ELSEVIER

www.elsevier.com/locate/ophopt

Ophthal. Physiol. Opt. Vol. 21, No. 6, pp. 430–440, 2001

0275-5408/01/$20.00

PII: S0275-5408(01)00022-9

Ocular signs of systemic hypertension: A review

Peter G. Hurcomb[1], James S. Wolffsohn[1] and Genevieve A. Napper[2]

[1]Neurosciences Research Institute, School of Life and Health Sciences, Aston University, Aston Triangle, Birmingham B4 7ET, UK and [2]Victorian College of Optometry, Melbourne, Australia

Summary

Cardiovascular disease and stroke continue to be the chief causes of death in developed countries and one of the leading causes of visual impairment. The individual with systemic hypertension may remain asymptomatic for many years. Systemic mortality and morbidity are markedly higher for hypertensives than normotensives, but can be significantly reduced by early diagnosis and then efficient management. However, the ability of Optometrists to detect and appropriately refer systemic hypertensives remains generally poor. This review examines the disease, its effects and detection by observation of the retinal signs, particularly those considered to be pre-malignant. Previous methods of classifying retinal hypertensive signs are discussed along with more recent image analysis techniques. The role of the optometrist in detecting, monitoring and appropriate referral of systemic hypertensives is discussed in relation to current research.

Systemic hypertension is the commonest cause of mortality in the developed world and often has very few warning signs. The diagnosis and treatment is primarily based on blood pressure (BP), measured by a sphygmomanometer, but this is influenced by factors such as time of day, stress, exercise, diet, age, and gender (Pickering, 1974) and may not indicate the degree of arterial wall degeneration and end organ damage (Black *et al*., 1999). Hypertension is defined as the persistent elevation of the arterial BP against the wall of the blood vessel (*Oxford Concise Medical Dictionary*, Martin, 1994). This rise in pressure is in response to increases in cardiac output and/or peripheral vascular resistance (Terry, 1976b). Systemic hypertension can be classified as either primary (90–95%), which has no known cause, or secondary (5–10%), where the causative factor could be renal disease, endocrine or coarctation of the aorta (North, 1999). Arterial BP measurement is traditionally recorded as the systolic and diastolic pressure:

Systolic pressure: The pressure in the arteries when the contraction of the heart forces blood into them (at the height of pulsation). In a young adult it is approximately 120 mm Hg.

Diastolic pressure: The pressure in the arteries, maintained by the elastic recoil of the large arteries during cardiac relaxation. In a young adult it is approximately 80 mm Hg.

BP has been classified, according to measures of systolic and diastolic BP, into groups by the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure to aid management (*Table 1*; JNC, 1997). Systolic BP is thought to be a better diagnostic indicator of coronary heart disease, cardiovascular disease, heart failure, stroke, and renal disease than is diastolic BP (Marshall and Malinovsky, 1998; Black *et al*., 1999).

The individual with systemic hypertension may remain asymptomatic for many years, and if diagnosed may lapse on their medication or, if undiagnosed, remain undetected (Good and Augsburger, 1989). Systemic mortality and morbidity are markedly higher for hypertensives than normotensives (Hayreh, 1996). These can be significantly reduced by early diagnosis and then efficient management of the individual for the remainder of their life (Harris *et al*., 1994).

Received: 8 January 2001
Revised form: 27 June 2001
Accepted: 4 July 2001

Correspondence and reprint requests to: Dr. J.S.W. Wolffsohn.

Table 1. Classification of blood pressure and management for adults (≥18 years) as recommended by the sixth report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (1997)

Category	Blood pressure (mm Hg)			Recommended follow-up
	Systolic		Diastolic	
Optimal < 120	< 80	and		
Normal < 130	< 85	and		Recheck in 2 years
High normal	130–139	and	85–89	Recheck in 1 year/provide lifestyle advice
Hypertension				
Stage 1	140–159	or	90–99	Confirm within 2 months/lifestyle advice
Stage 2	160–179	or	100–109	Evaluate/refer to care within 1 month
Stage 3	≧ 180	or	≧ 110	Evaluate/refer depending on clinical situation

Related ocular anatomy

Both the retinal and optic nerve head circulation possess autoregulation (Hayreh, 1996). The terminal arterioles can constrict or dilate in response to a change in BP and perfusion pressure, which alters their vascular resistance and maintains a constant blood flow (Hayreh, 1996). Autoregulation can become ineffective and breakdown following: a change in BP outside the critical range over which it operates (Tso and Jampol, 1982); variations in the lumen size of the pre-capillary arterioles (e.g. vasospasm and arteriolosclerosis); and variations in the vascular endothelial basal cell function (altering production of nitric oxide, a dilator) (Kishi *et al*., 1985; Hayreh *et al*., 1986b). The blood–retinal barrier blocks macromolecules leaking from the retinal vessels and the leakage of fluid from the choroid into the retina, but can be disrupted by malignant arterial hypertension. There is no blood–ocular barrier between the choroid and optic nerve head.

Prevalence

The prevalence of high BP over the age of 40 years (taken as >160/90 mm Hg) in the UK is 10–15% of the population, with malignant hypertension (>260/140 mm Hg) constituting 5% of all systemic hypertensive cases (Grundy, 1990). The prevalence is almost double (~21%) in Afro-Caribbeans (Sharp *et al*., 1995). The World Health Organization proposed that there are presently 600 million hypertensives in the world, of which 3 million die annually as a direct result of hypertension (Guidelines Subcommittee, 1999).

Morbidity

The patient with systemic hypertension has an increased mortality rate compared to normotensives from conditions such as stroke (×7), heart attack (×4), coronary artery disease (×3) and peripheral arterial disease (×2) (Kannel and Sorlie, 1975). Cardiovascular disease and stroke are among the chief causes of death in developed countries (Marshall and Malinovsky, 1998). The major risk factors for developing systemic hypertension are: hereditary factors, organic diseases, medicines and lifestyle (weight, diet, smoking, stress, emotional strain, alcohol consumption, and exercise). A 1 mm Hg rise in systolic BP is associated with a 1% rise in all-cause mortality (Silagy and McNeil, 1992). A slight elevation in diastolic BP is associated with a shortened life expectancy (Kannel *et al*., 1961). It is estimated that around 20% of mortality in the developed world is due to cardiovascular disease associated with diastolic BP of 90–100 mm Hg (Hickey and Graham, 1988). Additionally, it has been shown that hypotension (low BP) can be damaging to the body (e.g. increased cardiovascular complications) and may be implicated in ocular disease such as low tension glaucoma (Goldberg *et al*., 1981; Farnett *et al*., 1991).

