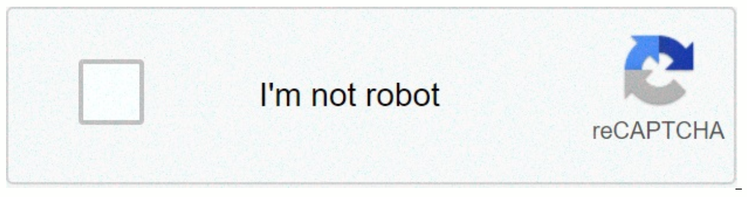

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Pathophysiology of hypertensive retinopathy pdf

Effects of high blood pressure, diabetes, and smoking on prediction of age and sex from sheeh-fund images. Kim YD, Noh KJ, Byun SJ, Lee S, Kim T, Sunwoo L, Lee KJ, Kang SH, Park KH, Park SJ. Kim YD, et al. Sci Rep. 2020 Mar 12;10(1):4623. doi: 10.1038/s41598-020-61519-9. Sci Rep. 2020. PMID: 32165702 Free PMC Article. Acute and chronic changes in blood pressure may manifest systemically in the eyes, respectively, from acute changes from malignant blood pressure and chronic changes from long-term, systemic blood pressure. Ocular involvement in the regulation of malignant blood pressure was first described by Librich, in 1859. Heer, during the 1970s and 1980s, clarified clear pathophysiological mechanisms for eye involvement, describing clinical findings through direct observations of patient management and animal models. Essentially, the ocular effects of high blood pressure are due to the effect of high blood pressure on the ocular vascular. Eye changes can be the primary finding in a patient without purpose with high blood pressure that requires primary care referral. In other cases, a symptomatic patient may be referred to an ophthalmologist for vision problems caused by changes in high blood pressure. Rapid and accurate diagnosis of high blood pressure retinopathy, especially when associated with malignant blood pressure, is essential to prevent vision and systemic disease. In addition, proper patient education about proper diet, exercise, and medication compliance is very important. For higher sources of patient education, visit the Ophthalmology Health and Diabetes Center. Also look at eMedicineHealth patient education articles, high blood pressure, and diabetic eye disease. Histolic arteries of artriol chronicles are continuous with 100 µm calibers without internal elastic lamina or muscular coats. Changes in the luminous diameter of arteries are the most important component in regulating systemic arterial blood pressure. Flow resistance is equivalent to qatar's fourth power. So lumen's 50 percent reduction leads to a 16-fold increase in pressure. Network arteries and morgues in anatomy are similar to brain vessels where they show automatic regulation mechanisms and narrow intersections to maintain the ocular barrier of blood. The croidial arteries and capillations have phenstratition (for example, without a blood ocular barrier) and do not show automatic regulation. The optical nerve vessels of the head show intermediate characteristics with automatic regulation but an inadequacy barrier of ocular blood as a result of choroidal peri papillar veins. Due to vascular differences between the retina, cheoroid, and optical nerve, each of these anatomical regions responds differently to high blood pressure. But together the clinical image shows an eye response to systemic blood pressure. Look at high blood pressure for more information. Artyosclerotic changes are chronic changes caused by systemic hypertension. in the retina, and arteriolosclerosis predominate. According to Spencer, the vaginal vascular natural light reflex is composed of reflections from the interface between the blood column and the vessel wall. [1] At first, increasing the thickness of the vessel wall makes the reflex more diffring and less clear. The progression of sclerosis and hyalinization causes the reflux to become more diffring and the arteries become red-brown chronicles. This is known as copper wiring. Advanced sclerosis of tyne vessels leads to increased optical density of the walls of retin blood vessels; It is visible in the ophthalmologist as a phenomenon known as sheathing of vessels. When the anterior surface is involved, the entire vessel appears opaque (sheathing the stem tube). The patience of such vessels has been shown by fluorescent angiography. When sheathing surrounds the wall, it produces a silver wire vessel. The general reduction of artriols from dediculous vasospasm concludes that it occurs when a significant height of blood pressure has continued for an appreciative period. The relationship between narrowing of artery caliber and diastatic pressure height has been considered. Increased intra-aluminum pressure, whether in the retinal arteries or in the central artery of the retina, narrows the arteries. Wang and his colleagues investigated the incidence of microvascular changes associated with systemic arterial hypertension in 2058. They identified that focal artery narrowing was closely related to controlling high blood pressure. They stated that focal artery narrowing is a foremic background for better known microvascular abnormalities associated with hypertension. [2] Focal narrowing of spasms occurs in local areas of the vascular muscle. Spencer guessed that either plasma in the vein and surrounding wall or vascular spasms would lead to focal narrowing, which can