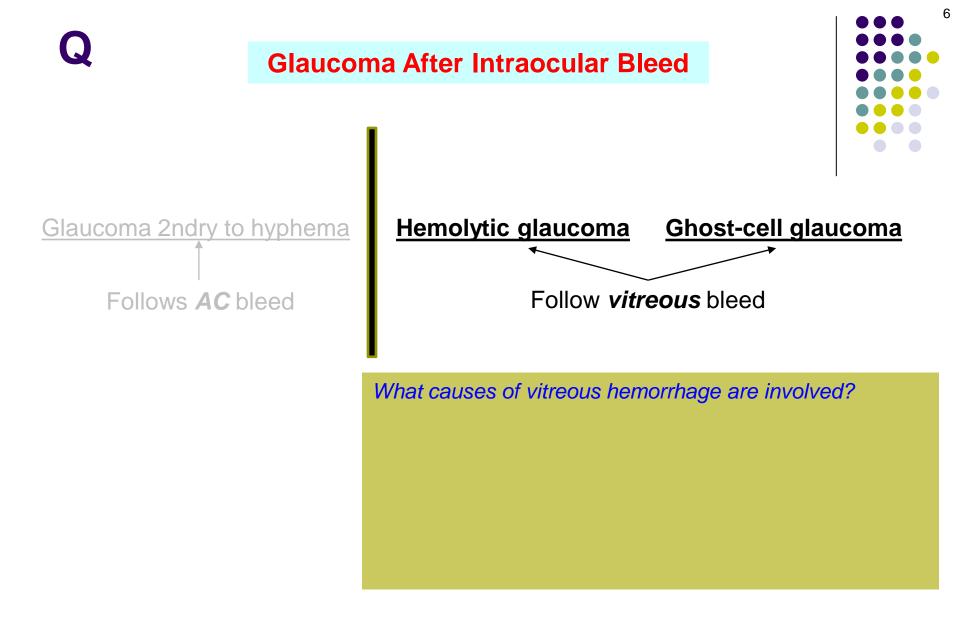


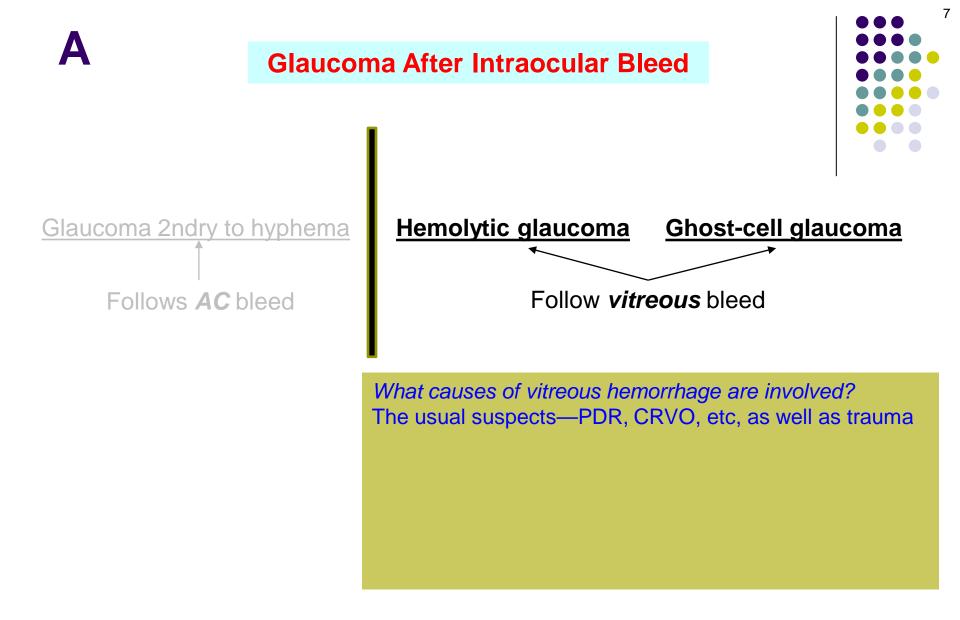


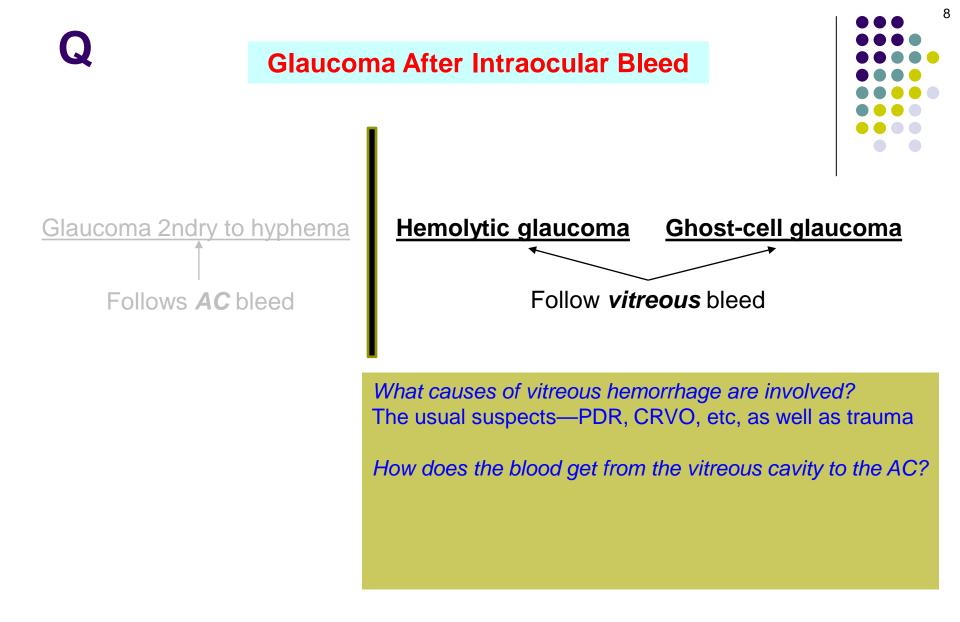


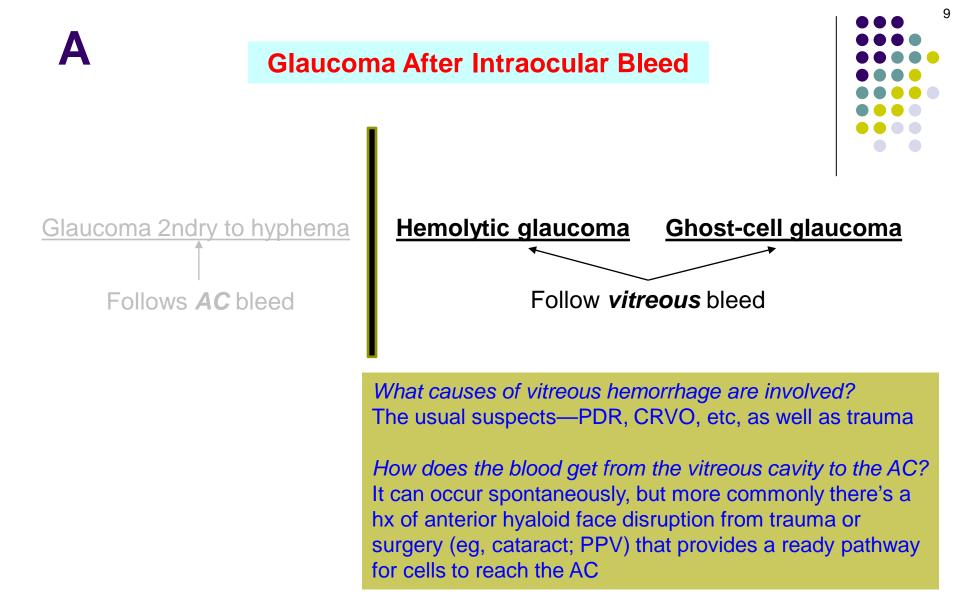


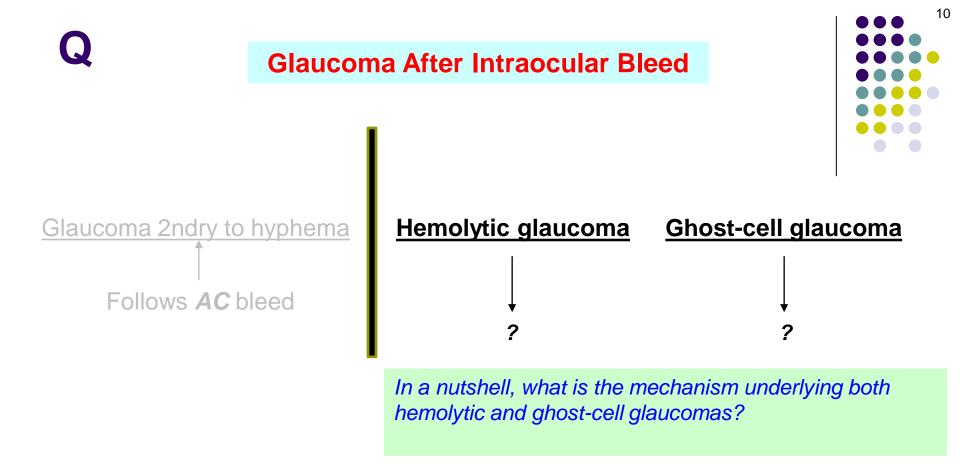
THIS IS IMPORTANT! Take a moment to file a mental note before proceeding: Hemolytic- and ghost-cell glaucoma follow a *vitreous* bleed, not an *AC* bleed!

