tuberculosis, and to a slowly growing extent in other departments, such as maternity and child welfare. The rapid advance in pathological science in so many conditions not recognized in the past as coming within the direct purview of Public Health Authorities has clearly demonstrated that systematic centralized pathological aid should be within the reach of everyone, being essential to any complete scheme of preventive medicine.

THE MANAGEMENT AND TREATMENT OF INCIPIENT CATARACT.*

BY

L. VERNON CARGILL, F.R.C.S.,

SENIOR OPHTHALMIC SURGEON, KING'S COLLEGE HOSPITAL.

THE treatment of early cataract has always exercised the minds of ophthalmic surgeons; it is a matter of daily importance to us and our patients, and a very great deal has been written and said about it. This is an opportune occasion for a renewal and exchange of our opinions and a confirmation or otherwise of our beliefs.

Incipient cataract is a condition in which localized grey opacities have appeared in the lens during adult life, and for which there is no obvious constitutional or local cause, either injury or disease. Such cases, for want of more precise knowledge, are usually labelled "senile cataract."

We might also include in our discussion the prodromal state preceding the development of actual opacity. Here there is an appearance of varying spots and lines on mirror illumination at different angles; fundus details may appear distorted by irregular lental astigmatism; fundus illumination may be relatively poor; the pupil reflex greyish; and a central dark circular zone may show on transmitted light. These phenomena we know are due to the formation of fluid clefts between the cortical lens fibres and to nuclear sclerosis.

In examining the lens I always employ the direct ophthalmoscopic method with +16 sph. behind the aperture, and use the slit-lamp if necessary for further details. On the discovery of lens changes we look for other accompanying senile ocular conditions such as vitreous degeneration, colloid bodies in the lamina vitrea, and macular or circum-papillary atrophy. We eliminate such complicating eye diseases as uveitis, retinitis pigmentosa, and detached retina, in which the lens opacity is likely to take the form of a subcapsular rosette at the posterior pole. We also exclude such special cataracts as "ray" cataract and those associated with dystrophia myotonica and parathyroid tetany.

Some forty years ago it was the custom in cases of immature cataract for some ophthalmic surgeons to combine preliminary iridectomy with artificial maturation of the cataract according to Foerster's method. The anterior chamber being emptied, the lens was massaged by rubbing the front of the cornea with a cataract spoon. The object was to avoid extracting an unripe cataract. Among these so-called "immature cases" there were probably many with useful vision which we should now classify as "incipient." The method was uncertain in its results; it did not affect the posterior cortex; it was liable to cause iritis, rupture the anterior capsule, or subluxate the lens with resulting vitreous loss when extraction was performed. It is unnecessary because we can extract immature cataracts successfully, and it is wrong because we cannot tell how slowly opacity will develop. In a paper on "The prognosis of incipient cortical opacities of the lens," Jessop drew attention to cases of striated cataract in which progress was extremely slow, and which he thought might be described as "arcus senilis lentis." Speaking of the terror of progressive blindness which the word cataract implies in the minds of the laity he advanced the opinion that a surgeon would be perfectly justified in saying that these were not cases of cataract if the patient asked the question. The case was cited of a patient who had 6/12 thirty years after being first told of cataract.

In this connexion the most striking fact, I think, in going through old records is the considerable number of patients over 60 years of age affected with incipient cataract, and yet the very small number of cases which reach the operative stage. Many cases that I have been watching for from ten to twenty years, or longer, have still useful vision (6/12 or more), and many others that have died were still seeing well.

Lens opacities are physiological in old age, and may be compared to white hair, baldness, and wrinkled inelastic skin. Old age is a relative term; it may be very premature or much deferred, and so we may find lens opacities acquired in middle age or even earlier. This applies particularly to "coronary" cataract, which occurs frequently, and may be found so early as to merit the term "pre-senile"; its course may be so gradual as to seem almost stationary.

It is our duty, therefore, in cases of incipient cataract to avoid frightening our patients with the dreaded word "cataract" when progress may be so slow and useful vision last so long. Where there is no appreciable defect in vision, as in coronary, striated, or dotted cataract, nothing need be said, unless to a discreet relation or friend.