Treatment

The treatment of systemic hypertension is a life-long co-operation between patient and doctor. Non-pharmacological management of the mild hypertensive patient may include weight reduction, reduction in alcohol consumption, regular exercise, change in diet (reduction in salt and saturated fat) and cessation of smoking (North, 1999). Pharmacological management utilises six main classes of hypertensive drugs: diuretics, beta-blockers, angiotensin converting enzyme inhibitors, calcium antagonists, alpha-adrenergic receptor blockers and angiotensin II antagonists. The prescribed drug will depend upon the co-existing medical conditions of the individual and previous toxicity to medications. A combination of two or more low dose antihypertensive medications can be more effective than monotherapy for reaching the target BP and has a lower risk of side effects (Black *et al*., 1999). More aggressive regimes are applied if target organ damage is present (Dodson and Kritzinger, 1997). Reduction in BP to <140/90 mm Hg is only achieved in 25% of hypertensive patients (JNC, 1997). Long-term treatment of patients with systemic hypertension has shown a 40% drop in strokes and 14% drop in coronary heart disease rate (Collins, 1994). The lowering of BP too drastically can be detrimental, inducing a rise in mortality

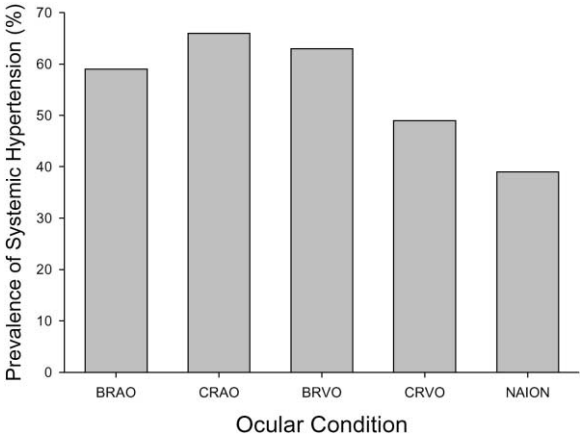

Fig. 1. Prevalence rate of systemic hypertension in patients with retinovascular disease (Dodson and Kritzinger, 1997). BRAO = Branch Retinal Artery Occlusion; CRAO = Central Retinal Artery Occlusion; BRVO = Branch Retinal Vein Occlusion; CRVO = Central Retinal Vein Occlusion; NAION = Non-Arteritic Anterior Ischaemic Optic Neuropathy.

and morbidity (Fletcher and Bulpitt, 1992). For example, a diastolic BP of 75 mm Hg, compared with 85 mm Hg, is associated with a doubling in the prevalence of cardiac events. (Farnett *et al*., 1991).

Ocular conditions associated with hypertension

The prevalence of the most common forms of retinovascular disease in the systemic hypertensive are shown in *Figure 1* (Dodson and Kritzinger, 1997). A patient who has had a single retinal vein occlusion has a 10–15% chance of it occurring in the fellow eye (Hayreh *et al*., 1994; Dodson and Kritzinger, 1987). Patients who have had a retinal artery occlusion show a significant mortality rate for myocardial infarction (Hankey *et al*., 1991). With increasingly effective antihypertensive medication there is a rise in visual disorders, such as optic nerve head ischaemia, vascular occlusive disorders and progressive field deterioration in glaucoma, in response to arterial hypotension and nocturnal hypotension (a drop in BP during sleep; Hayreh, 1996).

Ocular signs of systemic hypertension

The blood vessels are the primary tissue to respond to an acute rise in BP (Hayreh, 1989). In a similar manner to diabetes, elevated BP produces changes in the pericytes in the retinal capillary walls (Wallow *et al*., 1993). Hypertensive retinopathy is the second most common retinal vascular disease after diabetic retinopathy (Marshall and Malinovsky, 1998). Retinopathy associated with high BP has been found to occur in a 6.3% of the non-diabetic population without systemic hypertension (Klein *et al*., 1993, 1994).

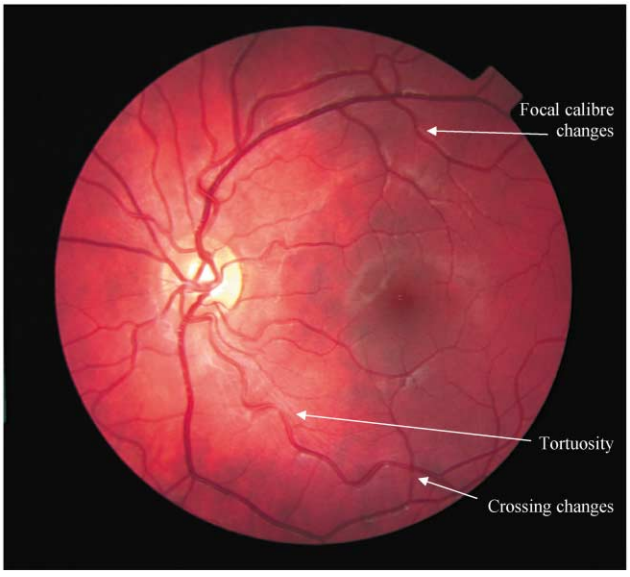

Fig. 2. Ocular fundus image demonstrating pre-malignant systemic hypertensive retinal vasculature changes.

The ocular manifestations of systemic hypertension involve the retinal, choroidal and optic nerve head vasculature (Hayreh 1996). In animal studies hypertensive retinopathy has been found to appear earlier than choroidopathy or optic neuropathy (Hayreh 1989). For the purpose of this review, ocular signs of systemic hypertension have been divided into those considered to be indicators of pre-malignant (early changes, prior to significant damage to the retinal vasculature) and malignant systemic hypertension (Dodson *et al*., 1996).

Pre-malignant retinal signs

The pre-malignant hypertensive retinal changes are pathological signs of systemic hypertension that are more subtle than malignant retinal signs. Similar observations can be physiological in origin or can occur with increasing age (Salus, 1958) (*Figure 2*). Improvements in the detection of pre-malignant retinal signs of systemic hypertension could provide better screening, monitoring and management of these patients in the future.

Arteriolar narrowing (A/V ratio changes)

The extent of arteriole narrowing is dependent upon the level of pre-existing involutional sclerosis (replacement fibrosis). In young individuals with little involutional sclerosis hypertensive narrowing will be more evident than in an elderly patient where vessels are more rigid due to involutional sclerosis (Kanski, 1995). Arteriolar narrowing is associated with prolonged raised BP, particularly diastolic (Scheie, 1953), and age (Bechgaard *et al*., 1950; Breslin *et al*., 1966; Svardsudd *et al*., 1978; Fuchs *et al*.,

1995). This arteriolar change is present in up to one third of systemic hypertensive patients and is thought to reflect systemic hypertension rather than arteriolar sclerotic changes (Bechgaard *et al*., 1950). Examination of the degree of arteriolar narrowing is best established beyond the second branch of the central retinal vessel or around the fovea (Salus, 1953, 1958). It is first observed in the nasal branches of the retinal vasculature (Cogan, 1974). The arterio-venous ratio must be assessed at a comparable branching of the arteriole and vein (Stokoe and Turner, 1966; Michaelson *et al*., 1967). However, the selection of two such vessels is difficult, tedious and time consuming by casual ophthalmoscopy. A recent large population study has found arteriolar narrowing to be inversely correlated to BP, with the A/V ratio decreasing by ~0.02 units for a 10 mm Hg increase in mean BP (Hubbard *et al*., 1999). The A/V ratio was found to be lower in males, current smokers, blacks compared to whites and older individuals.