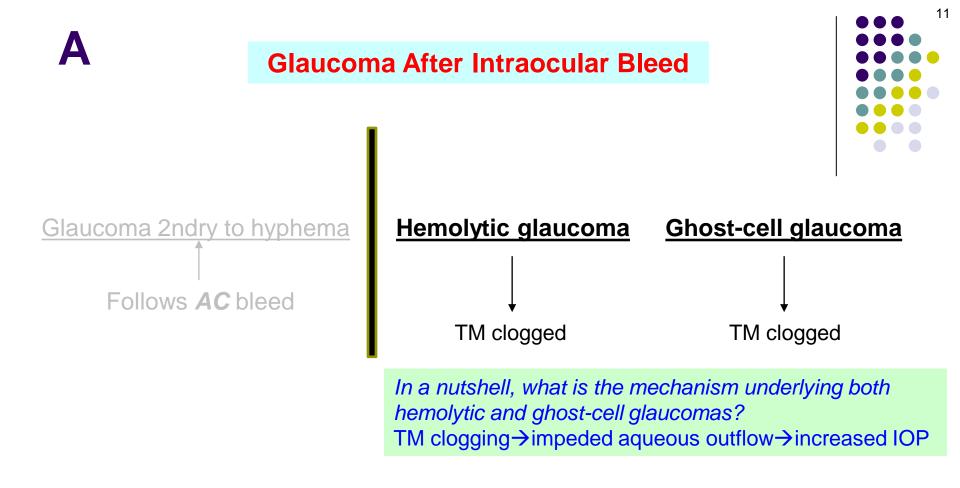


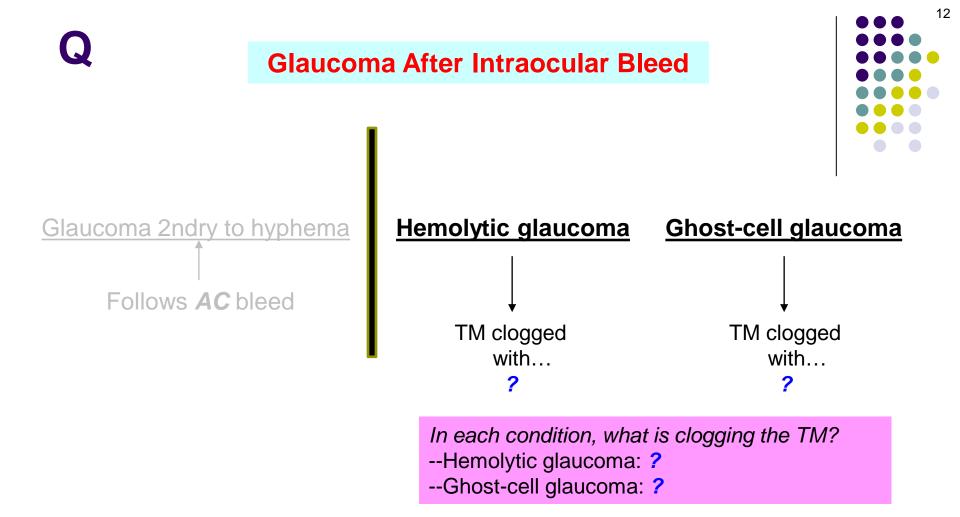


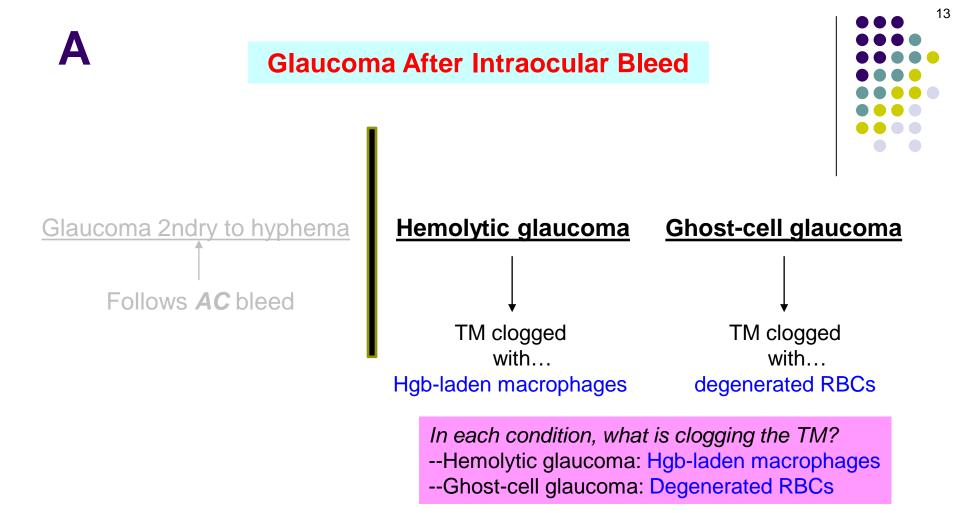


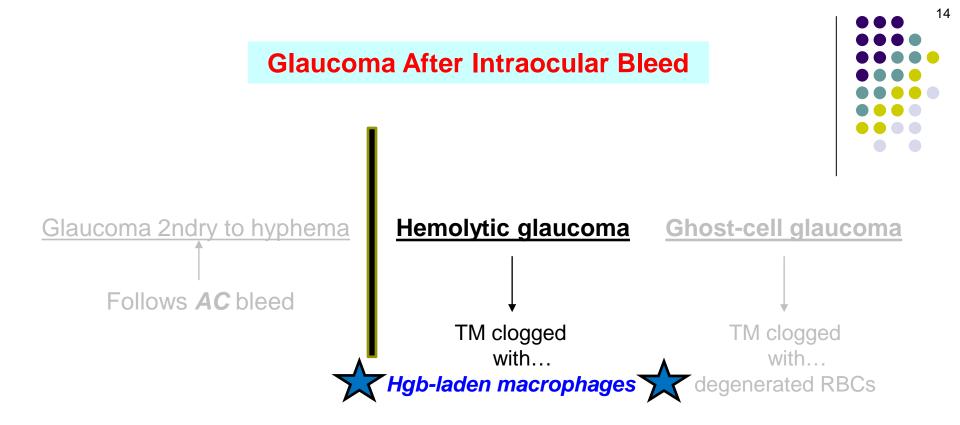




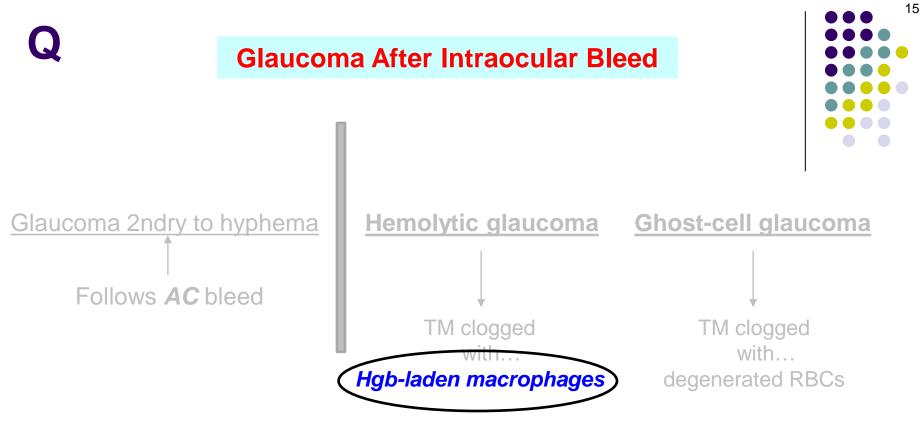




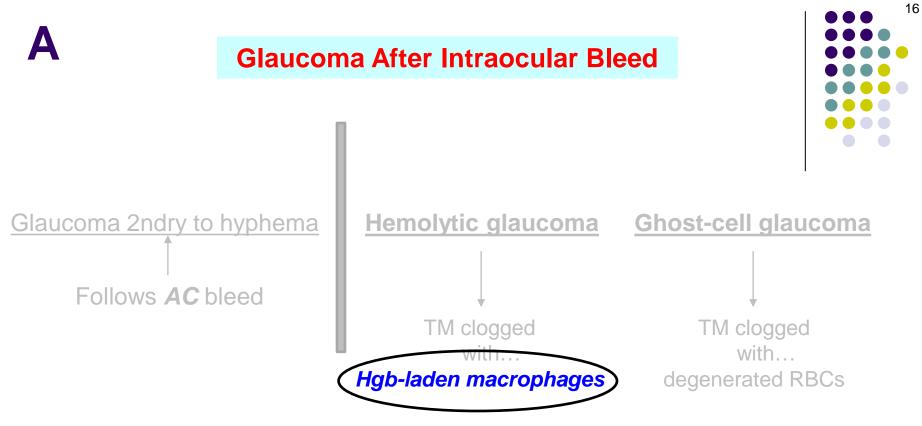




Make another mental note: While ghost-cell glaucoma involves RBCs as would be expected in a hemorrhage-related condition, in hemolytic glaucoma the culprit is not RBCs—it's macrophages



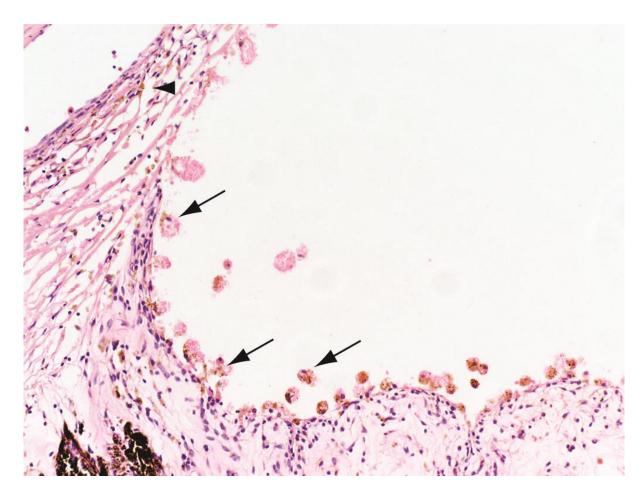
What's up with the macrophages? How do they figure in all this?



What's up with the macrophages? How do they figure in all this?

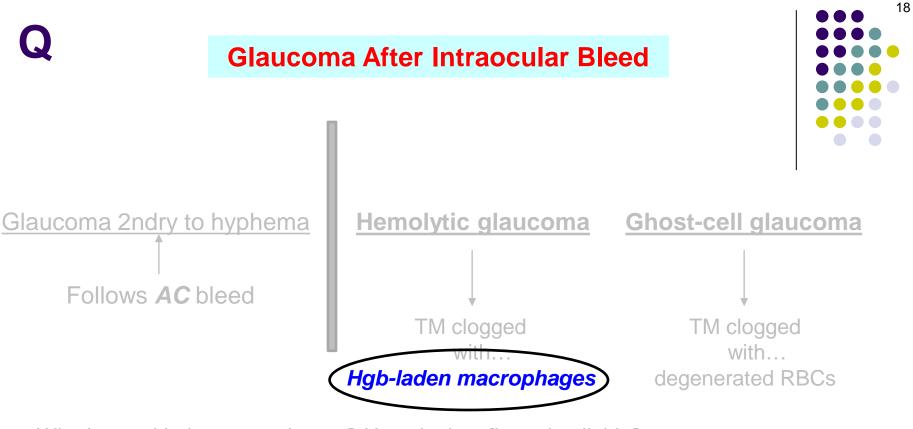
RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts macrophages, which consume both the effete RBCs as well as the hemoglobin-related material they release. Heavy-laden with globules of degenerated Hgb and other RBC detritus, these macrophages end up in the AC, and ultimately the angle.

Glaucoma After Intraocular Bleed

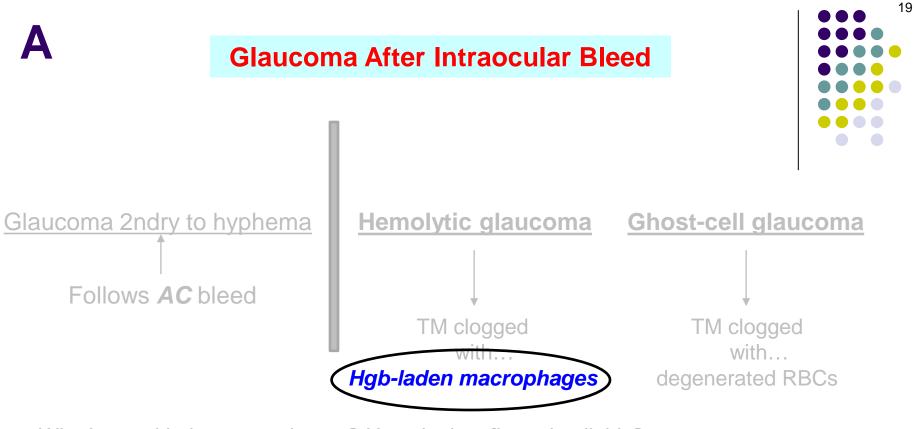


Hemolytic glaucoma. The anterior chamber angle contains macrophages with erythrocytic debris and rust-colored intracytoplasmic material (arrows).



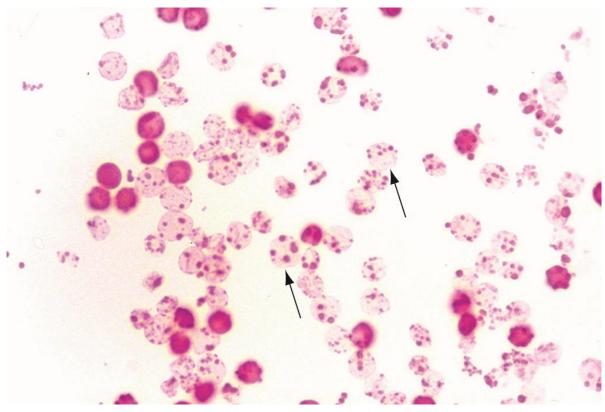


What's up with the macrophages? How do they figure in all this?RBCs in a vitreous hemorrhage start to break down after a week or two. The degenerationof these cells attract'Globules of degenerated Hgb' are knownhemoglobin-relatedby what eponymous name?and other RBC detri



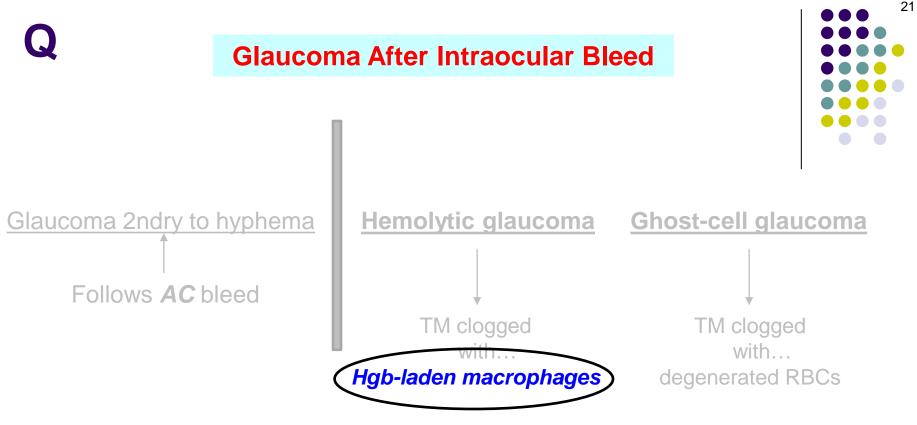
What's up with the macrophages? How do they figure in all this? RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attract *Globules of degenerated Hgb' are known* hemoglobin-related *by what eponymous name?* and other RBC detri Heinz bodies

Glaucoma After Intraocular Bleed



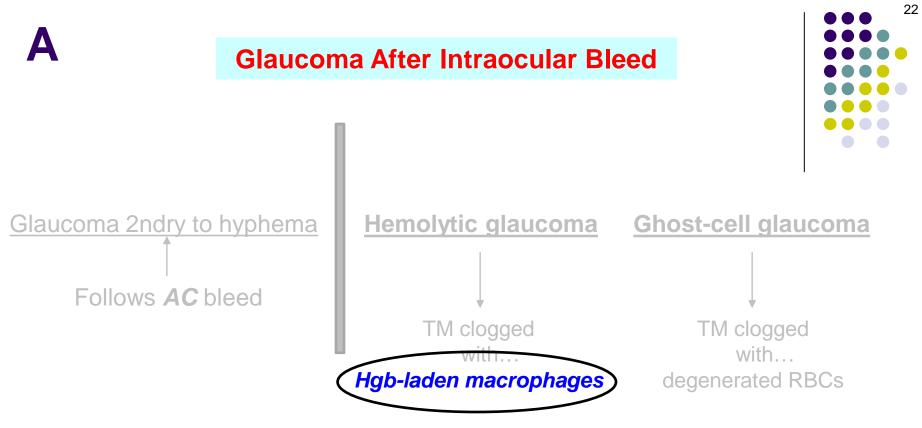
Hemolytic glaucoma. The degenerating hemoglobin is present as small globules known as Heinz bodies (arrows).





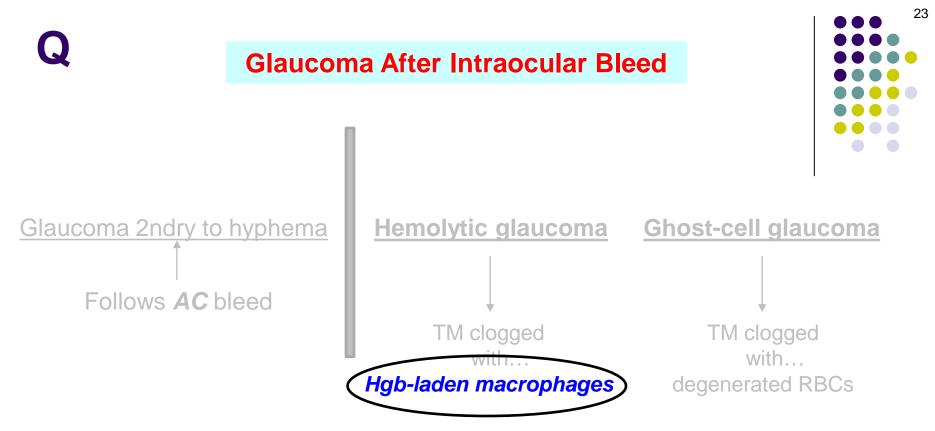
What's up with the macrophages? How do they figure in all this? RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attract hemoglobin-related by what epsnymous name? and other RBC detrice Heinz bodies

'Heinz bodies'? Bruh, the BCSC Glaucoma book does **not** mention Heinz bodies. Why are you including details we don't need to know?



What's up with the macrophages? How do they figure in all this? RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attract hemoglobin-related by what epsnymous name? and other RBC detric Heinz bodies

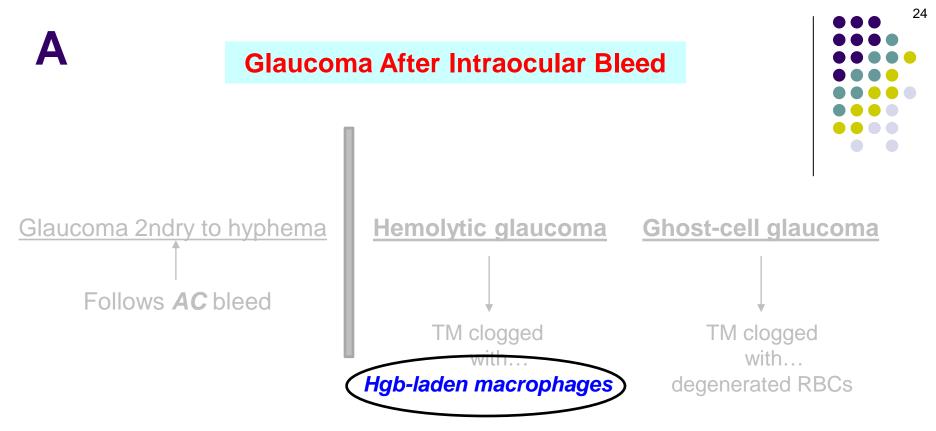
'Heinz bodies'? *Bruh, the BCSC* Glaucoma *book does* **not** *mention Heinz bodies. Why are you including details we don't need to know?* I wouldn't do you like that bruh—the *Pathology* book mentions Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP



What's up with the macrophages? How do they figure in all this? 'Macrophages clogging the TM' should bring to mind another form of 2ndry OAG—what is it?

The degeneration Cs as well as the degenerated Hgb ately the angle.

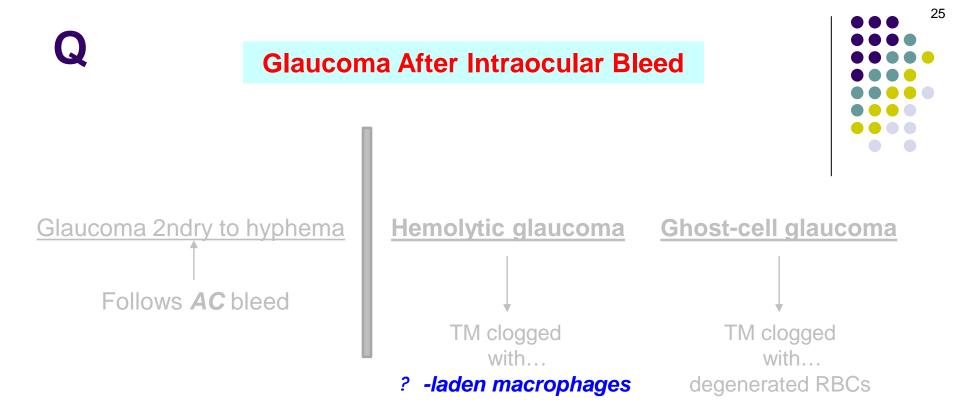
Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP



What's up with the macrophages? How do they figure in all this? 'Macrophages clogging the TM' should bring to mind another form of 2ndry OAG—what is it? Phacolytic glaucoma

Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP

The degeneration Cs as well as the degenerated Hgb ately the angle.