With defective vision, perhaps progressively diminishing, as in the cuneiform and radial-spoke forms of cataract, and in posterior saucer-shaped cataract, some explanation will be necessary, and then, as Story has remarked, it becomes a matter of "diagnosing and treating a patient, not a disease." With a sensible patient a true statement and clear explanation of the lens opacity, illustrated by a diagram or model, I have always found the best course. With a highly nervous individual we should speak guardedly; as, for instance, of "cracks" or "flaws" in the lens, "such as come to all with age." I know a patient who completely collapsed when told there was evidence of cataract in her eyes; and another, a nervous excitable woman, who perpetually vowed eternal vengeance against an oculist who bluntly told her she had cataract many years before an operation was needed. She suffered a life of apprehension from that day. We must on no account make our cataract patients hypochondriacs and take all the hope out of their lives. We should rather comfort them with a cheerful optimism. It is dangerous to predict when operation will be necessary. A patient who had been told by an eminent oculist that she would probably need an operation within five years was managing very well twenty years later with 6/9 and Jaeger 1 in one eye and 6/18 and Jaeger 8 in the other, and she was then 85 years old. I could give other instances.

As a matter of course we seek to obtain the best possible vision with optical corrections, and we may to some extent be retarding the disease by relieving accommodation strain. We also retest periodically and alter our corrections according to the demands of a changing lental refraction; hypermetropia may become less, myopia develop or increase, and astigmatism may alter. The use of Crookes's, feuzal, or amber glasses in outdoor glare may help vision and protect the eyes from that radiant energy which is considered a fundamental predisposing factor in causing cataract. As lenticular opacity develops we instruct the patient how to get the best results from illumination, more intense for marginal and less intense for central opacities, and we determine if a weak mydriatic is any help. Incidentally, we watch tension, and should it rise from a swelling lens, iridectomy may need consideration. Reading vision may be improved with extra power in the presbyopic glasses, convergence for the nearer distance being relieved by prisms. The hand magnifier, or the "reading screen" made by Hawes, may be helpful, or both together; and I have known Galilean spectacles useful, but in such cases we are passing beyond the stage of incipient cataract.

What guidance in dealing with cataracts do we derive from pathological studies and researches into the constitution and properties of the lens? We know that the lens derives its nutrition entirely by endosmosis from the intra-ocular fluid. This fluid may be affected by any abnormal or toxic state of the capillary blood from which it is derived, and changes in the permeability of the capillary walls will also affect its character, while changes in the permeability of the lens capsule or in the epithelium lining

*Read in opening a discussion in the Section of Ophthalmology at the Annual Meeting of the British Medical Association, Winnipeg, 1930.

will affect the normal metabolic interchange between the fluid and the lens. Changes in the ciliary epithelium, if it has a selective function, would also have their influence; but according to the latest researches the intraocular fluid is a "dialysate" from capillary blood, and passes into the eye direct from the vessel walls without the intermediation of ciliary epithelium, or glands of the ciliary body.

According to Friedenwald's researches, the permeability of the lens capsule decreases with age, and metabolic processes may be thereby sufficiently obstructed to cause cortical cataract. Defective metabolism may damage the vital capsular epithelium, causing degeneration and vacuolation; and it disturbs the delicate autoxidation process of cell metabolism within the lens, so that in cataract "glutathione," which is specifically concerned in the chemical defence of the body, entirely disappears. According to Adams, in experimentally produced naphthalene cataract, which resembles senile subcapsular cataract, the cause appears to be a metabolic disturbance in the balance of the inorganic constituents of the body, and in these cases calcium oxalate crystals (like synchysis scintillans) were deposited in the posterior layers of the vitreous and on the surface of the retina. The potassium in cataractous lenses is a tenth of that in normal lenses (Burdon Cooper), and the calcium is increased (Adams), but some experiments have not given results sufficiently definite to associate parathyroid deficiency with ordinary senile cataract (Pellathy). With age there is a decrease in the soluble lens proteins (α and β crystalline) while the insoluble proteins (albumin and albumoid) increase, and nuclear sclerosis, comparable to cuticular keratinization, increases in extent and degree. When cataract develops there is hydrolysis and autolysis of lens fibres and coagulation of lens protein. The problem is very complicated, but we are hoping to learn some day from the biochemist the exact nature of the physico-chemical processes of normal lens metabolism and the derangements which mark the onset of cataract. But shall we ever be able to prevent cataract any more than senility and death?