Arteriolar reflex

The normal arteriolar light reflex originates from the surface of the erythrocytes. In the hypertensive vessel the reflex will also originate from the thickened vessel wall and the associated increase in refractive index makes the vessel wall less transparent (Salus, 1958; Marshall and Malinovsky, 1998). The increase in the arteriolar reflex of the vessel is associated with increased BP, occurring in 46% of the hypertensive population and 10–33% of a general population (Bechgaard *et al*., 1950; Aurell and Tibblin, 1965). This is considered to be the first change of arteriolosclerosis retinopathy and reflects the state of general arteriolar circulation throughout the rest of the body (Marshall and Malinovsky, 1998). The reflex intensity is significantly reduced by the moderate lowering of systolic BP and to a lesser extent diastolic BP (Brinchmann-Hansen *et al*., 1990). The arteriolar reflex is correlated to age in those under 50 years (Svardsudd *et al*., 1978). The presence of silver and copper wiring in non-malignant hypertensives is rare (Bechgaard *et al*., 1950).

Arteriolar tortuosity

Few studies have examined arteriolar tortuosity. It has a prevalence of approximately 22% in a hypertensive population (Bechgaard *et al*., 1950), but the prevalence in normotensives has not been quantified. In that study, its association with systolic and diastolic BP was less marked than other observed vasculature changes and its presence was unrelated to age (Bechgaard *et al*., 1950). Quantitative findings indicate that the tortuosity in normotensives also does not change with age (Williams, 1982). From clinical observation, authors have suggested that arteriolar tortuosity occurs initially in the nasal fundus (Cogan, 1974), congenital tortuosity occurs uniformly throughout the fundus, whereas, segmental arterial tortuosity is always abnormal (Walsh, 1982) and that arteriolar tortuosity is not diagnostic of hypertension (Salus, 1958).

Focal arteriolar calibre changes

Focal arteriolar calibre changes, which were originally thought to reflect arteriolosclerosis (Bechgaard *et al*., 1950; Salus, 1958), have been found to be highly correlated with high systolic BP (Klein *et al*., 1993, 1997; Hubbard *et al*., 1999) and are an indicator of long term hypertension (North, 1999). These changes have been shown to be an important prognostic retinal indicator for mortality, stroke and death from malignancy over a 12.5 year period (Svardsudd *et al*., 1978). Their prevalence increases with diastolic BP: 17% when $<$100 mm Hg rising to 99% when $>$140 mm Hg (Breslin *et al*., 1966). The frequency of focal arteriolar calibre changes increases with age, with a prevalence of 7–30% reported (Svardsudd *et al*., 1978; Hubbard *et al*., 1999). Hayreh (1996) stated that arteriolar narrowing viewed by ophthalmoscopy was an artefact caused by retinal oedema partially masking the arteriole from both sides.

Arteriovenous crossing changes

Arteriovenous crossing changes and subsequent arching of the vein (nipping) causes an impedance of retinal blood flow, with the vein distal to the disc darker, larger and more tortuous than the proximal segment (Walsh, 1982). Their prevalence has been found to be 37% in a hypertensive population (Bechgaard *et al*., 1950) and less than 10% in a general population (Aurell and Tibblin, 1965; Svardsudd *et al*., 1978; Hubbard *et al*., 1999). Their frequency increases with increased systolic BP, the duration of cardiovascular disease, smoking, race (higher in blacks than whites) and age (from 0.8% in the 4th decade of life to 5.0% in those over the age of 75 years; Bechgaard *et al*., 1950; Svardsudd *et al*., 1978; Klein *et al*., 1993, 1997; Fuchs *et al*., 1995; Hubbard *et al*., 1999). The crossing changes remain even after high BP is reduced. Arteriovenous crossing changes were the least important prognostic retinal indicator for mortality, stroke and death from malignancy over a 12.5 year follow up period (Aurell and Tibblin, 1965; Svardsudd *et al*., 1978).

Malignant retinal signs

These can be broken down into: retinopathy, such as haemorrhages and lipid deposits; hypertensive choroidopathy, when vasoconstrictor agents leak freely from the choriocapillaries into the choroid interstitial fluid, causing choroid vasoconstriction and ischaemia (initially acute and then chronic); and hypertensive optic neuropathy (*Table 2*). Hypertensive choroidopathy tends to occur with acute rises in BP in young patients with severe malignant hypertension (Hayreh *et al*., 1986c). The changes are often masked by

Table 2. Malignant ocular signs of systemic hypertension and their characteristics

Retinopathy

Retinal haemorrhage

- Flame shaped—situated in the nerve fibre layer (Cogan, 1974).
- Located around the disc along the temporal and nasal vessels (Marshall and Malinovsky 1998).
- Increase in frequency and number indicates a more serious hypertensive state (Becker 1981).

Lipid deposit (hard exudates)

- Yellowish white in colour with well defined sharp borders (Grundy 1990).
- Hyperlipidaemia increases their presence (Hayreh, 1996).
- In youthful vessels they appear when the diastolic BP is above 130 mm Hg and in sclerotic cases are associated with the late stage of the illness, (Leishman, 1957).
- Usually indicative of subsiding malignant hypertension (Stokoe, 1975).
- Resolution usually takes over a year (Hayreh, 1996).

Inner retinal ischaemia spots (cotton wool spots)

- Fluffy white, feathery edged lesions situated within a few disc diameters of the optic disc along the radial peripapillary retinal capillaries (Hayreh, 1996).
- Lie superficially in the retinal nerve fibre layer obscuring the blood vessels (Hayreh *et al.*,1989).
- Observed during acute phases of severe hypertension and resolve within 6–12 weeks (Leishman, 1957; Hayreh *et al.*, 1989).

Focal intraretinal periarteriole transudates (FIPTs)

- Accumulation of plasma macromolecules in the deep retina layers along the retinal arterioles (Hayreh *et al.*, 1989).
- An early sign of malignant hypertension (Hayreh *et al.*, 1989).
- Round or oval dull white lesions up to a quarter of a disc diameter in size.
- Leave no trace on resolving (Hayreh *et al.*, 1989).

Intra retinal microvascular abnormalities (IRMAs)

- Occlusion of the retinal capillaries may cause: microaneurysms, arteriovenous shunts, looped convoluted vessels and venous sand venous collaterals.

Retinal and macular oedema

- Failure of autoregulation, ischaemia or a breakdown in the blood retinal barrier in the RPE results in leakage of sub retinal fluid into the retinal tissue. (North, 1999).