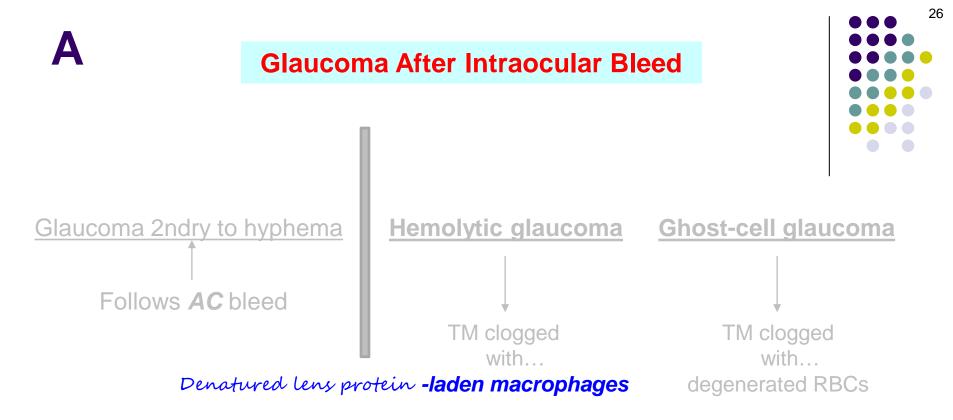


What's up with the macrophages? How do they figure in all this? 'Macrophages clogging the TM' should bring to mind another form of 2ndry OAG—what is it? Phacolytic glaucoma

In phacolytic glaucoma, the macrophages are not heavy-laden with Hgb. Instead, with what substance are they laden?

Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP

The degeneration S as well as the degenerated Hgb ately the angle.

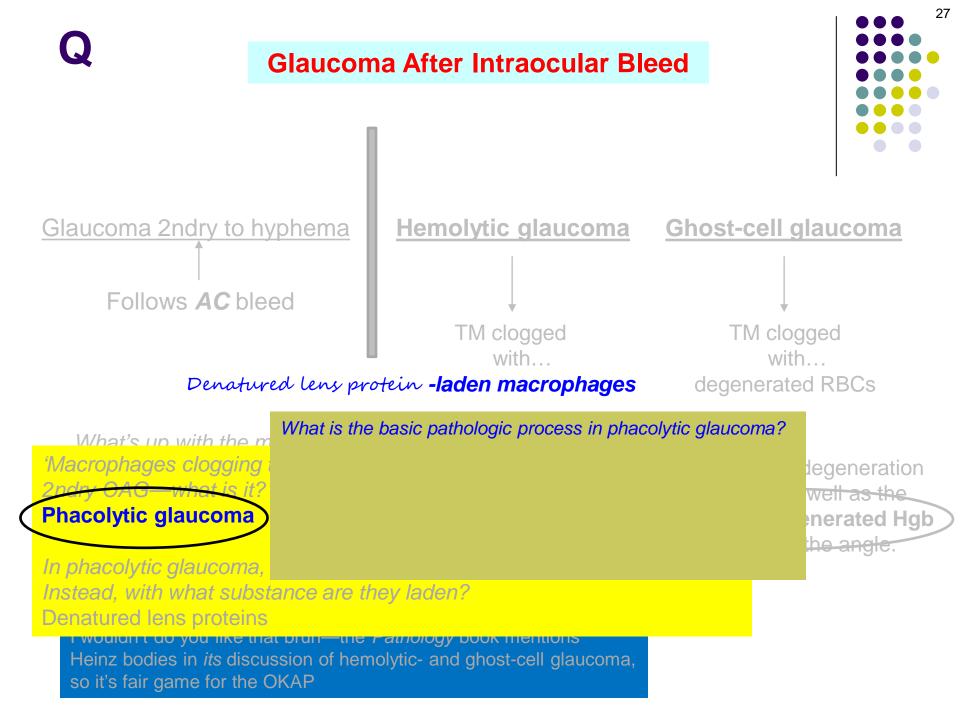


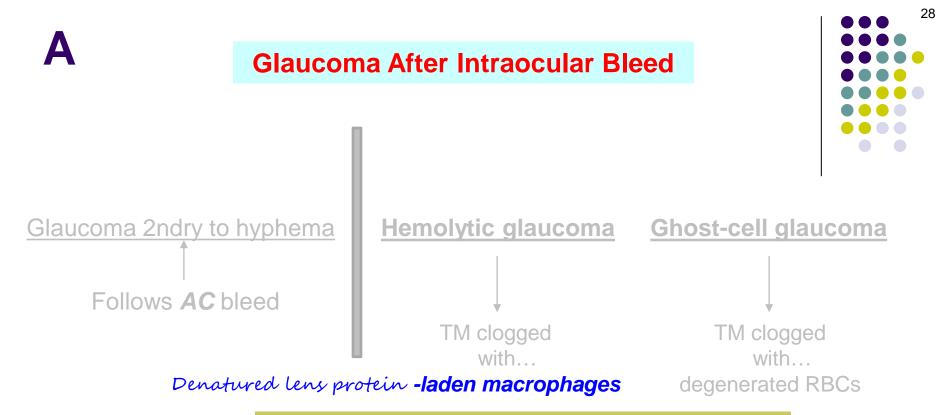
What's up with the macrophages? How do they figure in all this? 'Macrophages clogging the TM' should bring to mind another form of 2ndry OAG—what is it? Phacolytic glaucoma

In phacolytic glaucoma, the macrophages are not heavy-laden with Hgb. Instead, with what substance are they laden? Denatured lens proteins

Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP

The degeneration S as well as the degenerated Hgb ately the angle.







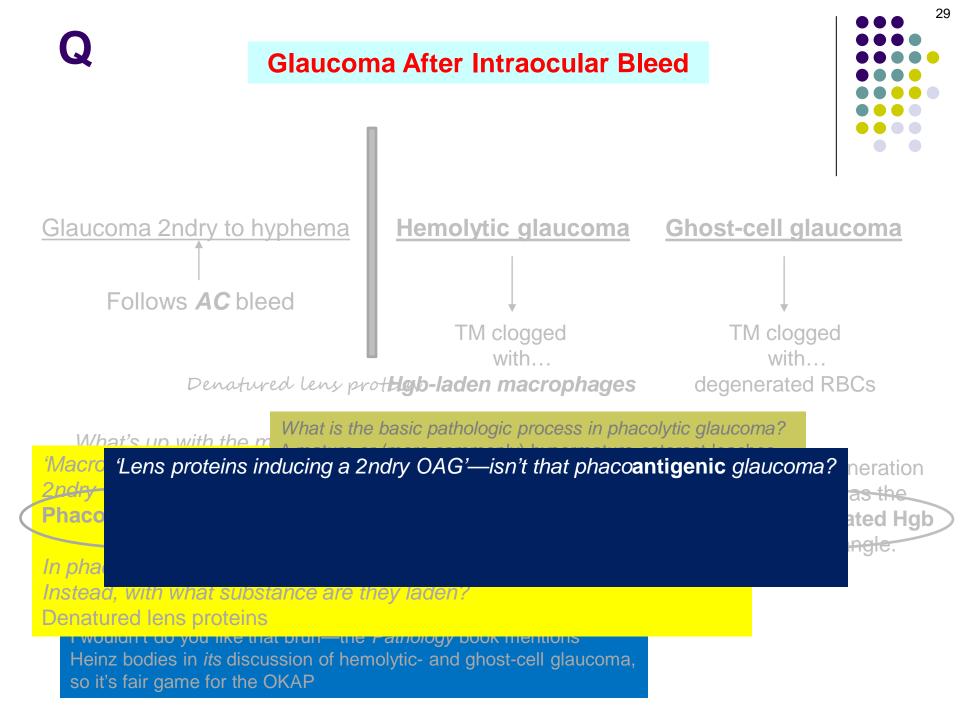
In phacolytic glaucoma,

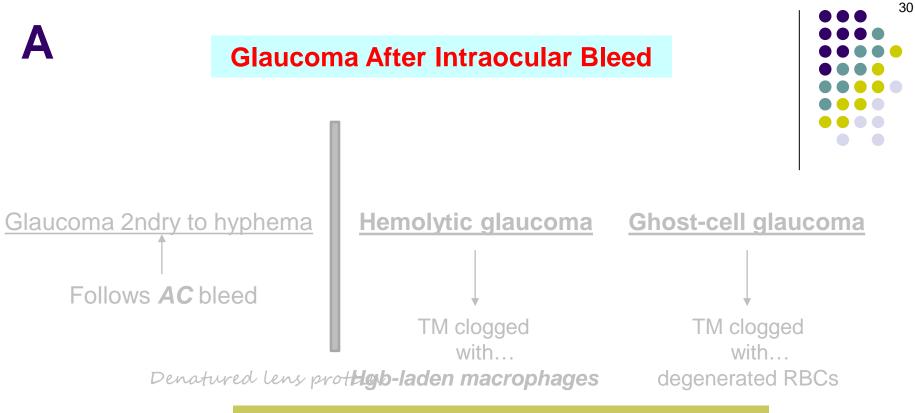
What is the basic pathologic process in phacolytic glaucoma? A mature or (more commonly) hypermature cataract leaches denatured lens proteins through its lens capsule. The proteins attract macrophages, which attempt to clear the protein from the AC. The protein-laden macrophages (and the protein) are swept into the angle, where they clog the TM and cause an IOP spike.

degeneration well as the nerated Hgb the angle.

Instead, with what substance are they laden? Denatured lens proteins

Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP





What is the basic pathologic process in phacolytic glaucoma?

Macro 'Lens proteins inducing a 2ndry OAG'—isn't that phacoantigenic glaucoma? neration No. Phacoantigenic glaucoma is a rare condition in which the appearance 2pdr/ **Phace** of *normal* (ie, not denatured) lens proteins in the AC after a breach in the anterior capsule (either traumatic or surgical) provokes a granulomatous *In pha* immune response.

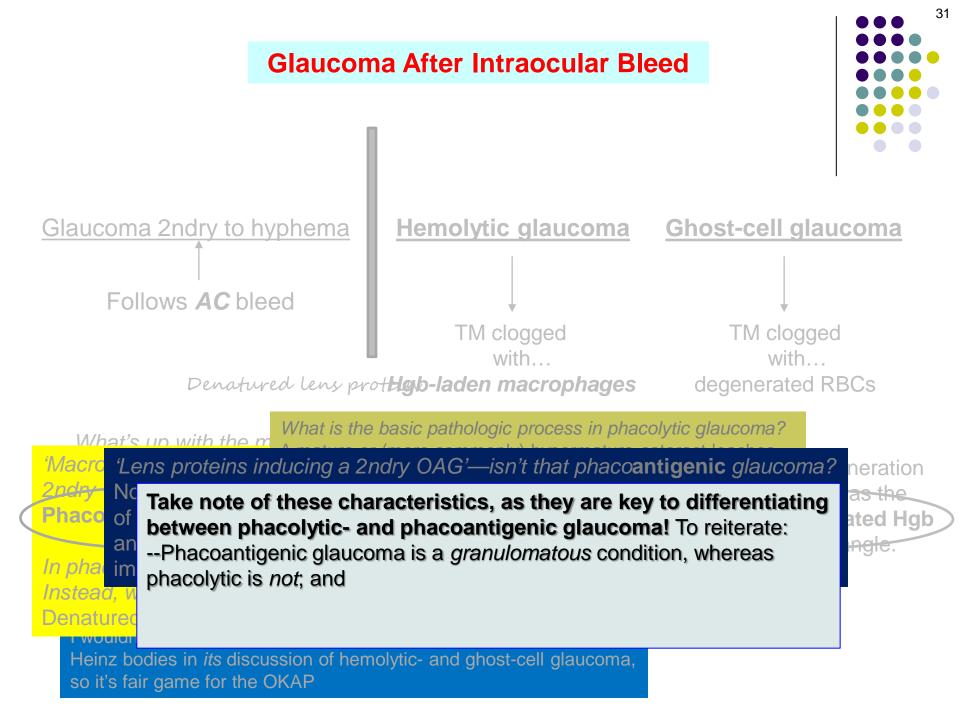
Denatured lens proteins

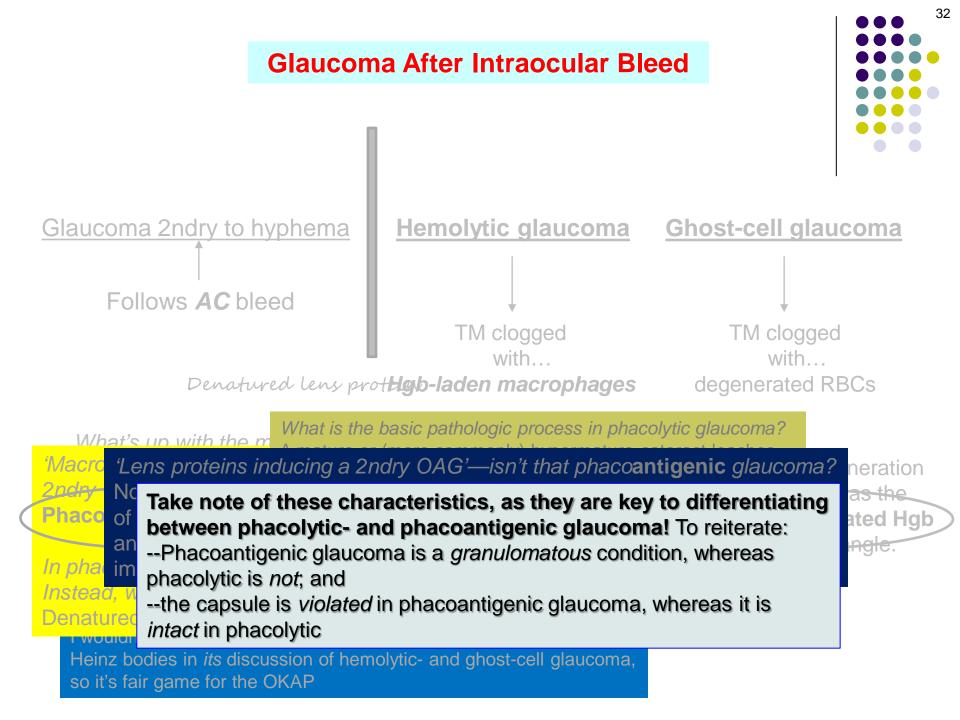
What's up with the r

The that brun-the *Pathology* book mentions Heinz bodies in its discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP

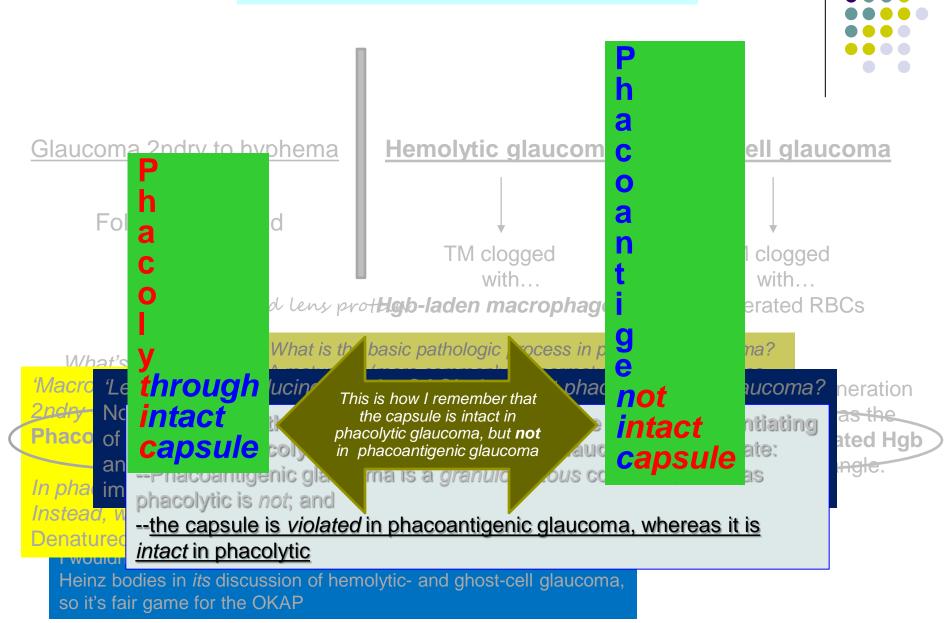
Instead, with what substance are they laden?