It is evident that we have little to guide us in direct local medical treatment; and no treatment will remove actual opacity, for that represents the coagulation of lens protein and is "an irreversible chemical change" (Duke-Elder). I have no notes of any case in which definite lens opacities have disappeared. Improved visual acuity in the course of a case must be due to other factors, changes in hydrolysis or metabolism, and in the intimate relationship of vacuoles, clefts, and transparent fibres. It may in some cases be associated with better health, less near work, and a more restful life. Dor, with the idea of causing lens dehydration, tried eye baths of calcium iodide (made by a solution of 1 per cent. crystallized calcium chloride with 1 per cent. desiccated sodium iodide) and reported retarded evolution, often for a long time. But he also advised attention to stomach and teeth. His method has been continued and its adoption extended in the form of ointments. I have tried these methods, but never satisfied myself that they did much good. Dionine has been tried for its lymphagogue effect. Some use potassium iodide in drops or subconjunctivally, and this may be justified by the diminished potassium content of cataractous lenses. Some reinforce local treatment with potassium iodide or tiodine internally. Van der Hoeve is said to have found that potassium iodide counteracts the swelling of the lens vesicles. It is difficult to assess the value of such local treatment in a particular case, seeing that without it progress may be so slow and variable and that there may be periods of apparent improvement. Prescribed with proper qualification it is a useful placebo; it comforts and satisfies certain patients that something is being done and helps to save them from unscrupulous quacks. On the other hand there are others who are better without a daily reminder of their incipient cataracts.

Smith eulogizes subconjunctival injections of neutral mercury cyanide (1 in 2,000 to 1 in 5,000). He thinks that the resulting intense hyperaemia of the ciliary body re-establishes normal metabolism. He holds that it will cure 95 per cent. of early cases, but agrees that it does

not remove lens opacities. It will be interesting to hear the experience of any who have tried this method. Ionization, radium, x rays, electricity, and local radiation have all been tried without success, and some are likely to do more harm than good. Lens antigen treatment has also proved useless. The existence of incipient cataract may lead us to the discovery of some complicating constitutional dyscrasia, hormonal defect, or focal condition which needs treatment, and the removal or treatment of which may stay or retard the lenticular changes. More good is likely to accrue in this indirect way than by any local medical treatment. There may be general debility or premature senility needing investigation, diet and hygiene may need attention, or there may have been some debilitating disease. Arterio-sclerosis may exist, and the condition of the blood vessels is of greater consequence than the pressure of the blood inside them. The urine should be examined as a general rule, and always in developing cases. Sugar may be discovered, and expert laboratory tests will then be required to determine if it is renal glycosuria and negligible, or whether there is mild or severe diabetes, the former requiring merely restriction of diet and the latter insulin.

Cases of senile cataract with diminished sugar tolerance are indistinguishable from ordinary senile cataract and differ in slit-lamp appearances from true diabetic cataract. In my experience, insulin treatment does not improve the lens condition appreciably, any more that it does the retinitis, although it may retard further changes, just as it prolongs life. Albuminuria also demands a medical examination, and in advancing cataract it is usually wise to have the patient generally overhauled.

We should not overlook evidences of endocrine deficiency and especially hypothyroidism. The thyroid is the great regulator of metabolism, and any obvious deficiency should be suitably supplemented. The function of the parathyroids is to destroy active waste products and regulate calcium appropriation, and by their power of stabilizing calcium in the blood they may check the tendency to the excessive lental calcium found in cataract. If there is insufficient parathyroid hormone the blood cannot hold so much calcium in solution, hence its deposition in the lens among other tissues; it has been thought that this may have some connexion with the development of cataract. Parathormone has, however, proved disappointing in arresting senile cataract, and observations have been made which seem to show that lens changes in chronic parathyroid deficiency may be primarily of another nature, and the deposition of calcium salts quite secondary. In parathyroid tetany the blood calcium falls to half the normal, whereas in ordinary cataract blood calcium is little affected. However, Siegrist recommends the trial of a preparation containing thyroid, parathyroid, and testis with calcium lactate.

As in some other eye affections it is, I think, of the greatest importance to eliminate chronic toxæmia and search for infective foci. The toxins of streptococci and coliform bacilli are commonly the infective agents. The teeth, tonsils, sinuses, gastro-intestinal and genito-urinary tracts, including the pelvic organs in women, should all be passed in review. In the mouth, crowned teeth, dead teeth, and stumps should be looked on with suspicion, and as apical abscesses are more potent for evil than pyorrhoea, radiograms are advisable when there is any doubt. I have seen cataract progress with unusual rapidity in the presence of dental sepsis. In the young, lamellar cataract has been caused by prolonged septic infection (mastoid and cerebral abscess), and such a toxic condition is likely to have a detrimental effect on the nutritional osmosis of an unstable adult or senile lens, especially where the sepsis is bottled up in the body. In elderly patients intestinal stasis, chronic constipation, and colitis are not uncommon, and should receive proper attention; and bacilluria should not be overlooked. It is not only our duty to have all these matters attended to in pursuing the treatment of incipient cataract, but we are thereby improving the general health and well-being of the patient, and ensuring a condition of vitality and resistance which will promote the success of a future extraction, should it be needed.