Hypertensive Choroidopathy

Filling defects in the choroidal vascular bed

- A patchy filling defect of the choroid vessels, more prominent in the macular/foveal region (Hayreh *et al.*, 1986). One of the earliest signs of choroidopathy.

Retinal pigment epithelium (RPE) lesions

- Acute focal RPE lesions, pale or white round pinhead sized sub-retinal lesions, usually present in groups situated in the macular region.
- Associated with delayed and patchy choroidal vascular bed filling.
- Early RPE degenerative lesions may merge together (becoming ill defined) or maintain their fairly defined margins (Elschnig's spots).
- Late RPE degenerative lesions are scattered over the fundus, the temporal part of the periphery and fovea being more involved.
- Changes present in the macular can resemble age-related macular degeneration and widespread changes resemble 'birdshot retinopathy' (Hayreh, 1996).

Serous retinal detachment

- Ischaemia of the RPE leads to a breakdown of the blood retinal barrier, allowing fluid to diffuse into the retina causing a retinal detachment, (mainly the macular region).
- A peripheral retinal detachment may extend 360° and produce a total retinal detachment (Hayreh, 1996).
- May resolve spontaneously with time.

Hypertensive Optic Neuropathy

- Swelling of the ganglion cell axons, in response to ischaemia, causes the optic nerve head to swell, resembling raised intracranial pressure. The disc will eventually be surrounded by flame shaped haemorrhages, exudates and inner retinal ischaemic spots (Hayreh *et al.*, 1986).
- Ischaemia of the optic nerve head and the inner retinal layer causes retinal nerve fibre loss (Hayreh *et al.*, 1986).

overlying retinal changes, but are of significant prognostic value when found.

Prevalence of hypertensive retinopathy

The prevalence of systemic hypertension (classified as systolic BP >160 mm Hg and/or diastolic BP >95 mm Hg) in the Beaver Dam Eye Study was 30.9% ($n = 4541$; Klein, 1992). Retinopathy was present in 7.8% of the non-diabetic population and 33.7% in the diabetic population (Klein, 1992). The prevalence of retinopathy in the general non-diabetic population, over the age of 40 years, was 10.7% in those with systemic hypertension and

Table 3. Summary of the Keith *et al.* (1939) grading scale

	Retinal arterioles					
	A/V ratio	General narrowing	Focal narrowing	Haemorrhage	Exudates	Papilloedema
Grade I	1/2	Mild	Mild	Absent	Absent	Absent
Grade II	1/3	Moderate–marked	Moderate–marked	Possible	Absent	Absent
Grade III	1/4	Marked	Marked	Possible	Possible	Absent
Grade IV	$< 1/5$	Marked	Marked	Possible	Possible	Present

6.3% in normotensives. Arteriolar narrowing occurred in 19 and 11% respectively. The prevalence of systemic hypertension increased with age from 21.0% in the 4th decade of life to 50.7% in those over the age of 75 years (Klein *et al*., 1993, 1997). The prevalence of hypertensive retinopathy also increased with age, from 5.5% in the 4th decade of life to 10.0% in those over the age of 75 years. The prevalence of retinal lesions increased with increased systolic BP, but not so reliably with diastolic BP (Klein *et al*., 1993).

In a 5-year follow up, 6.0% of the population developed retinopathy, 9.9% arterial narrowing and 6.5% arterial nipping (Klein *et al*., 1997). These findings are comparable with the Blue Mountains Eye Study in Australia, which found retinopathy to be present in 9.8% of a slightly older population (Yu *et al*., 1998). Both these studies found a considerably higher prevalence of retinopathy in the population than has been reported in other studies, such as 4.0% in the Atherosclerosis Risk In Communities Study (Hubbard *et al*., 1999), 0.8% in the Framingham Eye Study (Leibowitz *et al*., 1980) and 0.1% in a population of middle-aged Swedish men (Aurell and Tibblin, 1965; Svardsudd *et al*., 1978), possibly due to the more extensive retinal area examined.

Assessment of the retinal signs of systemic hypertension

Subjective grading (grading scales)

The retinal circulation has two main characteristics that make it an important prognostic indicator for assessing the vascular state of the whole body. Firstly it is an end arteriolar system and as such is particularly vulnerable to the effects of high arterial BP and secondly, it is easily observable with simple non-invasive techniques, such as ophthalmoscopy. Grading scales for examining the retinal vasculature have been in use since the late 1930s and assist in predicting the prognosis for survival within groups (Walsh, 1982).

The most commonly used grading scale of the retinal vasculature was devised by Keith *et al*. (1939) and grades arterial characteristics and retinopathy into four groups (*Table 3*). The non-malignant signs (arteriovenous ratio, general vessel narrowing, and focal vessel narrowing) are graded compared to what the examiner considers to be normal. Additional malignant signs of haemorrhages, exudates and papilloedema are graded as to their presence or absence. As the group number increases the prognosis for the individual worsens (*Table 3*). Several fundus examinations may be required before assigning a patient to a group to take account of the fact that some lesions (such as exudates) are only visible temporarily.

Despite the longevity of utilisation of this scale, it suffers from a number of major problems with respect to early detection and monitoring of systemic hypertension: The scale was based mainly on malignant hypertension, and has little prognostic value in those with less severe hypertension (Bechgaard *et al*., 1950; Aurell and Tibblin, 1965; Fuchs *et al*., 1995). Clinically the allocation of a patient between groups 1 and 2 is very subjective (Stokoe and Turner 1966; Hayreh 1989). Group 3 and 4 only differ on the presence of papilloedema, the presence of which does not influence the prognosis of a treated patient who already has bilateral retinal haemorrhages and exudates (McGregor *et al*., 1986). Vascular changes, which are due to arteriolar sclerosis (due to ageing irrespective of BP), are not distinguished and are recorded as hypertensive in origin.

Other attempts at grading the state of the retinal vasulature have been made, many based on the scale by Keith and colleagues (Scheie, 1953 (*Table 4*); Evelyn *et al*., 1958), but they suffer from: being highly complex and time consuming to perform, limiting widespread clinical use (Wagener *et al*., 1947; Leishman, 1957; Evelyn *et al*., 1958; *Figure 3*); presumptions such as vasulature changes occur just with systemic hypertension, not age (Scheie, 1953; Cogan, 1974); or that arteriolar constriction is the only important sign of hypertensive retinopathy (Cogan, 1974; Tso and Jampol, 1982). Further details are given in reviews by Hayreh (1989); Dodson *et al*. (1996).

More recently, Dodson *et al*. (1996) proposed a simplified grading scale, categorising systemic hypertensive retinal fundus changes into two categories: non-malignant and malignant hypertension (*Table 5*). All abnormalities, along with the grade, should be recorded by the examiner to avoid any loss of information (Dodson *et al*., 1996).