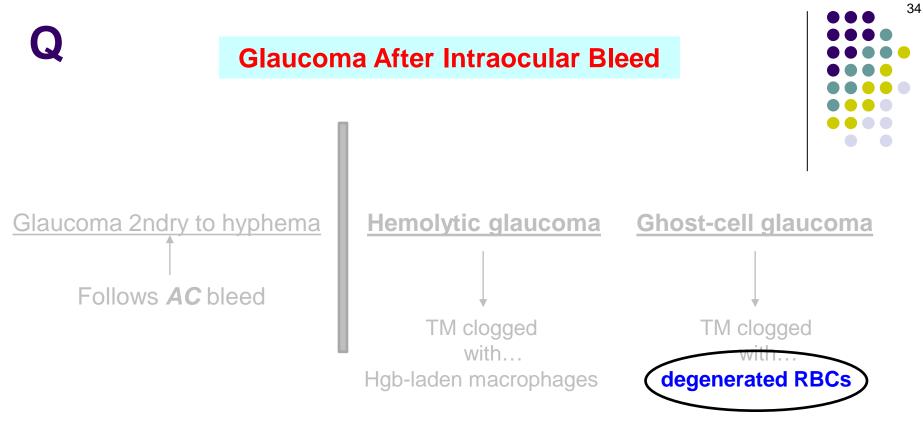
as the ated Hgb ngle.



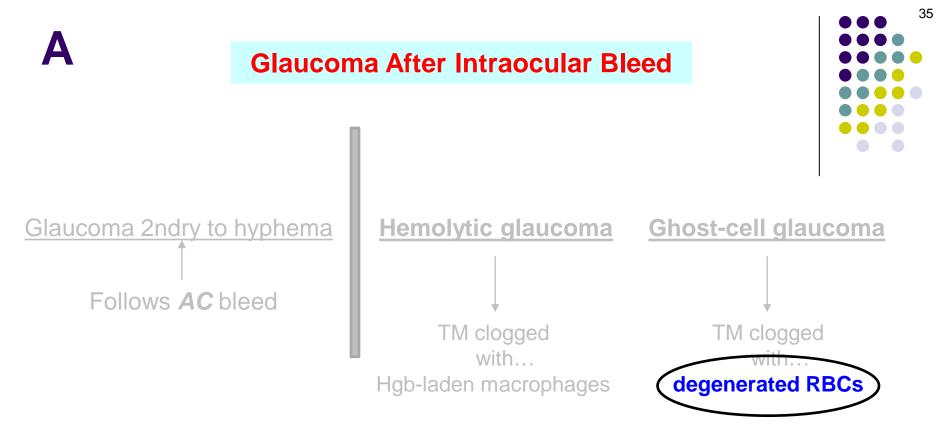


Glaucoma After Intraocular Bleed

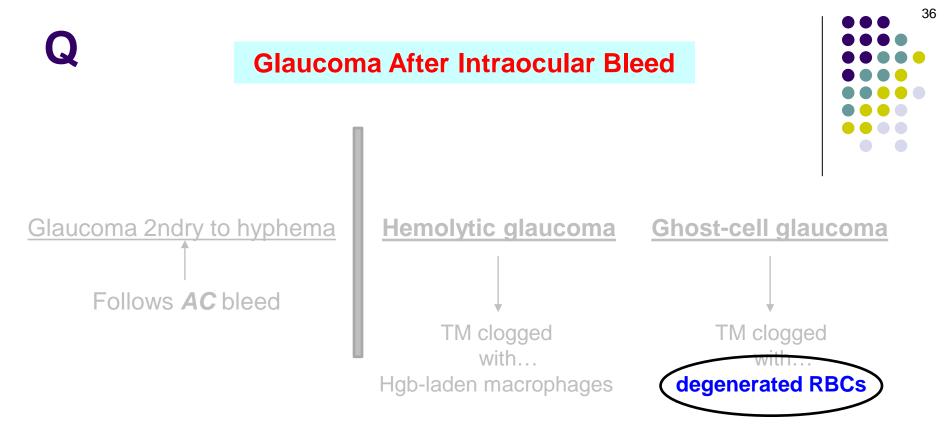




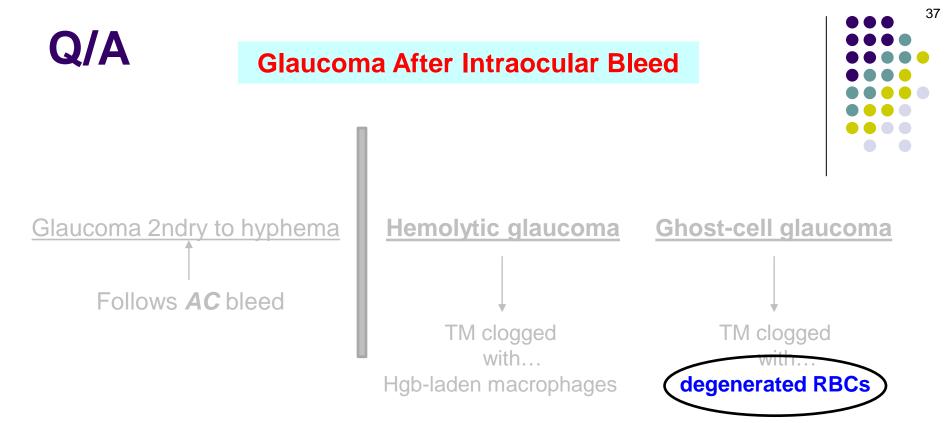
Degenerated RBCs pose a special problem for the TM--why?



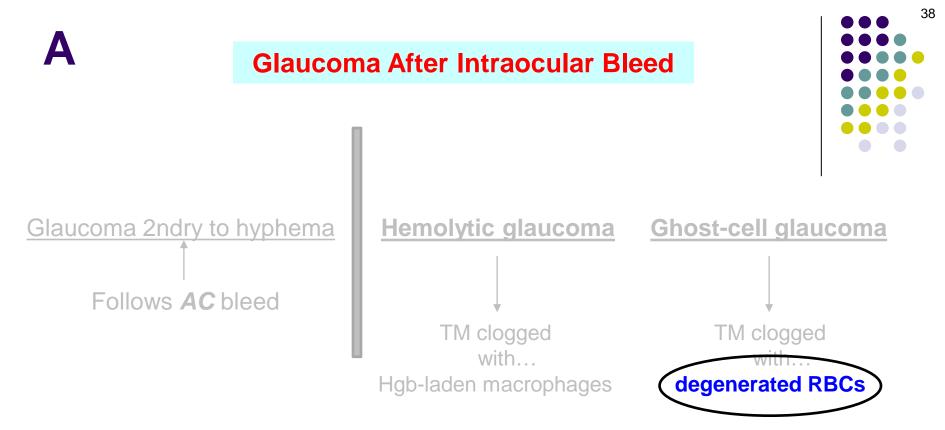
Degenerated RBCs pose a special problem for the TM--why? Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and do not pass easily through it; instead, they pile up in and clog the angle, preventing aqueous egress.



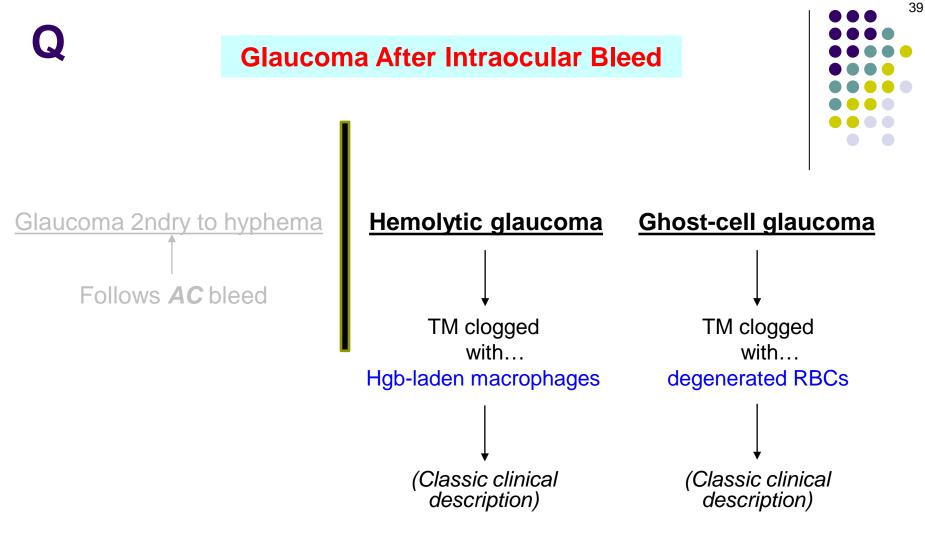
Degenerated RBCs pose a special problem for the TM--why? Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and <u>do not pass easily through it</u>; instead, they pile up in and clog the angle, preventing aqueous egress 'RBCs that do not pass easily through the TM'—what other clinical scenario does that sound like?



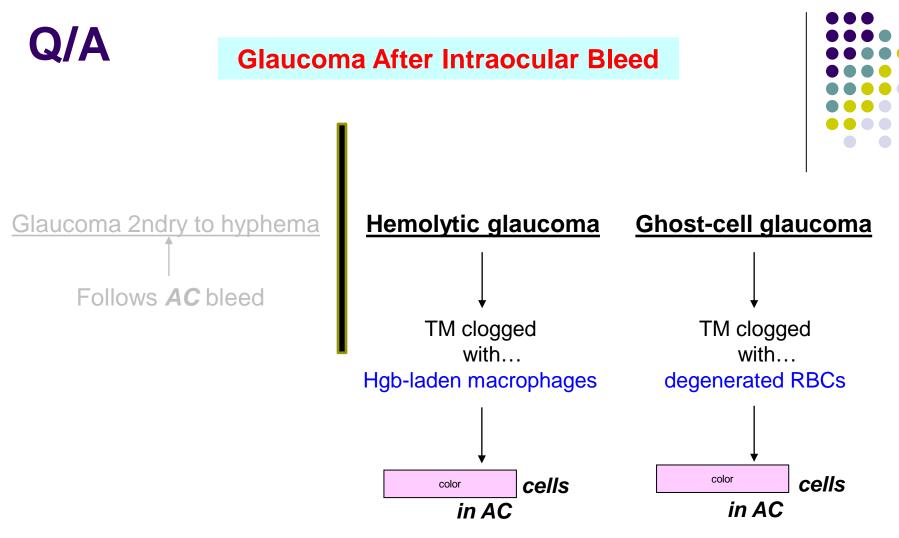
Degenerated RBCs pose a special problem for the TM--why? Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and <u>do not pass easily through it</u>; instead, they pile up in and clog the angle, preventing aqueous egress 'RBCs that do not pass easily through the TM'—what other clinical scenario does that sound like? Hyphema in a sickle-cell pt. Recall that the relatively basic v aqueous promotes RBC sickling. Sickled RBCs are significantly stiffer, and thus unable to pass easily through the TM.



Degenerated RBCs pose a special problem for the TM--why? Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and <u>do not pass easily through it</u>; instead, they pile up in and clog the angle, preventing aqueous egress 'RBCs that do not pass easily through the TM'—what other clinical scenario does that sound like? Hyphema in a sickle-cell pt. Recall that the relatively acidic nature of aqueous promotes RBC sickling. Sickled RBCs are significantly stiffer, and thus unable to pass easily through the TM.

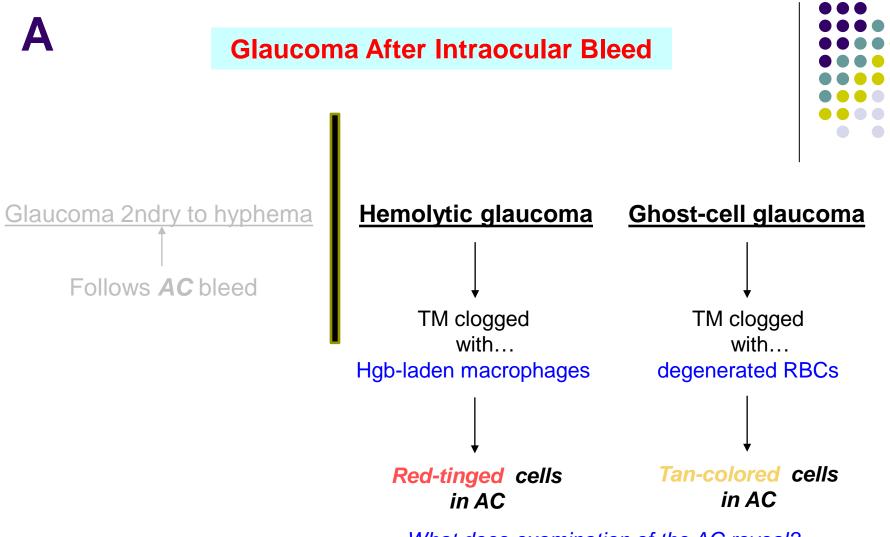


What does examination of the AC reveal?



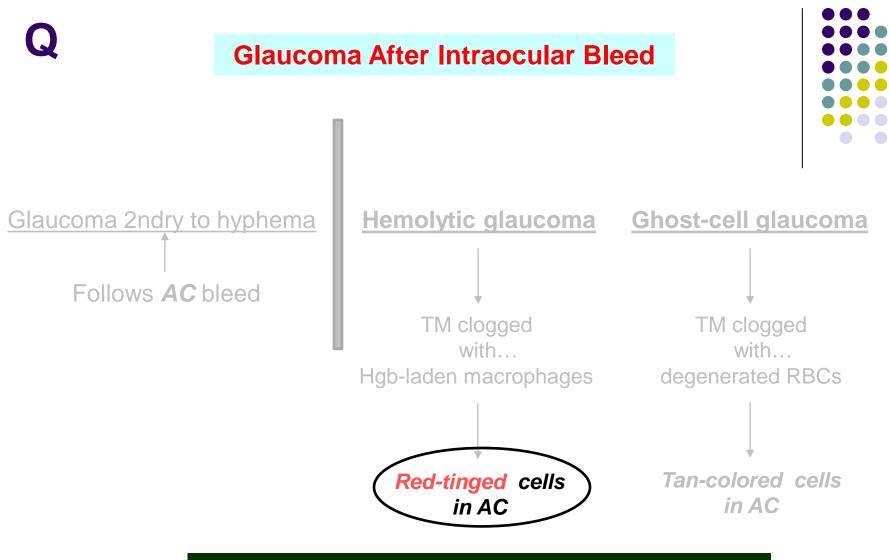
What does examination of the AC reveal?

