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Early detection of glaucoma

Primary open-angle glaucoma (often loosely called "chronic glaucoma") is one of the main causes of preventable loss of sight in Britain[1 2] and throughout the world. All too often it is diagnosed only when loss of the visual fields is advanced and the condition is resistant to treatment.[3] Early detection is therefore of the highest importance, especially with recent evidence that appreciable neuronal damage has occurred even before tests show impairment of the visual fields and before the optic disc shows glaucomatous change.[4] After diagnosis careful lifelong monitoring is essential.[5]

The identification of primary open-angle glaucoma depends on three tests. Ophthalmoscopy detects glaucomatous cupping and pallor of the optic disc, tonometry measures the intraocular pressure, and perimetry permits detection of the characteristic visual-field defects.[6] Both tonometry and perimetry require special skill and apparatus, and testing the visual fields in particular is often time consuming.

This type of glaucoma is found in about 1% of the population aged over 40.[7–9] Surveys have shown that at this level of prevalence no one test provides a cost-effective screening measure: the sensitivity and specificity of each test individually are not good enough to prevent an unacceptably high yield of false-negative and false-positive results.[9 10] Any screening procedure which has a high probability of either missing the disease or overloading the hospital services with suspects who prove to be normal will rapidly come into disrepute.[9]

How then can the ophthalmic services be so organised that the three essential tests are carried out in a cost-effective manner? In Britain we are fortunate in having an existing National Health Service framework within which this objective can be achieved progressively. We need to make staff, time, and instruments available for the tests when in the examiner's judgment they are necessary. It is crucial to recognise factors which indicate the high-risk subgroup—namely, increasing age over 40; a family history of glaucoma, particularly in siblings[11–13]; diabetes mellitus[14 15]; high myopia[16]; haemorrhages on the optic disc; the presence of coronary disease; and a history of a bleeding episode requiring blood transfusion (M Schultzer and S M Drance, personal communication). In the presence of such factors the possibility of chronic glaucoma must be considered if patients with early disease are not to be missed.

In any endeavour rules and check lists have far less influence on the result than the morale of those actually carrying out the work. High morale depends on the aims being fully understood and seen to be worth while. The preservation of sight is the most fundamental of aims for the ophthalmic and optical professions, but the understanding of multifactorial diseases is always difficult.

How are patients suspected of having glaucoma identified at present? Four short papers have recorded experience in Middlesbrough,[17] Oxford (p 1091), Southampton (p 1093), and South London.[18] Patients being treated for primary open-angle glaucoma in hospitals in these areas have been studied and their primary source of referral traced. The results show the same general pattern. In about one-fifth of patients some ocular or visual disability was first detected by the general medical practitioner, in another fifth by ophthalmic medical practitioners, and in about three-fifths by ophthalmic opticians (optometrists). Almost all patients were ultimately referred to hospital by their general practitioners in the correct manner. The patients with glaucoma who had been primarily referred by their family doctors had usually sought advice for ophthalmic symptoms, though in only a quarter or less had the doctor suspected glaucoma, whereas the probability of glaucoma had been the basis of referral in about three-quarters of those, frequently asymptomatic, patients originating from ophthalmic medical practitioners and ophthalmic opticians.

The conclusion must be that detection of glaucoma will become more frequent when general practitioners have a heightened awareness of its importance in the middle aged and elderly and of the risk factors concerned. Diagnosis could be improved by the more frequent and informed use of the ophthalmoscope by doctors to identify optic discs showing glaucomatous features, even though the latter may sometimes be deceptive.[19] Though tonometry or examination of the visual fields clearly presents difficulty in general practice, this can be remedied by collaboration with ophthalmic medical practitioners and ophthalmic opticians whenever symptoms, risk factors, or appearances of the fundus make these tests desirable. Such a policy would reduce delay in the treatment of those requiring it while avoiding any swelling of the numbers of unnecessary hospital outpatient appointments.