Subjective grading scales of systemic hypertensive retinal changes, aim to group patients in terms of their prognosis if left untreated. They do not aid practitioners in their ability to quickly and comprehensively describe the state of the retinal vasculature to assist in the monitoring and early detection of systemic hypertension. If referral is made on

Table 4. Summary of the Scheie (1953) grading scale

	Hypertension				Arteriolosclerosis	
	A/V ratio	Haemorrhage	Exudates	Papilledema	Light reflex	Vessel crossings
Normal	3/4–3/5	Absent	Absent	Absent	Fine line	None
Grade I	1/2	Absent	Absent	Absent	Increased	Mild compression
Grade II	1/3	Absent	Absent	Absent	More marked	Greater compression
Grade III	1/4	Present	Present	Absent	Copper Wire	Angular deviation
Grade IV	$< 1/5$	Present	Macula Star	Present	Silver Wire	Distal congestion

the presence of malignant hypertensive retinopathy, irreparable damage to the vasculature system of the body as a whole may have occurred and treatment may be less beneficial than if started earlier (Terry and Schoessler, 1976; Augsburger and Good, 1986). It has been suggested that classification groups for hypertensive retinopathy are inadequate and result in the loss of clinical information (Svardsudd *et al*., 1978; Hayreh, 1989). A more detailed description of fundus features is more informative for assessing the current vascular status and for follow-up of any changes.

Objective grading (image analysis)

Subjective grading of the fundus vasculature has been found to have poor inter- and intra-observer repeatability (Kagan *et al*., 1966; Michaelson *et al*., 1967; Dimmitt *et al*., 1989). Fundus images coupled with computer image processing allows a more objective grading of the retinal circulation to be achieved, overcomes the loss of information which can occur in a hand written record of a patient's fundus and allows an improved accuracy in the monitoring of the condition over time (Gilchrist, 1987; Eaton and Hatchell, 1988; Newsom *et al*., 1992).

Early attempts to quantitatively measure the width of blood vessels involved the projection of a fundus image onto a screen for manual measurement with a gauge (Hodge *et al*., 1969; Parr and Spears, 1974). Red-free filter photography has been utilised as monochromatic illumination can enhance the visibility of various ocular features accentuating the difference in reflectance between the structure and the background, e.g. vascular walls have their optimum visibility between 445 and 510 nm and vessels, haemorrhages, and microaneurysms between 530 and 590 nm (Duerey *et al*., 1979; Weinberger *et al*., 1999). Over the last decade, improvements in technology have allowed objective and higher resolution measures using edge detection (Eaton and Hatchell, 1988; Rassam *et al*., 1993, 1994; Kergoat and Lovaski, 1995; Wu *et al*., 1995; Sugiyama *et al*., 2000).

The precision of image analysis has shown that the diameter of arterioles decrease after exercise (Kergoat and Lovaski, 1995), vary with the cardiac cycle (Chen *et al*., 1994), are larger in hypertensives (Houben *et al*., 1995) and can be increased by a carbonic anhydrase inhibitor (Rassam *et al*., 1993). There is debate over whether the change in blood vessel width is correlated with ocular blood flow (Wolf *et al*., 1998; Sugiyama *et al*., 2000), but it is correlated with BP (Houben *et al*., 1995; Hubbard *et al*., 1999).

Other than blood vessel width, the only feature of the

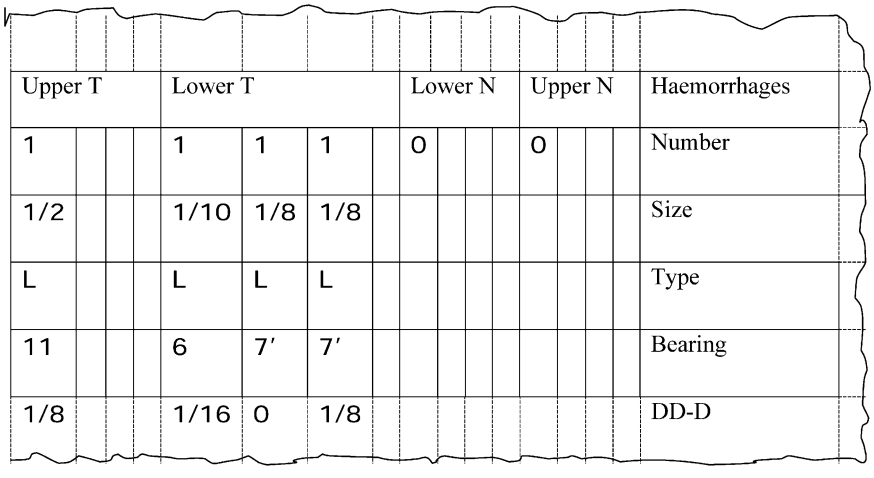

Fig. 3. A sample record sheet for the recording of haemorrhages in the right eye of a patient using the hypertensive grading scale suggested by Evelyn *et al.* (1958). Key: Size = Maximum dimension expressed as a fraction of a disc diameter. Type = L = linear/F = flame shaped/R = round. Bearing = Clock face: 7 = 7 o'clock, 7' = 7.30. DD-D = Distance in disc diameters measured from disc margin

Table 5. Summary of Dodson *et al.*, 1996 hypertensive retinopathy grading scale

Grade	Non-malignant	Malignant
Retinal changes	General arteriolar narrowing Focal constriction (not AV nicking)	Haemorrhages Hard exudates CWSs ± optic disc swelling
Hypertensive category	Established hypertension	Malignant hypertension Retinovascular damage
Prognosis	Depends on BP, age and other concomitant cardio-vascular factors	Untreated—survival < 2years Treated—survival ≈ 12 years

retinal vasculature that has been quantitated is tortuosity, by manually marking 20 points along the path of the central arteriole in 290 normotensive subjects (Williams, 1982). The path length along these points was then computed and compared to the distance between the first and last point, giving an index of tortuosity. Improved methods for quantifying tortuosity of retinal blood vessels were made by Hart *et al*. (1999), who found their measures agreed well with ophthalmologist's perception of tortuosity.

The apparent width of the retinal vessel when observed with white light is the width of the streaming column of erythrocytes, as the surrounding plasma zone and vessel wall are transparent (Brinchmann-Hansen and Heier, 1986). The measurement of the complete blood vessel width (erythrocyte column and plasma zone) is possible with fundus fluorescein angiography, as the fluorescein mixes with the plasma. The contribution of the plasma zone to the vessel diameter has been recorded between 3% (Deupree *et al*., 1985) and 11 and 22% (Hodge *et al*., 1969). Brinchmann-Hansen and Heier's (1986) theoretical modelling showed that the thickness of the vessel wall, its refractive index and the width of the plasma zone has only a negligible influence on the apparent width of the blood column. The complete blood vessel width can be measured from fundus images without the use of fluorescein angiography if the threshold is appropriately set (Rassam *et al*., 1994).