40



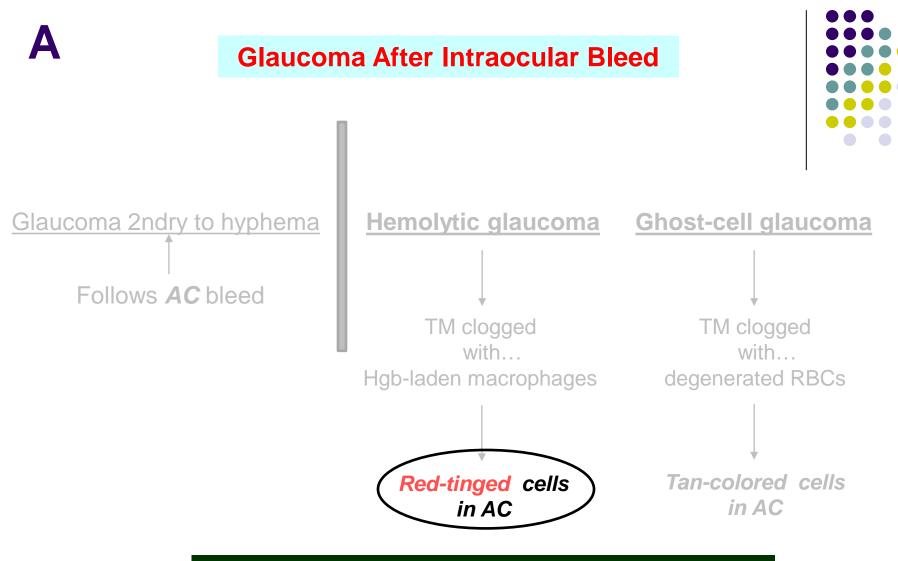
What does examination of the AC reveal?

41



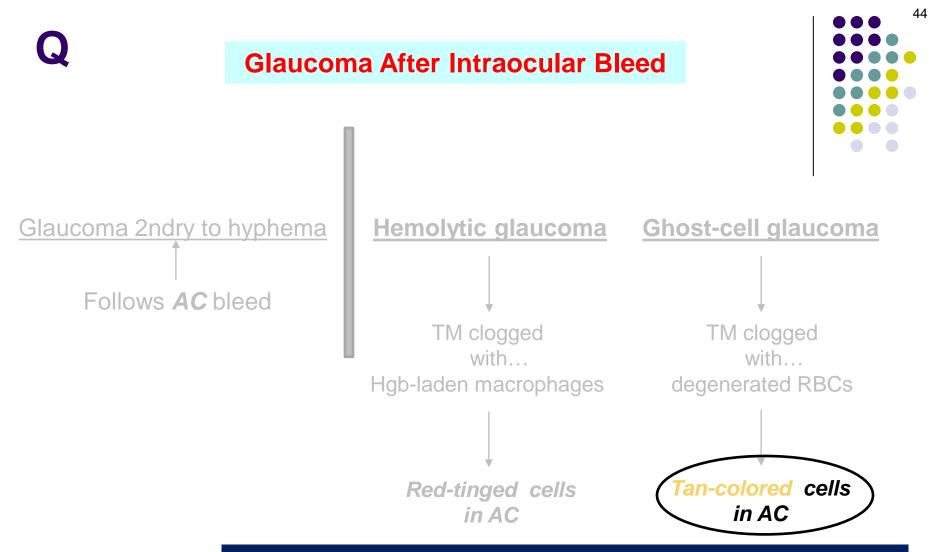
42

Would these 'red-tinged cells' be Hgb-laden macrophages?

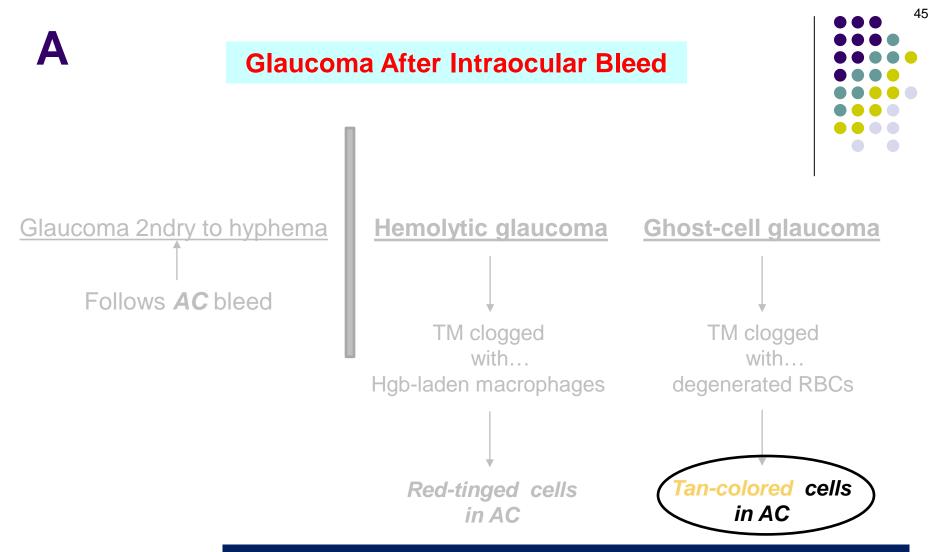


43

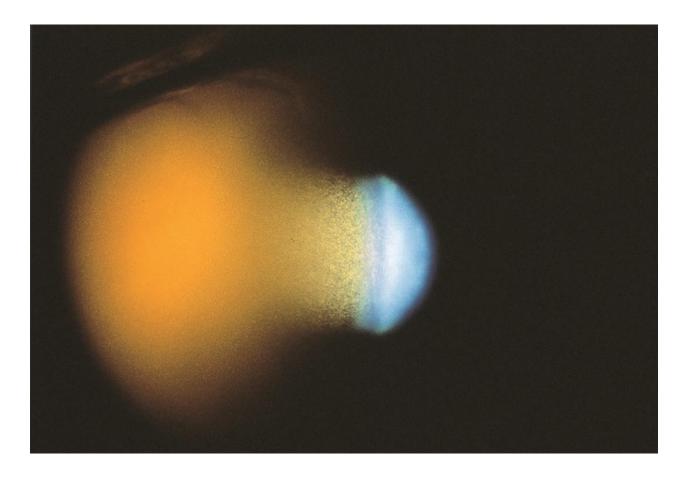
Would these 'red-tinged cells' be Hgb-laden macrophages? Indeed they would



Would these 'tan-colored cells' be the ghost cells after which the condition was named?

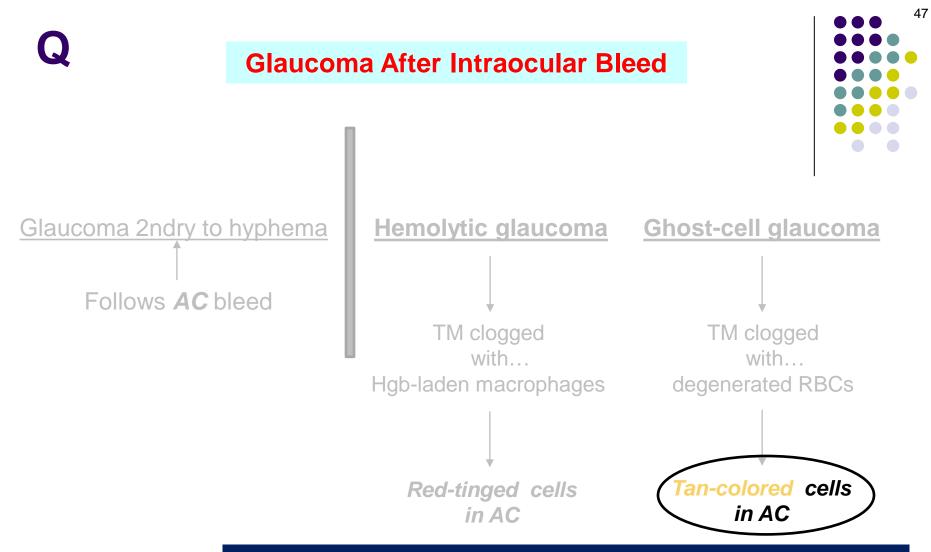


Would these 'tan-colored cells' be the ghost cells after which the condition was named? Indeed they would



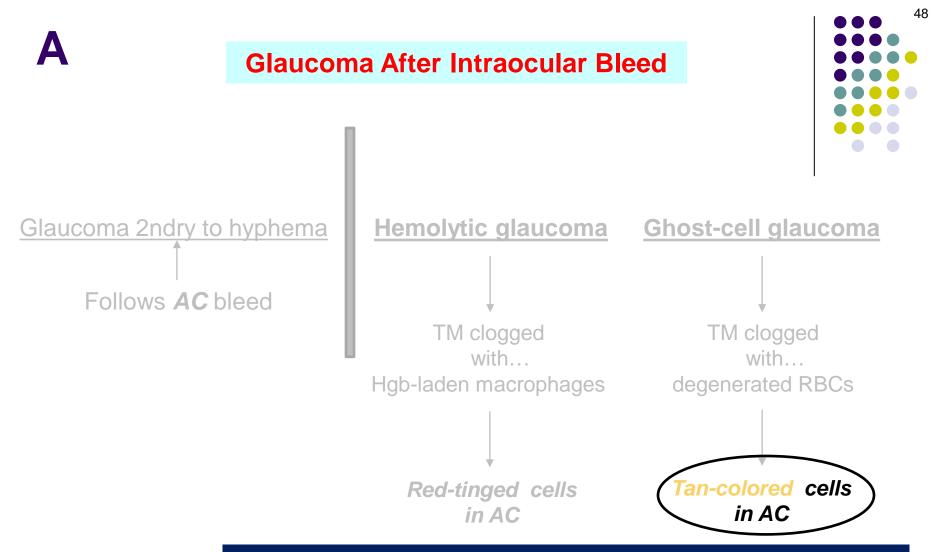
Ghost-cell glaucoma. Copious tan-colored cells in the AC.





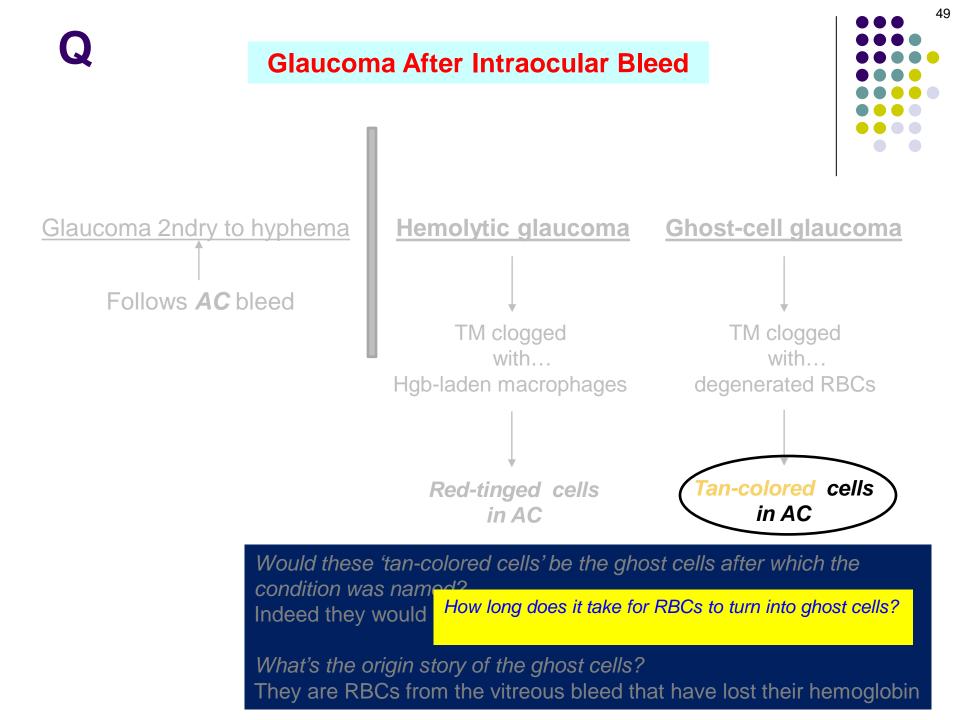
Would these 'tan-colored cells' be the ghost cells after which the condition was named? Indeed they would

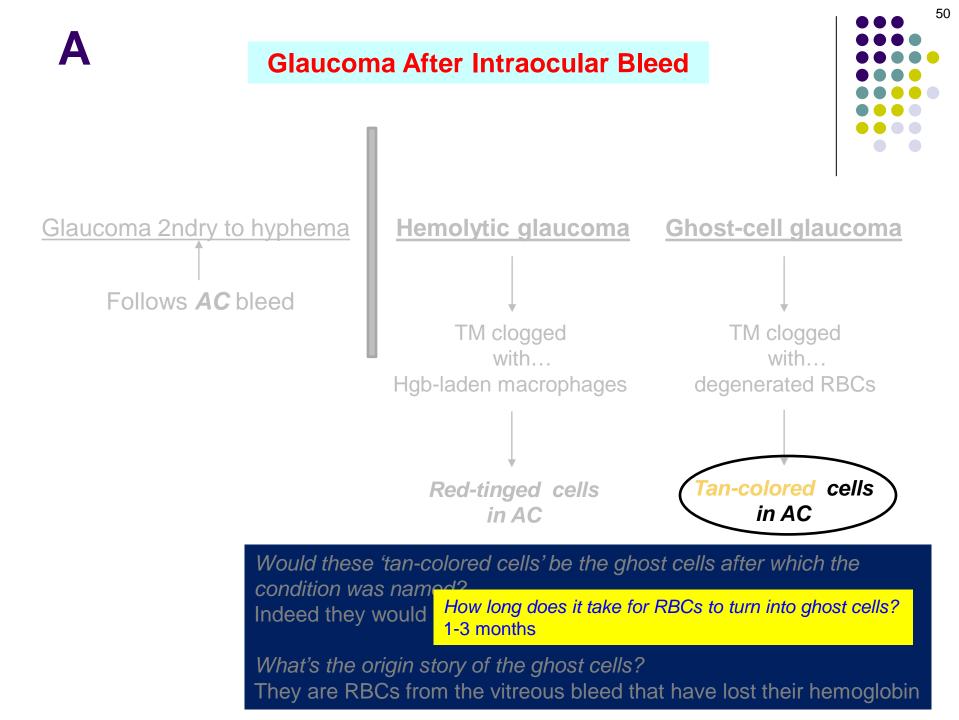
What's the origin story of the ghost cells?