In the 1966 survey of the whole population in Ferndale[7] only about one in three of those found to have glaucomatous visual-field defects had been previously detected. Some improvement may have occurred, but many people with field loss must remain unidentified. For this reason the patients' self-help

charity, the International Glaucoma Association, has included in its aims both the improvement of the general awareness of glaucoma, especially among those concerned with primary eye care,[20] and the encouragement of even closer collaboration among general practitioners, ophthalmologists, ophthalmic opticians, nurses and health visitors, medical auxiliaries, patients, and the public.

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The nature of piles

Few who reach middle age can claim never to have had any symptoms related to the anus. Thomson[1] has shown elegantly, if not originally, that what many regard as piles are normal vascular cushions. We all have them, and they are as natural as the vascular cushions at the upper end of the alimentary tract that we call lips. We are prepared to accept a wide variety of lips: thin lips, pouting lips, petulant lips, wet lips, and even hot lips. Similarly, variations in the vascular cushions at the anus should possibly be regarded as signs of character rather than disease. Furthermore, the degree of congestion of our cushions depends on factors as varied as hormonal, biochemical, emotional, and, for all I know, barometric. Who can say then what is the incidence of piles in the population? Perhaps the most accurate we can be is to say that they are ubiquitous.

What we, as doctors, are concerned with is not an anatomical or pathological entity but a collection of symptoms. The very words we use imply this. "Piles" comes from the Latin for a pill or a ball and implies the complaint of a lump, either a prolapsed or thrombosed cushion. "Haemorrhoids" comes from the Greek for the flowing of blood—a symptom if ever there was one. To the layman and the comedian, piles implies pain, though pedantic proctological purists may deny this. To those symptoms mentioned we must add, for completeness, itching and discharge. One or more of the five constitute what our patients mean when they "suffer from piles."

What converts these normal functional God-given cushions to symptomatic monsters? The two principal factors are the laxity of the fixation of the vascular mucosa to the wall of the rectum and upper anal canal and the muscular tone or tightness of the anal sphincter. The fixation of mucosal cushions is normally very lax, like the skin at the back of a kitten's neck. The cushions may even have muscular fibres within them that pull the cushion back to the safety of the upper anal canal after defecation.[1] Once the anal cushions are submitted to congestion they hypertrophy with engorgement—just as do the upper alimentary lips of some African ladies who value labial hypertrophy and use metal or leather constriction to achieve it. The anal canal may be tight because of muscle tone or, some would have us believe, fibrosis.[2] What causes variations in tone is not clear, but measurement of resting pressures in the anal canal indicates that they are normally higher below the age of 60. I suspect that emotional tension plays a part and I have a syndrome that I designate with the eponym YETAS, the "Young executive tight-anus syndrome." Certainly this seems to be one group of people prone to piles; others are golfers and professional cricketers.

In addition to spasm there may be true fibrosis in the anal sphincters, though this has never been shown histologically. An easily confirmed observation from life's vast store of observables is that young children, left to their own devices, will pass stools of such a width as to frighten their mother but not daunt themselves. The close ratio of stool-to-arm diameter in childhood must surely be normal. In the Western developed world, the much smaller ratio in adults probably represents the sequel of an unbalanced diet. Men who supplement, or substitute for, their food with cathartic beers often have anal symptoms, though I must admit that the lesions are more often perianal dermatitis or fissure than piles. I use for this another eponym, "bitterman's bottom." Such patients' anal symptoms respond dramatically to the restoration of anal resilience and elasticity that follows a manual dilatation.

The secondary pathological changes that accompany protrusion and congestion provoke the complications and principal symptoms of piles. Congestion may cause discomfort from increasing tissue tension; hypertrophy facilitates further protrusion and impedes withdrawal into the anal canal. The congested cushions are easily injured and bleed as readily and as vividly as does a cut lip. Severe unrelieved congestion predisposes to thrombosis giving rise to that proctological "full-house," strangulated piles.

Cushions that are so large that they do not return to the anal canal after defecation will leave exposed mucous membrane with consequent discharge and skin maceration; bacterial or fungal invasion may then cause pruritus.

An understanding of the nature of piles and the causation of symptoms enables us to devise rational treatment. If we accept that mucosal laxity and sphincter congestion are the two