Optometrists role

Many patients with high BP are asymptomatic, unaware of their condition and may not be attending their GP regularly. These people may attend an optometrist every 1–2 years and therefore optometrists can play an important public health role in screening for systemic hypertension, performing ocular fundus examination, measuring BP, emphasizing the importance of regular GP assessment and referring suspect patients for appropriate management (Terry, 1976a; Klein *et al*., 1993). Daubs (1974) estimated that an optometrist performing sphygmomanometry could detect one undiagnosed hypertensive patient for every 14 adults tested. Barnard *et al*. (1991) found that referrals of patients for systemic hypertension on fundus lesions present and other information obtained from the eye examination without the use of BP monitoring equipment would produce a 77.8% false positive referral. BP measurement on patients with normal fundi resulted in 9.1% of them being referred for systemic hypertension. Referrals of patients to their GP for systemic hypertension following a routine eye examination accounted for 2.4% of referrals, rising to 15.7% when BP readings were taken into account (Barnard *et al*. 1991). In the optometric profession, a survey of referrals of patients in 1986 found that 5% were referred for hypertension/arteriosclerosis compared to 12% for glaucoma (Port and Pope, 1988). Given that systemic hypertension is ten times more prevalent in society than glaucoma, this could imply that the optometric profession is poor at detecting and or appropriately managing possible systemic hypertension in their patients. Barnard *et al*. (1991) found that 18% of the patients referred to their GP for systemic hypertension were already on hypertensive medication and 31% presented with signs or symptoms warranting a review.

BP measurement is a more specific and reliable predictor of cardiovascular disease than ophthalmoscopy (Schubert, 1998). Bechgaard *et al*. (1950) examined 485 people with non-malignant hypertension (BP > 160/100 mm Hg or SBP > 180 mm Hg) and found 33% had no vascular fundus changes even after 10 years or more of hypertension, 37% had slight constriction and arteriolosclerosis (generally in older subjects), with retinopathy (haemorrhages, lipid deposits and one case of papilloedema) being rare (6%).

However, examination of the retinal vasculature is important, as the fundus appearance is a useful prognostic indicator of the general state of the body's vascular network via a non-invasive technique (Scheie, 1953). The degree of sclerosis also serves as an excellent index of the duration of hypertension and hence prognosis (Scheie, 1953). Therefore, retinal vasculature characteristics may be a clinical sign of greater value to the GP in the management of these patients than hypertensive retinopathy or BP measurement in some instances. With systemic hypertension being a major contributing factor to the leading killers in the developed world, there is a need for the optometrist to improve their interaction with the GP in the life long management of these patients as a whole. The use of improved grading scales or image analysis of fundus images may allow improved detection and monitoring of

systemic hypertension by optometrists in the future (Wolffsohn *et al*., 2001).

References

Aurell, E. and Tibblin, G. (1965). Hypertensive eye—ground changes in a Swedish population of middle-aged men. *Acta Ophthalmol.* **43,** 355–361.

Augsburger, A. R. and Good, G. W. (1986). Hypertension: an optometric problem for the 80s. *J. Am. Optom. Assoc.* **57,** 355–359.

Barnard, N. A. S., Allen, R. J. and Field, A. F. (1991). Referrals for vascular hypertension in a group of 45–64 year old patients. *Ophthal. Physiol Opt.* **11,** 201–205.

Bechgaard, P., Porsaa, K. and Vogelius, H. (1950). Ophthalmological investigations of 500 persons with hypertension of long duration. *Br. J. Ophthalmol.* **34,** 409–424.

Becker, R. A. (1981). Hypertension and arteriosclerosis. In: *Clinical Ophthalmology* (eds E. A. Jaeger and W. E. Benson), Harper and Row, Philadelphia, PA, pp. 1–26.

Black, H. R., Kuller, L. H., O'Rourke, M. F., Weber, M. A., Alderman, M. H., Benetos, A., Burnett, J., Cohn, J. N., Franklin, S. S., Mancia, G., Safar, M. and Zanchetti, A. (1999). The first report of the systolic and pulse pressure (SYPP) working group. *J. Hypertens.* **17** (5), S3–S14.

Breslin, D. J., Gifford, R. W., Fairbairn, J. F. and Kearns, T. P. (1966). Prognostic importance of ophthalmoscopic findings in essential hypertension. *JAMA* **195,** 335–338.

Brinchmann-Hansen, O. and Heier, H. (1986). The apparent and true width of the blood column in retinal vessels. *Acta Ophthalmol.* **64** (179), 29–132.

Brinchmann-Hansen, O., Christensen, C. C. and Myhre, K. (1990). The response of the light reflex of retinal vessels to reduce blood pressure in hypertensive patients. *Acta Ophthalmol.* **68,** 155–161.

Chen, H. C., Patel, V., Wiek, J., Rassam, S. M. and Kohner, E. M. (1994). Vessel diameter changes during the cardiac cycle. *Eye* **8,** 97–103.

Cogan, D. G. (1974). Ophthalmic manifestations of systemic vascular diseases. *Saunders* 74–80.

Collins, R. (1994). Hypertension treatment trials: update metanalysis. *Br. Med. Bull.* **52,** 272–290.

Daubs, J. G. (1974). Cost efficiency of optometric screening. *Opt. J. Rev. Optom.* **111,** 9–18.

Deupree, D. M., DeLori, F. C., Feke, G. T. and Weiter, J. J. (1985). Measurement of retinal blood vessel diameter. *ARVO Abstracts. Ophthalmol Vis. Sci.* **26,** 37.

Dimmitt, S. B., Eames, S. M., Goslin, P., West, J. N. W., Gibson, J. M. and Littler, W. A. (1989). Usefulness of ophthalmoscopy in mild to moderate hypertension. *Lancet* **1,** 1103–1106.

Dodson, P. M. and Kritzinger, E. E. (1987). Management of retinal vein occlusion. *BMJ* **295,** 1434–1435.

Dodson, P. M. and Kritzinger, E. E. (1997). Medical cardiovascular treatment trials: relevant to medical ophthalmology in 1997? *Eye* **11,** 3–11.

Dodson, P. M., Lip, S. M., Gibson, J. M. and Beevers, D. G. (1996). Hypertensive retinopathy: a review of existing classification systems and a suggestion for a simplified grading system. *J. Hum. Hypertens.* **10,** 93–98.

Duerey, N. M., Delori, F. C. and Gragoudas, E. S. (1979). Monochromatic ophthalmoscopy and fundus photography. *Arch. Ophthalmol.* **97,** 288–293.

Eaton, A. M. and Hatchell, D. L. (1988). Measurement of retinal blood vessel width using computerized image analysis. *Invest. Ophthalmol. Vis. Sci.* **29,** 1258–1264.

Evelyn, K., Nicholls, J. V. V. and Turnbull, W. (1958). A method of grading and recording the retinal changes in essential hypertension. *Am. J. Ophthalmol.* **45,** 165–178.

Farnett, L., Mulrow, C. D., Linn, W. D., Lucey, C. R. and Tuley, M. R. (1991). The J-curve phenomenon and the treatment of hypertension. *JAMA* **265,** 489–495.