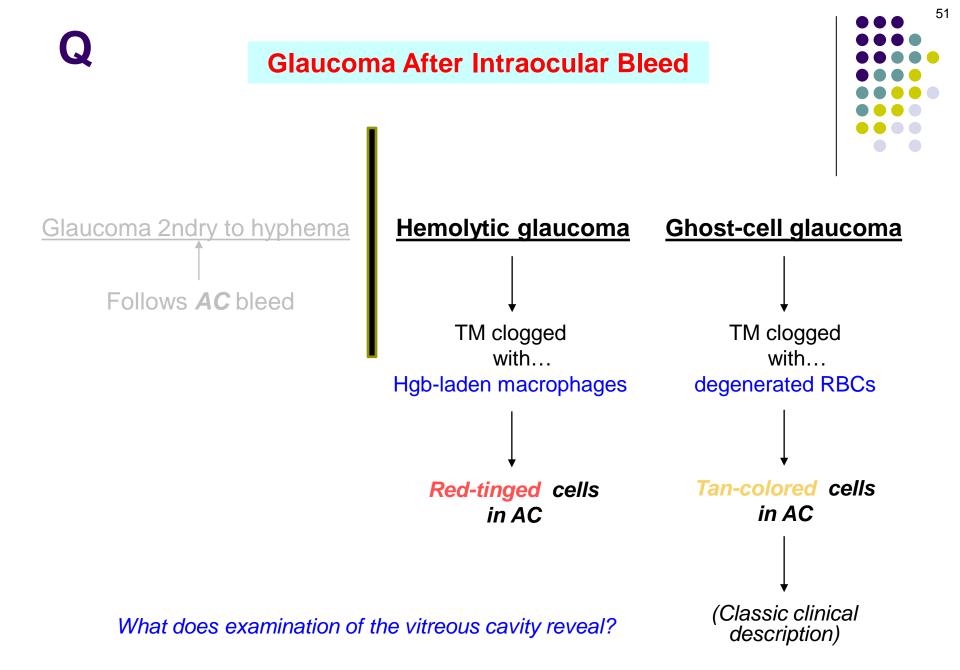


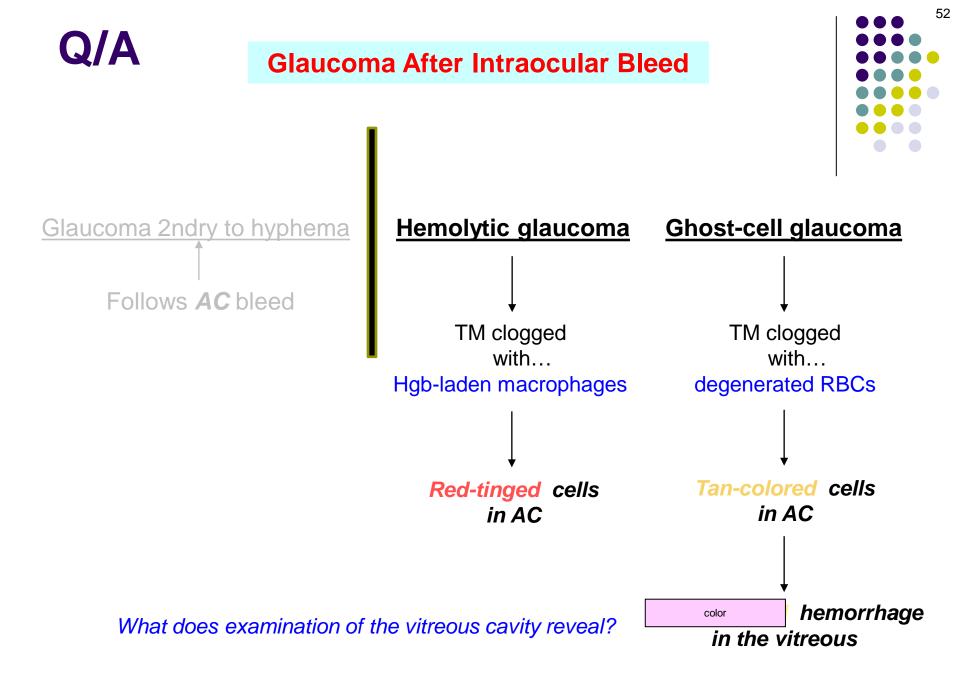
Would these 'tan-colored cells' be the ghost cells after which the condition was named? Indeed they would

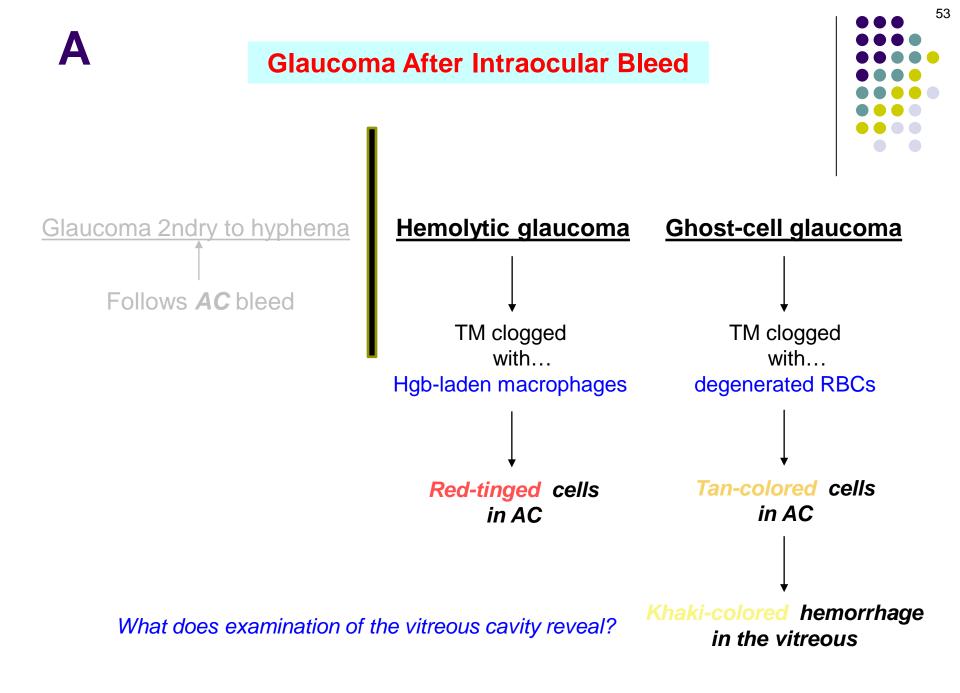
What's the origin story of the ghost cells? They are RBCs from the vitreous bleed that have lost their hemoglobin

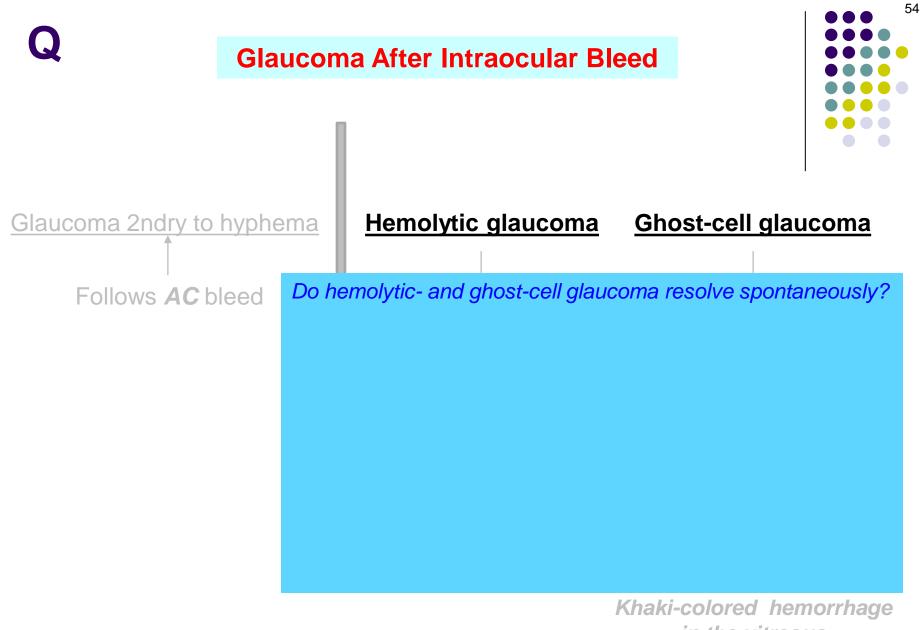




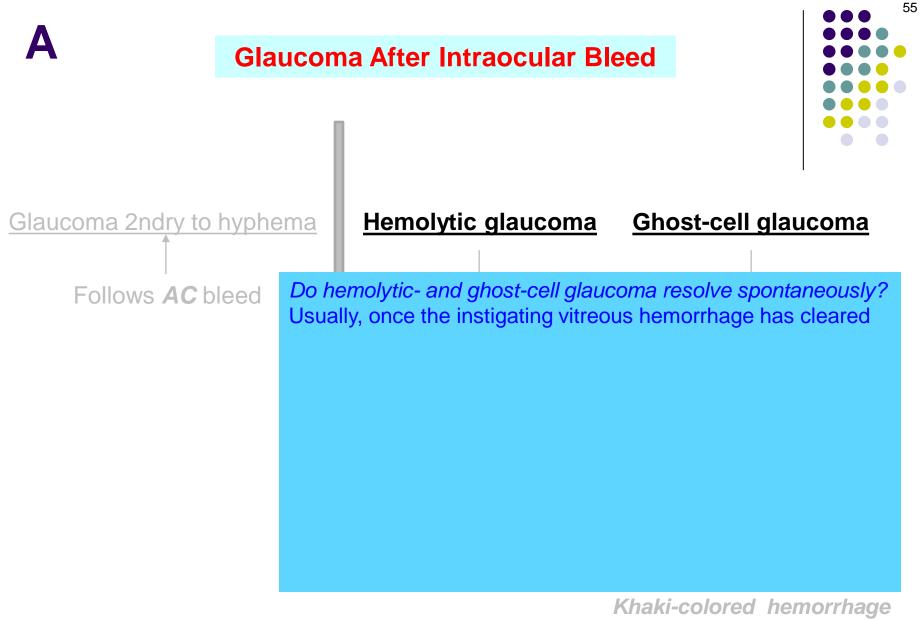




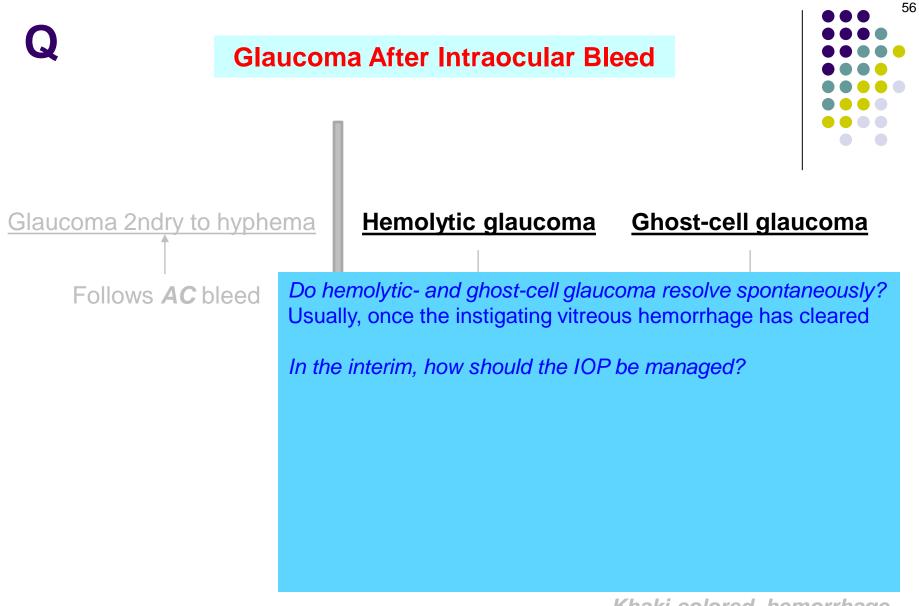




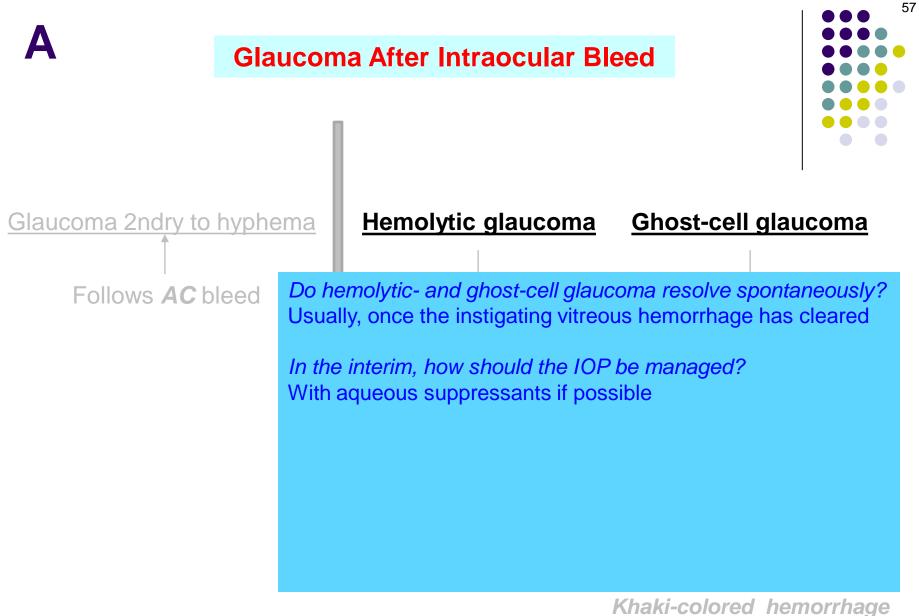
in the vitreous



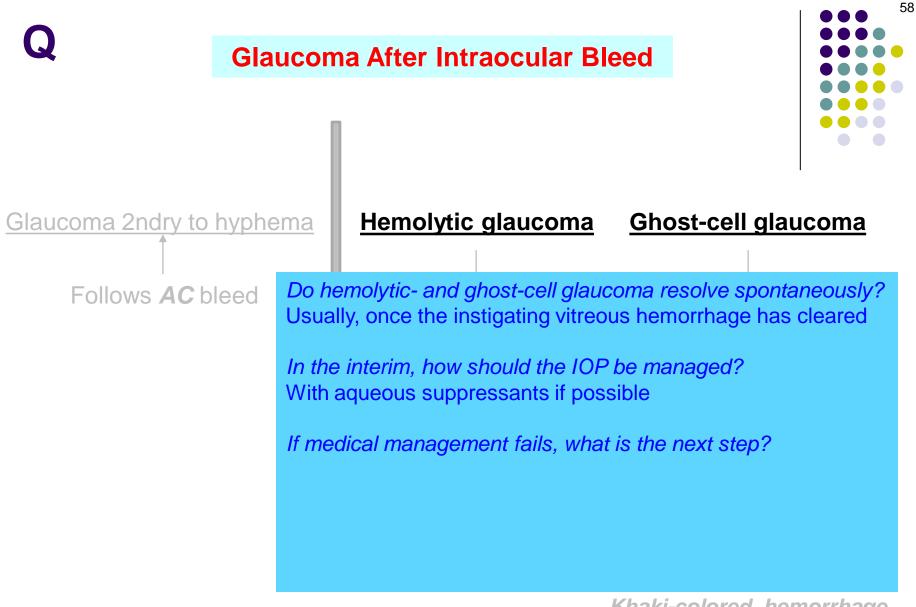
in the vitreous



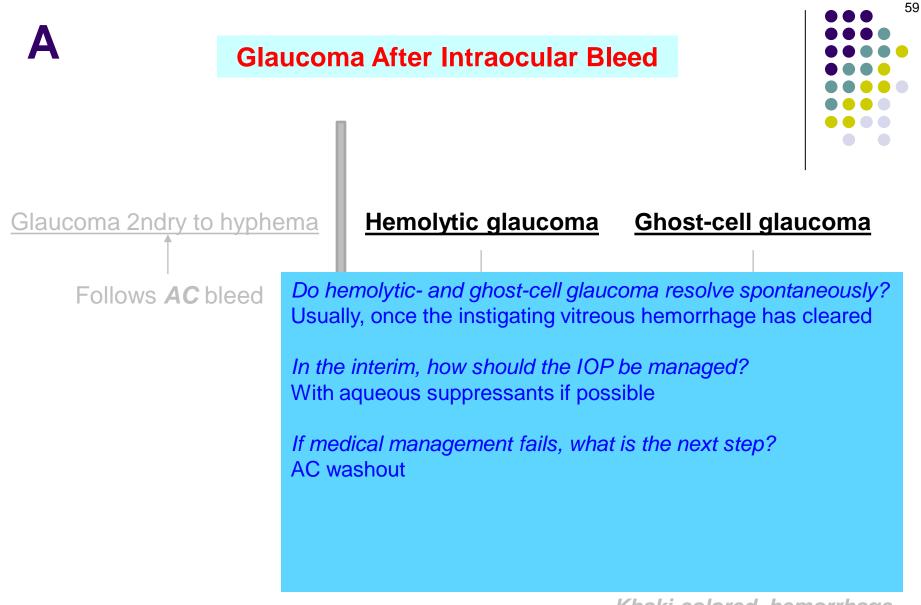
Khaki-colored hemorrhage in the vitreous