be permanently fibrosis. In arterial venous nicking (symptom), the circulatory barrier leads to a loose or swollen swelling to the crossing, causing hourglass stenosis on either side of the crossing and aneurysmal swelling. Eiqui noted that arterial and venous basement membranes adhere to common collagen fibers at passing points. Thickening of basement membranes and arterial media in high blood pressure prevents zinc and causes passing phenomenon. [1] Mimatsu stated that the passing changes caused by the sclerotic thickening of the wall of the venule and not by compression by the artery, while Seitz attributed the phenomenon of passage to vascular sclerosis and proliferation of perivascular glial cells and not to intravenous compression. [1] Sclerosis may elongate the short retina artery, with branches coming off at right angles. This length change distracts the verins in the common sheath and redirects the Verin (Salus mark). According to Albert et al., Main The angle, degree of thickening of the vessels, and the pressure of the di die-dial affect this phenomenon. [3] Changes in circulation in the acute stage of high blood pressure primarily involve the terminal artery rather than the main retin arteries. The main arterial changes of retin are seen and recognized as a response to chronic systemic hypertension. First described by Heir, focal periartile transodes (FIPTs) are observed in malignant arterial blood pressure. They are composed of small, white, focal, ellipse deep in the retina, and are associated with major arterial vessels and are among the first retinal injuries caused by malignant blood pressure. FIPTs may be related to the dice of terminal arteries and the breakdown of automated regulation mechanisms due to acute and malignant increases in blood pressure. This leads to the breakdown of the blood-retion barrier, which allows the transmission and accumulation of macromolecules. FIPTs are not associated with capsule fading and are not cotton-wool spots. They are hyperfluorescent and leak on fluorescent angiography. Fluffy, a whiteness found on the surface of the nerve fiber layer, ischemic spots of the inner retina, also called cotton-wool spots, are mostly located in the posterior pole and are related to the distribution of radial peri papillary capilles. These woolly cotton spots last approximately 3 to 6 weeks before fading. The angiography appearance of fluorescence is due to lack of hyperactivity and capillary cracks in the hypofluorescent. Fading collateral leads to the development of microorganisms, shuning vessels, and collaterals. Hear noted that the development of retinblate hemorrhages is neither an early finding nor an apparent finding associated with malignant blood pressure. The effects of high blood pressure on the croid are related to anatomical and functional differences found in the cheoroidal vessels, compared to the retinal vascular. Sympathetic internalization makes terminal arteries more susceptible to vascularity. Phenestratations in capilles and consequently the absence of ocular blood barrier allow the free passage of macromolecules. No automatic adjustment increases sensitivity to high perfusion pressures. Acute ischemic changes in choriocapillaris and over-retin pigment epithelium lead to acute, retine epithelium canonical pigments. This focus is white spots on the epithelium surface of the aetic pigment similar to FIPTs. Cerus Retini detachments, which preferably affect the macular region, cause neural retin (NSRD) and cystoid macular edema. Ischemic damage to retinoal pigment epithelial leads to the failure of the blood-retinal barrier. Heer observed that the existence of NSRD's was correlited with the degree of coroidal circulation disorder. Optical disc edema is a primary manifestation of high blood pressure optical neuropathy. Blood supply reaches the optical nerve through posterior silydrial arteries Choroidal peri papiller vessels. Vasconestrication and ischemia of spherical in regulating malignant blood pressure lead to optical disc edema and axoblastemia flow staz. [4] Chronic changes in blood pressure to the retina include the following (see vascular changes in high blood pressure): Arteriolosclerosis - local or general narrowing of copper wiring vessels and silver wiring of arteries resulting in artrioloclerosis (p See the evaluation.) Arteriovenous Nicking (AV) as a result of arthrolosclerosis changes increase vascular toretocyte regeneration changes due to lack of capillary injections, such as shuning vessels and microaneurysms, changes in epithelium pigment retin including seed development Pigmentation is published and the appearance of the butterfly is eaten. Areas of pigment epithelium clough and atrophy (Elschnig spots), formed from the concentration of acute focal white pigment epithelium. Triangular fragments of atrophy caused by the obstruction of a larger caliber corral vessel of optical disc paller develop in chronic blood pressure. Hypertension.