Fletcher, A. E. and Bulpitt, C. J. (1992). How far should blood pressure be lowered. *New Eng. J. Med.* **326,** 251–254.

Fuchs, F. D., Maestri, M. K., Bredemeier, M., Cardozo, S. E., Moreira, F. C., Wainstein, M. V., Moreira, W. D. and Moreira, L. B. (1995). Study of the usefulness of optic fundi examination of patients with hypertension in a clinic setting. *J. Hum. Hypertens.* **9,** 547–551.

Gilchrist, J. (1987). Computer processing of ocular photographs—a review. *Ophthal. Physiol. Opt.* **7,** 379–386.

Goldberg, I., Hollows, F. C., Kass, M. A. and Becker, B. (1981). Systemic factors in patients with low-tension glaucoma. *Br. J. Ophthalmol.* **65,** 56–62.

Good, G. W. and Augsburger, A. R. (1989). Role of optometrists in combating high blood pressure. *J. Am Optom. Assoc.* **60,** 352–355.

Grundy, J. W. (1990). Hypertension—the facts. *Optometry Today* **30** (7th May), 10–14.

Guidelines Subcommittee. (1999). World Health organization—international society of hypertension guidelines for the management of hypertension. *J. Hypertens.* **17**, 151–183.

Hankey, G. J., Slattery, J. M. and Warlow, C. P. (1991). Prognosis and prognostic factors of retinal infarction: a prospective cohort study. *BMJ* **302,** 499–504.

Harris, M. G., Gan, C. M., Revelli, E. J., Imamura, C. and Kinoshita, B. (1994). Blood pressure measurement by eye care practitioners. *J. Am. Optom. Assoc.* **65,** 512–516.

Hart, W. E., Goldbaum, M., Cote, B., Kube, P. and Nelson, M. R. (1999). Measurement and classification of retinal vascular tortuosity. *Int. J. Med. Inf.* **53,** 239–252.

Hayreh, S. S. (1989). Classification of hypertensive fundus changes and their order of appearance. *Ophthalmologica* **198,** 247–260.

Hayreh, S. S. (1996). Duke-Elder Lecture: Systemic arterial blood pressure and the eye. *Eye* **10,** 5–28.

Hayreh, S. S., Servais, G. E. and Virdi, P. S. (1986a). Fundus lesions in malignant hypertension IV. Focal intraretinal periarteriolar transudates. *Ophthalmology* **93,** 60–73.

Hayreh, S. S., Servais, G. E. and Virdi, P. S. (1986b). Fundus lesions in malignant hypertension V. Hypertensive optic neuropathy. *Ophthalmology* **93,** 74–87.

Hayreh, S. S., Servais, G. E. and Virdi, P. S. (1986c). Fundus lesions in malignant hypertension VI. Hypertensive choroidopathy. *Ophthalmology* **93,** 1383–1400.

Hayreh, S. S., Servais, G. E. and Virdi, P. S. (1989). Cotton wool spots (inner retinal ischaemic spots) in malignant hypertension. *Ophthalmologica* **198,** 197–215.

Hayreh, S. S., Zimmermann, M. B. and Podhajsky, P. (1994). Incidence of various types of retinal vein occlusion and their recurrence and demographic characteristics. *Am J. Ophthalmol.* **103,** 429–441.

Hickey, N. and Graham, I. M. (1988). *Hypertension*, Croom Helm, London.

Hodge, J. V., Parr, J. C. and Spears, G. F. S. (1969). Comparison of methods of measuring vessel width on retinal photographs and the effect of fluorescein injection on apparent retinal vessel calibers. *Am. J. Ophthalmol.* **68,** 1060–1069.

Houben, A. J., Canoy, M. C., Paling, H. A., Derhaag, P. J. and

de Leeuw, P. W. (1995). Quantitative analysis of retinal vascular changes in essential and renovascular hypertension. *J. Hypertens.* **13,** 1729–1733.

Hubbard, L. D., Brothers, R. J., King, W. N., Clegg, L. X., Klein, R., Cooper, L. S., Sharrett, A. R., Davis, M. D. and Cai, J. (1999). Methods for evaluation of retinal microvascular abnormalities associated with hypertension/sclerosis in the atherosclerosis risk in communities study. *Ophthalmology* **106,** 2269–2280.

Kagan, A., Aurell, E., Dobree, J., Hara, K., McKendrick, C., Michaelson, I., Shaper, G., Sundaresan, T. and Tibblin, G. (1966). A note on signs in the fundus oculi and arterial hypertension: conventional assessment and significance. *Bulletin WHO* **34,** 955–960.

Kannel, W. B. and Sorlie, P. (1975). Hypertension in Framingham. *Epidemiology and Control of Hypertension. Stratton New York*, pp. 553–592.

Kannel, W. B., Drawber, T. R., Kagan, A., Revotskie, N. and Stokes, J. (1961). Factors of risk in the development of coronary heart disease; six year follow-up experience. *Ann. Int. Med.* **55,** 33.

Kanski, J. J. (1995). *Clinical Ophthalmology*, 3rd, Butterworth-Heinemann, Oxford, pp. 367–370.

Keith, N. M., Wagener, H. P. and Barker, N. W. (1939). Some different types of essential hypertension: their course and prognosis. *Am. J. Med. Sci.* **191,** 332–343.

Kergoat, H. and Lovaski, J. V. (1995). Response of parapapillary retinal vessels to exercise. *Optom. Vis. Sci.* **72,** 249–257.

Kishi, S., Tse, M. O. M. and Hayreh, S. S. (1985). Fundus lesions in malignant hypertension. I. A pathologic study of experimental hypertensive choroidopathy. *Arch. Ophthalmol* **103,** 1189–1206.

Klein, R. (1992). Retinopathy in a population-based study. *Trans. Am. Ophthalmol. Soc.* **90,** 561–594.

Klein, R., Klein, B. E. K., Moss, S. E. and Wang, Q. (1993). Blood pressure, hypertension and retinopathy in a population. *Trans. Am. Ophthalmol. Soc.* **91,** 207–222.

Klein, R., Klein, B. E. K., Moss, S. E. and Wang, Q. (1994). Hypertension and retinopathy, arteriolar narrowing, and arteriovenous nicking in a population. *Arch. Ophthalmol.* **112,** 92–98.

Klein, R., Klein, B. E. K. and Moss, S. E. (1997). The relation of systemic hypertension to changes in the retinal vasculature: the Beaver Dam Eye Study. *Trans. Am. Ophthalmol. Soc.* **95,** 329–350.

Leibowitz, H. M., Krueger, D. E., Maunder, L. R., Milton, R. C., Kini, M. M., Kahn, H. A., Nickerson, R. J., Pool, J., Colton, T. L., Ganley, G. L., Loewenstein, J. I. and Dawber, T. R. (1980). The Framingham Eye Study monograph: An ophthalmological and epidemiological study of cataract, glaucoma, diabetic retinopathy, macular degeneration, and visual acuity in a general population of 2631 adults, 1973–1975. *Surv Ophthalmol.* **24,** 335–610.