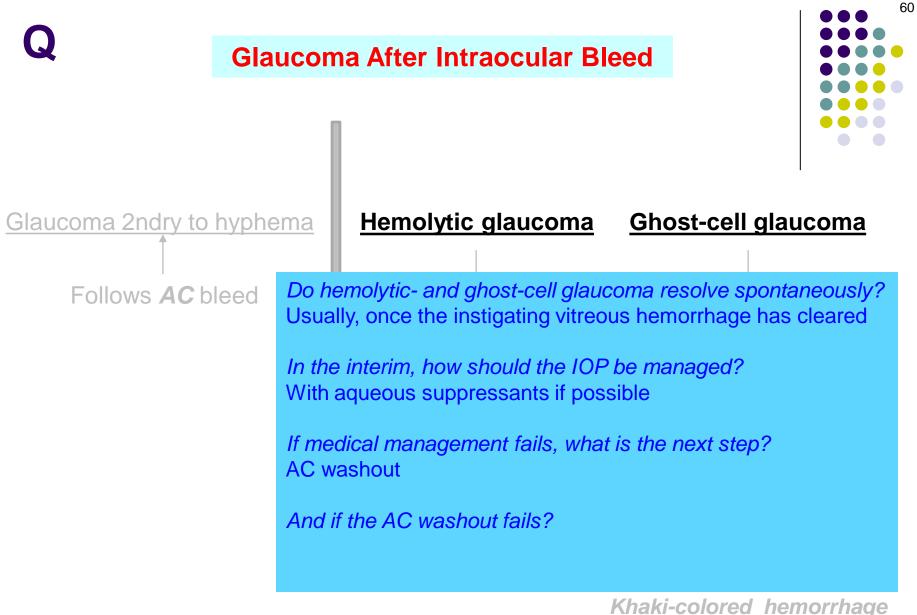
haki-colored hemorrhage in the vitreous



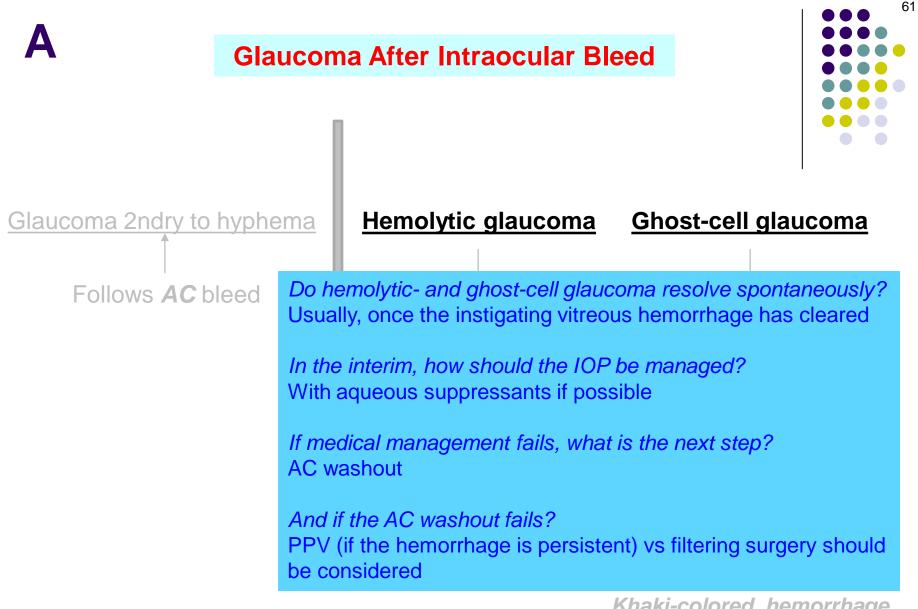
Khaki-colored hemorrhage in the vitreous



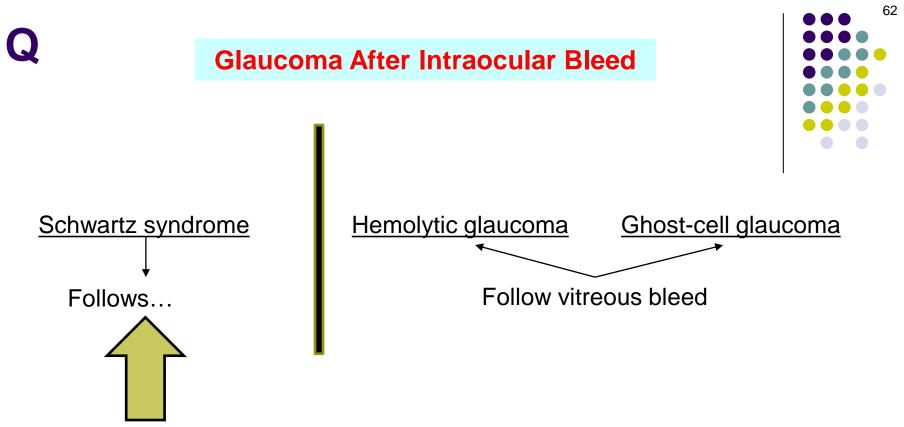
Khaki-colored hemorrhage in the vitreous

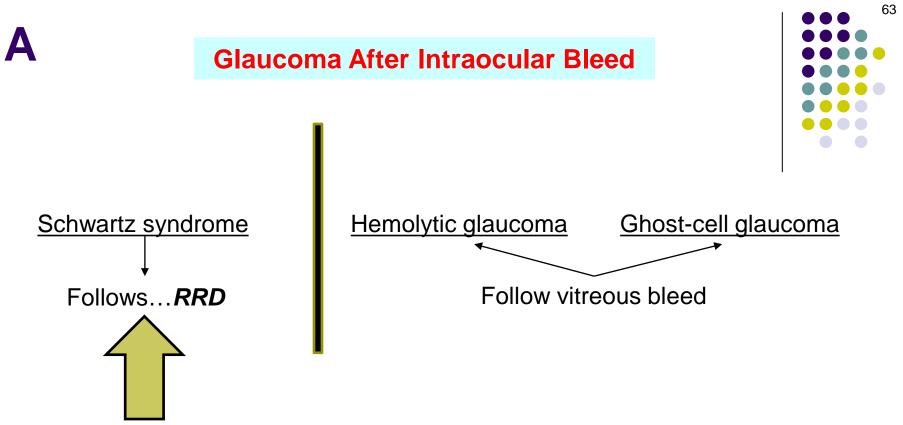


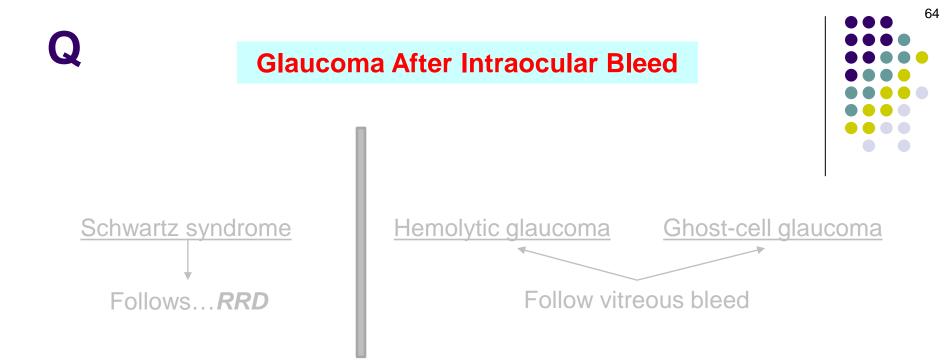
in the vitreous



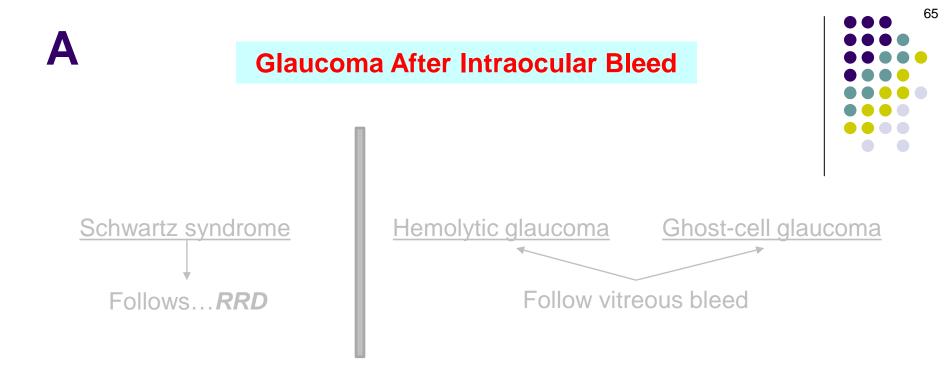
Khaki-colored hemorrhage in the vitreous



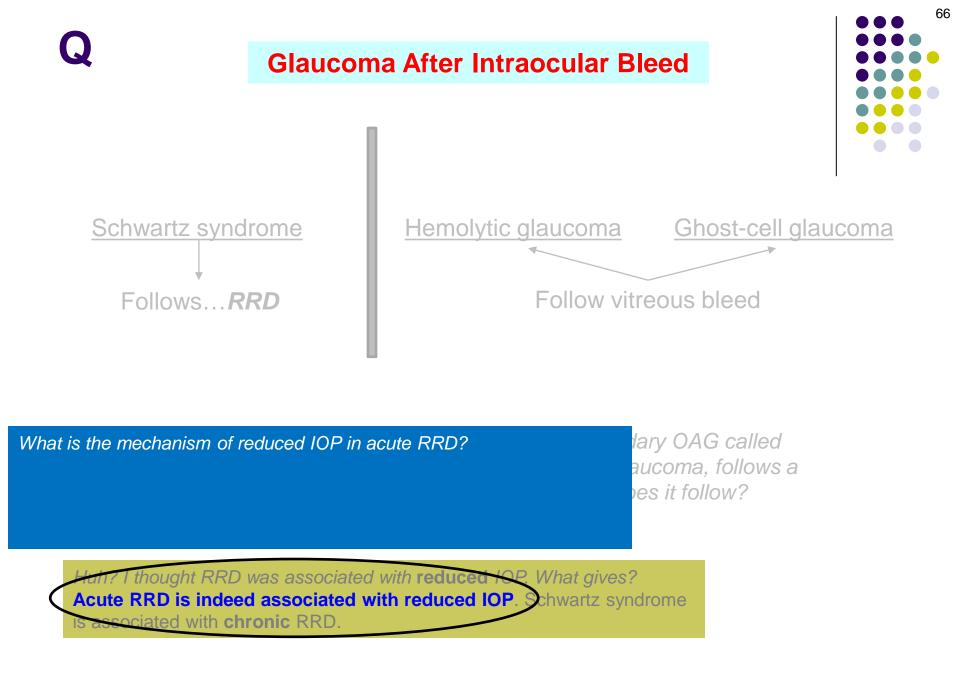


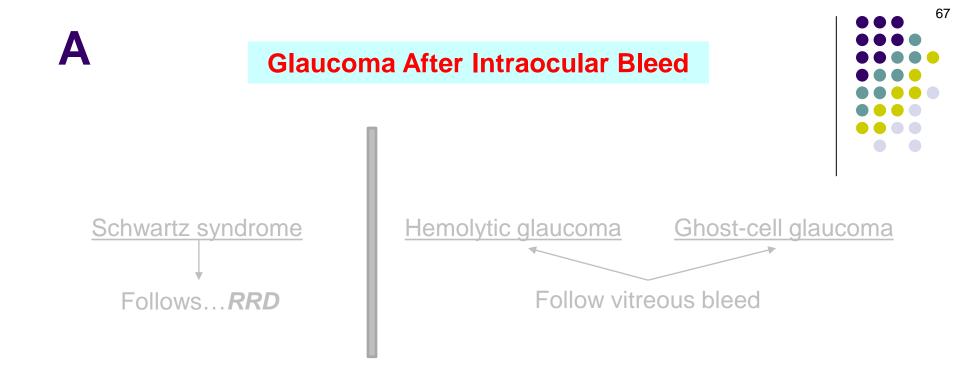


Huh? I thought RRD was associated with reduced IOP. What gives?



Huh? I thought RRD was associated with **reduced** *IOP. What gives?* **Acute** RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with **chronic** RRD.

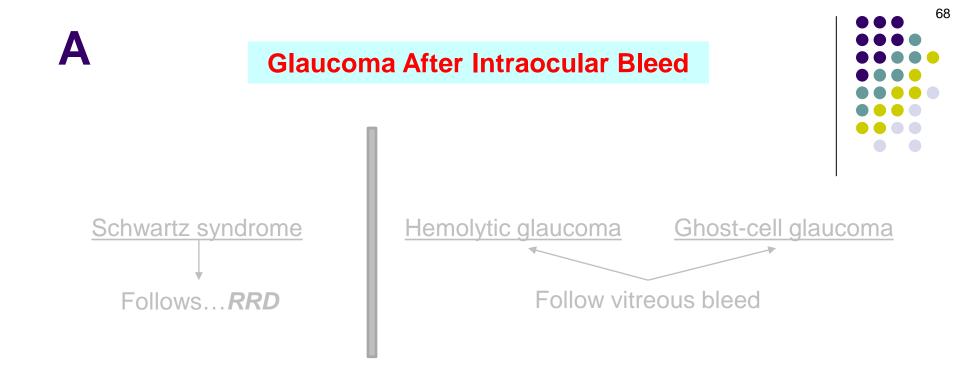




What is the mechanism of reduced IOP in acute RRD? Recall that one function of the RPE is to deturgesce the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it.

lary OAG called aucoma, follows a pes it follow?

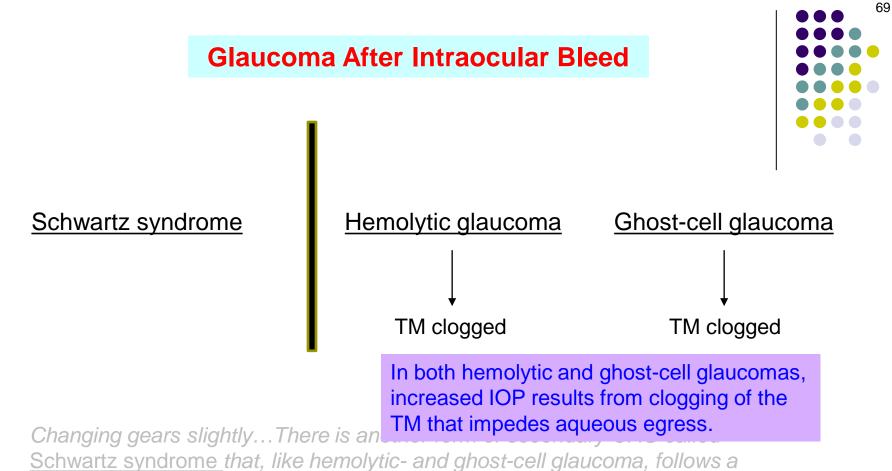
Acute RRD is indeed associated with reduced HOP. What gives? Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.



