division is performed to allow total gut assessment, not in the hope of curing or preventing symptoms. A single tough band like adhesion should, however, be divided to prevent possible strangulation. In my opinion there is no scientifically reliable evidence in man that washing out the peritoneum or instilling antiseptics, anti-inflammatory drugs, or antibiotics reduces adhesion formation.

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Wellcome developments in tropical medicine

The seemingly conflicting aims of Sir Henry Wellcome to provide educational entertainment for the casual visitor and serious research material for the student continue to be fulfilled by the Wellcome Tropical Institute. Established by the Wellcome Trust in 1984 the institute is working, firstly, to update and develop the Wellcome Museum of Medical Science and, secondly, to help governments and universities in the tropics to run their own courses in tropical medicine and to develop continuing education for medical officers away from teaching hospitals.

The two ventures are closely intertwined. As well as revising existing museum material and acquiring fresh exhibits the institute hopes to hold special exhibitions every two years on topics of current interest in tropical medicine. The first, on malaria, opens next week and is both visually striking and informative (p 709). It has clear, detailed diagrams and micrographs of the stages in the life cycle of both the anopheles mosquito and the plasmodium parasite and a broader overview of the problems of control and treatment. As well as inviting sixth formers and undergraduates to visit the exhibition the institute is sending copies of the exhibition panels to medical schools in Africa. Schools in Nairobi and Addis Ababa have already received material.

More ambitious is the institute's aim to develop a distance learning programme for continuing postgraduate education. The programme, devised for district medical officers in rural areas, is being worked out in collaboration with governments and universities to ensure that the methods of self instruction, including slide-sound and video, are suitable. As Dr Alan Knell, deputy director of the institute, points out, sending display materials over is easy compared with implementing the programme at the other end. More input and organisation are needed. Nevertheless, plans are progressing, and Dr Knell hopes that programmes will start later this year in Kenya and Addis Ababa. Sir Henry, I feel, would approve.

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Through the carpal tunnel

The carpal tunnel syndrome is the commonest entrapment neuropathy, affecting the median nerve as it lies between the carpal bones, the flexor tendons, and the unyielding transverse carpal ligament. A general practitioner may expect to see on average two patients each year with the syndrome.[1] It affects women between two and 10 times more often then men, is most common between 30 and 50 years of age, and is bilateral in one third of patients.[1]

The carpal tunnel syndrome is secondary to other conditions in 10-50% of patients, and in general practice it is seen most often with pregnancy, the premenstrual syndrome, and obesity. The tunnel may be narrowed by disruption of its bony architecture, by thickening of the synovium and flexor tendon sheaths, or by tumours and deposits. Thus the syndrome may be secondary to Colles' fracture and other bony deformities (as first described by James Paget[2]); to rheumatoid arthritis, and arthritides caused, for instance, by rubella, or to tuberculous tenosynovitis; to lipomas, and to amyloid or tophaceous deposits; and to the increased venous engorgement caused by inserting an arteriovenous shunt for haemodialysis. General conditions that affect the carpal tunnel include myxoedema, acromegaly, diabetes, polycythaemia vera, and haemolytic anaemias.

Diagnosis in general practice depends on an accurate history, examination, and various provocative tests. In hospital these are supplemented by electrodiagnosis. The characteristic symptoms are nocturnal tingling and pain in the thumb, index, and middle fingers of one or both hands; the paraesthesia may spread to other fingers, the hand, and the forearm and may be accompanied by numbness. Often symptoms are precipitated by unaccustomed use of the hands and initially are relieved by shaking the hand or hanging it out of bed. Stiffness or clumsiness of the fingers is experienced by less than half of patients, and few complain of swollen fingers.

Usually nothing abnormal is found on examination. Thenar wasting is present in only one third of patients, and less than one sixth of patients have objective hypoaesthesia of the finger tips. Provocation tests are thus an essential adjunct to diagnosis. In the Phalen test symptoms are produced or exacerbated by complete flexion of the wrist for 30-60 seconds.[3] A positive Tinel's test demands producing tingling in the fingers by gently tapping the area over the median nerve in the wrist. In the tourniquet test a blood pressure cuff applied to the upper arm is inflated above systolic blood pressure for 60 seconds; the production or exacerbation of symptoms in the fingers means the test is positive. Nerve conduction studies may show reduced conduction velocities in the median nerve as it crosses the wrist, with changes in sensory nerves preceding those in motor nerves; distal motor latency time may be prolonged. Alternatively, results may be within normal limits but show slower nerve conduction on the affected side.

Gellman and colleagues have recently assessed the sensitivity and specificity of provocation tests in comparison with electrodiagnosis.[4] Phalen's test was most useful—positive in three quarters of patients and falsely positive in only 20% of controls. The tourniquet test was the least reliable—only 65% of patients were positive and 40% of controls were falsely positive. Tinel's percussion test was interesting because, although relatively insensitive with only 44% of patients positive, it was highly specific with only 6% of false

positive controls. Mossman and Blau (p 680) studied Tinel's sign in a smaller group of patients and showed that the sensitivity of the test was increased by extending the wrist, to tense the contents of the carpal tunnel, and by percussing with a broad based "Queen Square" tendon hammer. The proportion of false positive results was not increased.