Leishman, R. (1957). The eye in general vascular disease: hypertension and arteriosclerosis. *Br. J. Ophthalmol.* **41,** 641–701.

Marshall, E. C. and Malinovsky, V. E. (1998). Hypertension and the eye: applications of the sixth report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. *J. Am. Optom. Assoc.* **69,** 281–291.

McGregor, E., Isles, C., Jay, J. L., Lever, A. F. and Murray, G. D. (1986). Retinal changes in malignant hypertension. *BMJ* **292,** 233–234.

Michaelson, I. C., Eliakim, M., Avshalom, A., Medalie, J. H., Ivry, M. and Neumann, E. (1967). An approach to the investigation of the vascular changes in the fundus of the eye in hypertension and arteriosclerosis. *Acta XX Conc Ophthalm Germania.* **1,** 207–219.

Newsom, R. S., Sullivan, P. M., Rassam, S. M., Jagoe, R. and Kohner, E. M. (1992). Retinal vessel measurement: comparison between observer and computer driven methods. *Graefes Arch. Clin. Exp. Ophthalmol.* **230,** 221–225.

North, R. V. (1999). Hypertension and its effect on the eye. *CE Optometry* **2,** 60–65.

Martin, E. A. (eds) (1994). *Oxford Medical Dictionary.* Oxford University Press.

Parr, J. C. and Spears, G. F. S. (1974). General calibre of the retinal arteries. *Trans. Ophthalmol. Soc. NZ* **26,** 55–60.

Pickering, G. (1974). Hypertension—definitions, natural histories and consequences. In: *Hypertension Manual* (ed J. H. Laragh), 1st, Dun-Donnelly Publishing Corporation, New York, pp. 3–30.

Port, M. J. A. and Pope, C. A. (1988). Referrals and notifications by British optometrists. *Ophthalmol. Opt.* **8,** 323–326.

Rassam, S. M., Patel, V. and Kohner, E. M. (1993). The effect of acetazolamide on the retinal circulation. *Eye* **7,** 697–702.

Rassam, S. M., Patel, V., Brinchmann-Hansen, O., Engvold, O. and Kohner, E. M. (1994). Accurate vessel width measurement from fundus photographs: a new concept. *Br. J. Ophthalmol.* **78,** 24–29.

Salus, R. (1958). Diagnosis of arteriosclerosis and hypertension. *Am. J. Ophthalmol.* **45,** 81–92.

Scheie, H. G. (1953). Evaluation of ophthalmoscopic changes of hypertension and arteriolar sclerosis. *A.M.A. Arch. Ophthalmol.* **49,** 117–138.

Schubert, H. D. (1998). Ocular manifestations of systemic hypertension. *Curr. Opin. Ophthalmol.* **9,** 69–72.

Sharp, P. S., Chaturvedi, N., Wormold, R., McKeigue, P. M., Marmot, M. G. and McHardy Young, S. (1995). Hypertensive retinopathy in Afro-Caribbeans and Europeans: prevalence and risk factor relationships. *Hypertension* **25,** 1322–1325.

Silagy, C. A. and McNeil, J. J. (1992). Epidemiologic aspects of isolated systolic hypertension and implications for future research. *Am. J. Cardiol.* **69,** 213–218.

Sixth Report of the JNC on detection, evaluation, and treatment of high blood pressure. (1997). *Arch. Intern. Med.* **157**, 2413–2446.

Svardsudd, K., Wedel, H., Aurell, E. and Tibblin, G. (1978). Hypertensive eye ground changes: prevalence, relation to blood pressure, and prognostic importance. *Acta Med. Scand.* **204,** 159–167.

Stokoe, N. L. (1975). Clinical assessment of the hypertensive fundus. *Trans. Ophthal Soc.* **95,** 463–471.

Stokoe, N. L. and Turner, R. W. D. (1966). Normal retinal vascular patterns. *Br. J. Ophthalmol.* **50,** 21–40.

Sugiyama, T., Schwartz, B., Takamoto, T. and Azuma, I. (2000). Evaluation of the circulation in the retina, peripapillary choroid and optic disk in normal-tension glaucoma. *Ophthalmic. Res.* **32,** 79–86.

Terry, J. E. (1976a). The interpretation of systemic arterial blood pressure. Part III: Hypertensive retinopathy. *J. Am. Optom. Assoc.* **47,** 1521–1531.

Terry, J. E. (1976b). The measurement of systemic arterial blood pressure. *J. Am. Optom. Assoc.* **47,** 571–577.

Terry, J. E. and Schoessler, O. D. (1976). Systemic arterial blood pressure and its relevance to optometric practice. *J. Am. Optom. Assoc.* **47,** 48–52.

Tso, O. M. and Jampol, L. M. (1982). Pathophysiology of hypertensive retinopathy. *Ophthalmol.* **89,** 1132–1145.

Wagener, H. P., Clay, G. E. and Gipner, J. F. (1947). Classification

of retinal lesions in the presence of vascular hypertension. *Trans. Am. Ophthamol. Soc.* **45,** 57–73.

Wallow, I. H., Bindley, C. D., Reboussin, D. M., Gange, S. J. and Fisher, M. R. (1993). Systemic hypertension produces pericyte changes in retinal capillaries. *Invest. Ophthalmol. Vis. Sci.* **34,** 420–430.

Walsh, J. B. (1982). Hypertensive retinopathy. Description, classification, and prognosis. *Ophthalmology* **89,** 1127–1131.

Weinberger, D., Stiebel-Kalish, H., Priel, E., Barash, D., Axer-Siegel, R. and Yassur, Y. (1999). Digital red free photography for the evaluation of retinal blood vessel displacement in epiretinal membranes. *Ophthalmology* **106,** 1380–1383.

Williams, T. D. (1982). Quantification of arteriolar tortuosity in two normotensives age groups. *Am J. Optom. Physiol. Optics.* **59,** 675–679.

Wolf, S., Werner, E., Schulte, K. and Reim, M. (1998). Acute effect of metipranolol on the retinal circulation. *Br. J. Ophthalmol.* **82,** 892–896.

Wolffsohn, J. S., Napper, G. A., Ho, S-M., Jaworski, A. and Pollard, T. L. (2001). Improving the description of the retinal vasculature and patient history taking for monitoring systemic hypertension. *Ophthal. Physiol. Opt.* **21,** 441–449.

Wu, D. C., Schwartz, B., Schwoerer, J. and Banwatt, R. (1995). Retinal blood vessel width measured on color fundus photographs by image analysis. *Acta. Ophthalmol. Scand. Suppl.* **215,** 33–40.

Yu, T., Mitchell, P., Berry, G., Weining, L. and Wang, J. J. (1998). Retinopathy in older persons without diabetes and its relationship to hypertension. *Arch. Ophthalmol.* **116,** 83–89.