What is the mechanism of reduced IOP in acute RRD? Recall that one function of the RPE is to deturgesce the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it. If a significant enough portion of this fluid is removed, IOP will go down.

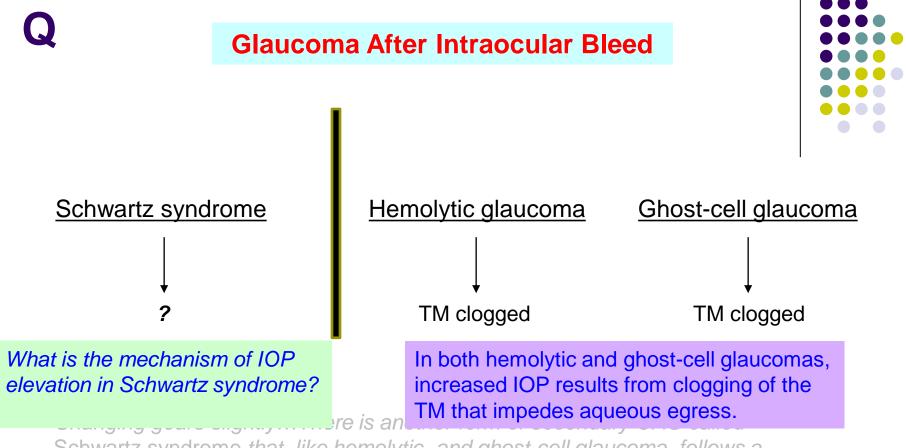
lary OAG called aucoma, follows a bes it follow?

Han? I thought RRD was associated with reduced HOP. What gives? Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.



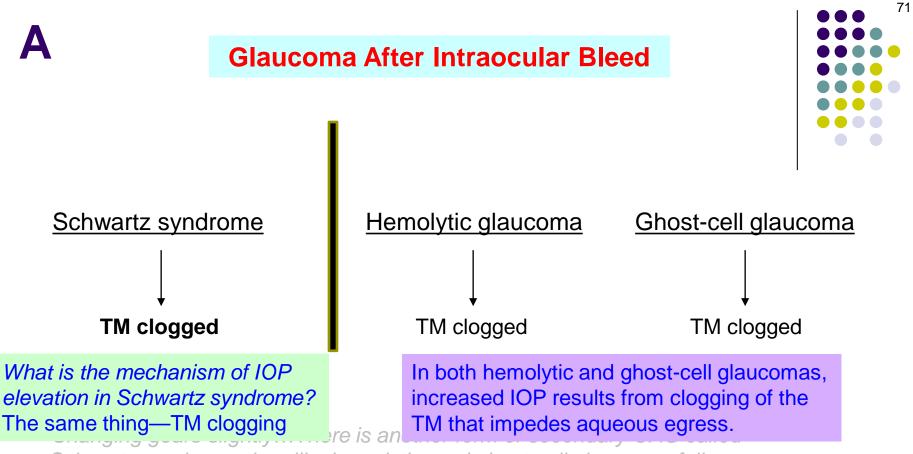
posterior-segment event—but **not** a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

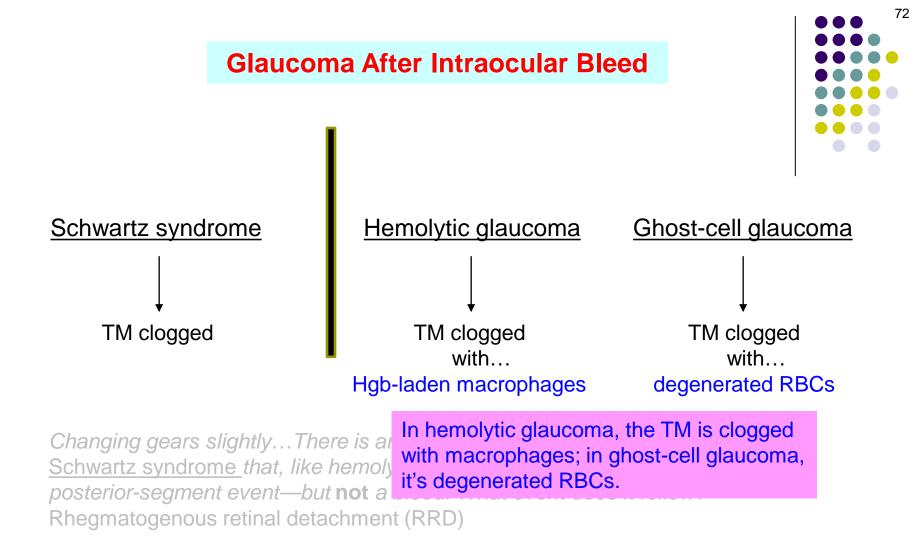


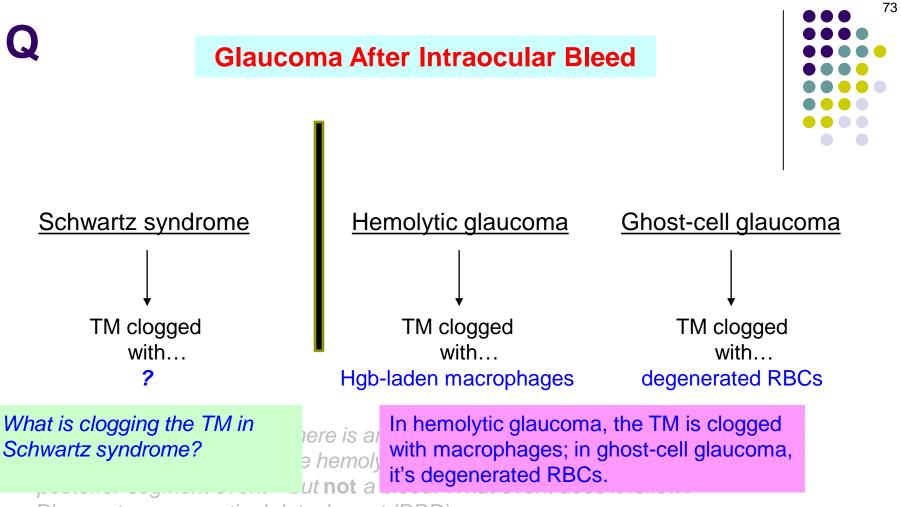
70

<u>Schwartz syndrome</u> that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but **not** a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)

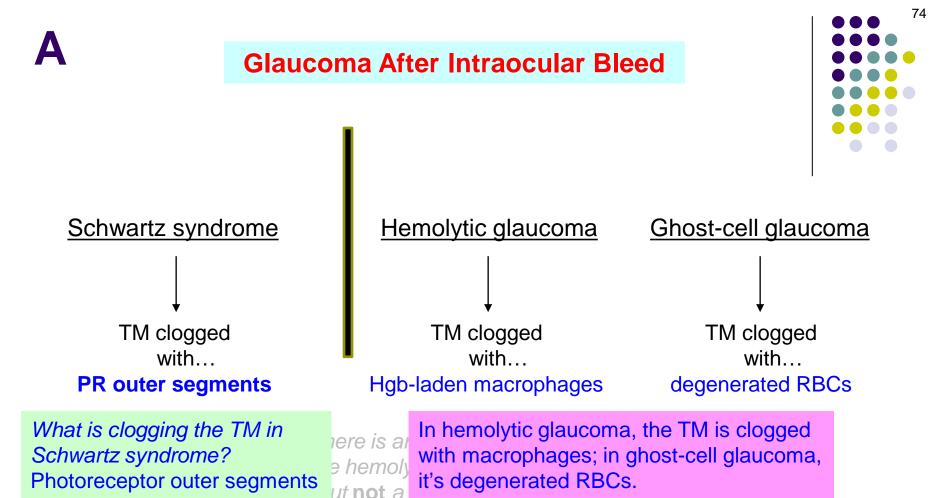


<u>Schwartz syndrome</u> that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but **not** a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)

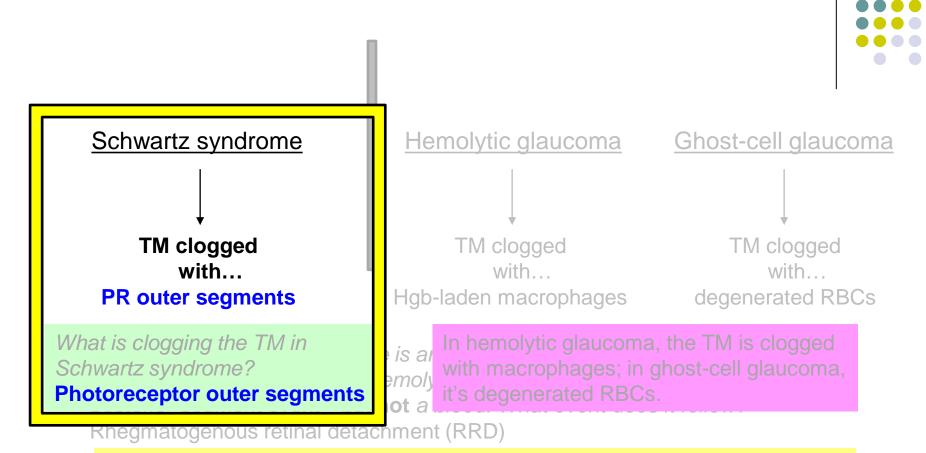




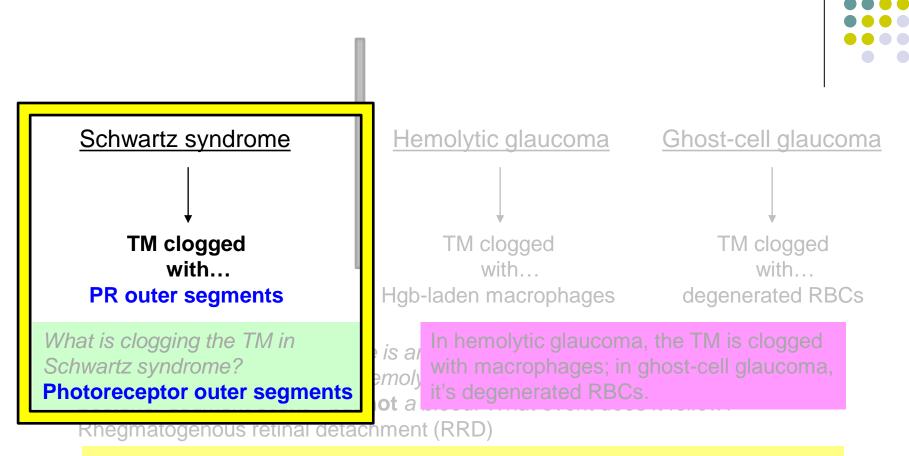
Rhegmatogenous retinal detachment (RRD)



Rhegmatogenous retinal detachment (RRD)



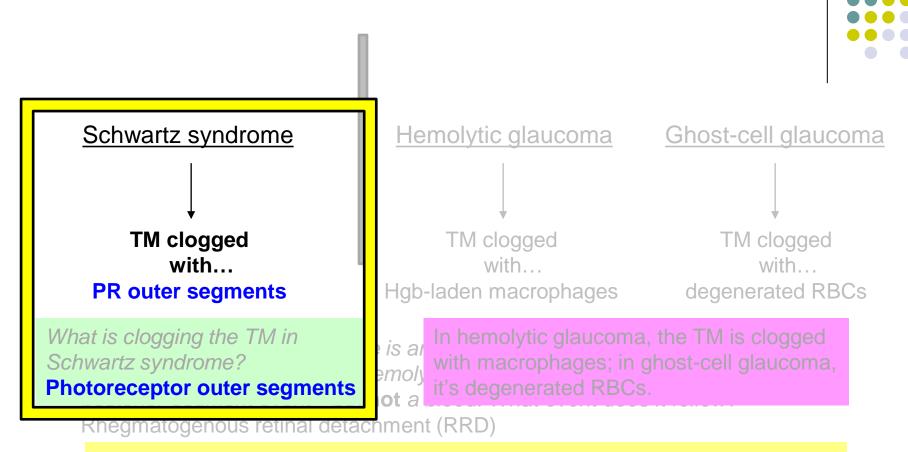
To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated PR outer segs to migrate into the AC, where their accumulation in the angle ends up clogging the TM and elevating IOP.



To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated **PR outer segs to migrate into the AC**, where their accumulation in the angle ends up clogging the TM and elevating IOP.

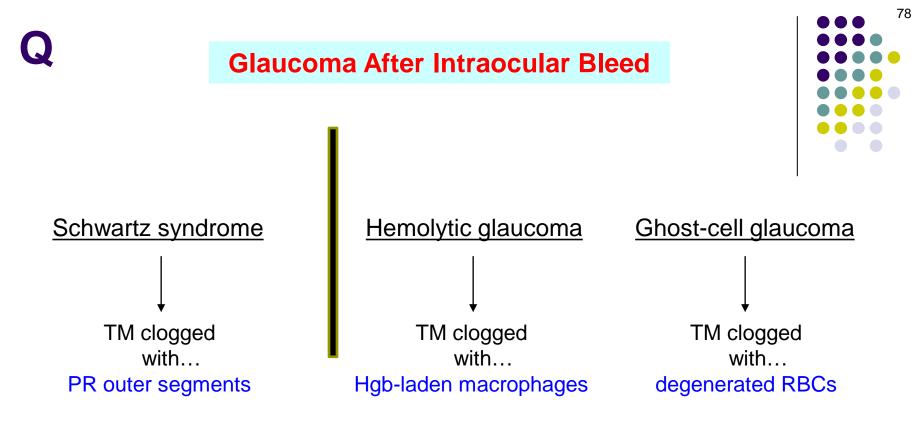
All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells?



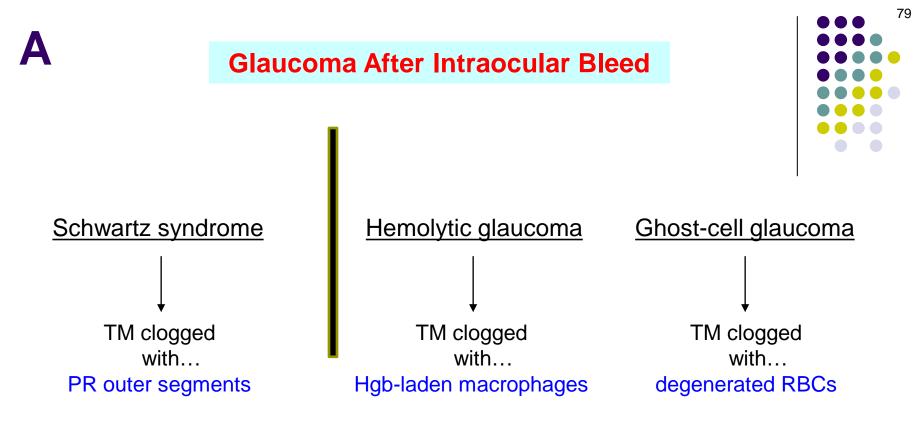


To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated **PR outer segs to migrate into the AC**, where their accumulation in the angle ends up clogging the TM and elevating IOP.

> All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells? Yes, uveitic glaucoma is a common misdiagnosis in Schwartz syndrome



What's the best way to manage Schwartz syndrome?



What's the best way to manage Schwartz syndrome? Repair the RRD