Treatments for carpal tunnel syndrome include rest, night splints, diuretics, steroid injection into the carpal tunnel, and wide surgical division of the flexor retinaculum. Many hospital studies report a poor or only temporary response to non-surgical treatment, but uncontrolled experience from general practice suggests some benefit from rest and diuretics and considerable benefit from one or two injections of hydrocortisone for patients who have only acroparaesthesia. Perhaps some patients with milder symptoms have self limiting swelling of the synovium and flexor tendon sheaths and reach hospital less often. An operation is essential, however, if the patient has muscular weakness or wasting. Dysaesthesia is common after operation, and sensory recovery may take up to 12 months; in contrast, there may be little or no motor recovery.

Gellman's study is a stimulus and a challenge. The doctor must progressively refine his differential diagnosis, and to do this he needs to choose those tests that most quickly and certainly discriminate between one condition and another. General practice teachers have emphasised the need to know the specificity as well as the sensitivity of factors that constitute a diagnosis.[5] Yet, although there are some data on the prevalence of symptoms in healthy people,[6,7] the specificity of the various parts of the clinical examination and tests used by doctors are largely unknown. This study is a small but useful step in the right direction.

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Retinal detachment

Retinal detachment is the separation of sensory retina from the retinal pigment epithelium. Fluid accumulates between the two layers, entering usually from the vitreal compartment through a break in the sensory layer. The appreciation of the importance of the retinal break over 60 years ago[1] has led to high success rates in treatment. Rarely there is no break, and fluid accumulates through effusion from choroidal blood vessels (non-rhegmatogenous detachment).

Retinal breaks are usually a consequence of degeneration in the vitreous. Normally the vitreous is closely applied to the inner surface of the retina, but with degeneration it may collapse and peel away (vitreous detachment). This process is not necessarily harmful, but traction may be exerted on the retina; on normal movement of the eye the vitreous is set in motion and tractional forces acting on points of vitreoretinal attachment may result in retinal breaks and detachment. Patients who have vitreous degenerative change are thus more likely to suffer retinal detachment. The most common predisposition is myopia, and the higher the myopia the greater the risk of detachment.[2] Others at risk are those who have cataract extractions, particularly if vitreous is lost,[3] although the modern extracapsular technique (with or without intraocular lens implantation) reduces the tendency for retinal detachment.[4,5] Injuries to the eye—either penetrative or contusive—may also result in detachment. Retinal breaks may also be produced as a result of degenerative change (for example, lattice degeneration) in peripheral retina.

About half the patients who develop retinal detachment have one or more of the classic premonitory symptoms. Flashes of light, usually in the temporal field of vision, are caused by vitreous traction on the underlying retina. A sudden shower of black spots—either previously unseen or appreciated as an increase in pre-existing floating opacities—is caused by a retinal blood vessel rupturing when the retinal break forms. A field defect will be experienced after the retina has detached and fluid accumulated between its layers. This field defect, initially peripheral, will usually extend rapidly over hours or days, resulting in almost complete loss of vision in the affected eye. In some cases retinal detachment will not present with the classic symptoms, and the most common atypical presentation is a generalised reduction of visual acuity. This is the usual presentation in children.

Before the introduction of modern surgery retinal detachment almost always led to blindness in the affected eye. Today an experienced surgeon will achieve about a 95% reattachment rate when the patient is seen quickly, but the condition is still one of the least welcome ophthalmic emergencies owing to the complexities of management. A successful operation will lead to a satisfactory field of vision being restored, but the extent of recovery of central vision depends on whether and for how long the macula has been detached. The longer the macula detachment the poorer the prognosis.[6]

Modifications to the surgical principle of closing the retinal break and creating an adhesion between the sensory retina and pigment epithelium have progressed apace. In simple cases solid silicone rubber or soft silicone sponge explants are sewn on to the outside of the eye, resulting in buckling of the sclera, choroid, and pigment epithelium towards the detached sensory retina. Correct buckling relieves vitreous traction near the retinal breaks and closes them. Cryotherapy effects a secure adhesion between the sensory and pigment epithelial layers.[7] Sometimes subretinal fluid is drained from the eye, which can be combined with injecting an air bubble into the vitreous cavity to encourage the sensory retina to move towards the pigment epithelium.[8,9]

If the retina is detached for long or if surgical reattachment fails the detached retina becomes extensively infiltrated with a growth of fibrous cellular membranes. The contraction of these membranes converts the detached retina from an undulating freely mobile curtain to a fixed, more board like structure and makes surgical reattachment complicated. In more difficult types of detachment—awkward retinal breaks, opacities in the media, and a fixed immobile retina —an internal approach is used and the vitreous removed (vitrectomy).[10,11] Membranes can be dissected from the retina and materials introduced into the vitreous cavity to