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THE DIAGNOSIS OF CANCER OF THE LARYNX.*

BY

EDWARD D. D. DAVIS, F.R.C.S.,

SURGEON, NOSE, THROAT, AND EAR DEPARTMENT, CHARING CROSS HOSPITAL.

EXTRINSIC laryngeal cancer or cancer of the pharynx which has extended to the larynx usually comes under observation so late that the diagnosis is obvious. These cases are generally seen by the laryngologist much too late for successful treatment either by operation or by radium, and earlier diagnosis is urgently needed. The problem of early diagnosis is intensified by the fact that cancer in itself does not produce any specific signs or symptoms, and it is only when the cancer cells form a tumour or ulcer that a series of mechanical symptoms and signs appear. These symptoms also develop slowly and insidiously, and it is unusual for the patient to consult a doctor until such symptoms are well marked and until it is often too late for a successful removal of the growth. Moreover, in a hidden cavity like the pharynx where there is room for the growth to expand unseen, the first symptoms are trivial and may be neglected until secondary growths are manifest in the cervical glands.

The intrinsic cancers of the larynx are seen at an earlier stage, as the early symptom of hoarseness sends the patient to the doctor much sooner, but cancer seen in its earliest stage is more difficult to diagnose accurately. Hence cases of intrinsic laryngeal cancer present more frequent and more difficult problems in accurate diagnosis. I propose, therefore, to spend most of the time available in discussing the diagnosis of intrinsic cancer of the larynx. It is to be hoped that the more frequent use of the modern directoscope and direct laryngoscope, and suitable cancer propaganda, will produce the much needed earlier diagnosis of extrinsic cancer. The earlier stages of these cases may then present as many problems in diagnosis as the intrinsic cases.

Extrinsic Cancer.

The characteristic and offensive epitheliomatous ulcer commencing just below the aryteno-epiglottidean fold and rapidly involving that side of the larynx, produces fixation of the vocal cord with its consequent hoarseness. The first symptoms are sore throat and pain radiating to the ear, with a definitely tender spot near the greater cornu of the thyroid cartilage; then follows the characteristic hoarseness or roughness of the voice, with a copious frothy blood-stained expectoration. The top edge of the growth can be seen with the laryngoscope and palpation with the finger reveals the stony hard ulcer.

A post-cricoid carcinoma, which commences as a superficial growth on the posterior aspect of the cricoid cartilage in women, soon surrounds the lumen of the pharynx and spreads upwards to form the whitish warty upper edge seen in the later stages behind the oedematous arytenoids. These patients first complain of a soreness or pricking sensation with pain in the ears during swallowing. Pressure on the cricoid backwards against the spine or lateral movement of

the larynx produces pain in practically all cases. Dysphagia occurs early and when these symptoms are present without a satisfactory explanation the patient should be examined with a directoscope or oesophagoscope. The laryngoscopic mirror will not show any signs in the early stage, and when the upper edge of the growth is visible the disease is advanced, and matters are still more hopeless when a vocal cord is paralysed. Neither is the growth within reach of the longest finger, and the passage of a bougie is valueless and even harmful. In the absence of physical signs, the dangerous diagnosis of neurasthenia is still made; but such a diagnosis should never be concluded until the presence of a growth has been eliminated by a direct examination.

Carcinoma in the vallecula, either on the base of the tongue or on the anterior surface of the epiglottis, excavates the base of the tongue and rapidly extends to the surrounding structures, and the submaxillary and deep cervical lymphatic glands of both sides are soon involved. The only symptoms are persistent severe pain and a catch on swallowing located by the patient to the base of the tongue, with blood-stained salivary expectoration. Occasionally a chronic paroxysmal cough is the first symptom. Later, the painful and limited protrusion of the tongue with the accompanying trismus impedes laryngoscopic examination, but the growth is within easy reach of the palpating finger and does not require a skilled laryngologist or endoscopist to detect and gauge the extent of the growth. The situation, the characteristic appearance, the odour, and the history of these growths make the diagnosis more or less obvious; but the differential diagnosis should always be considered, as I have seen two cases of syphilis in the post-cricoid region which have been mistaken for carcinoma.

Intrinsic Cancer.

A rapid and accurate diagnosis of a typical case of intrinsic cancer of the larynx is easily made. The characteristic miniature epitheliomatous wart or ulcer situated on the middle or anterior third of the vocal cord in a patient of cancer age, in which the differential diagnosis of chronic laryngitis with pachydermia, tuberculosis, syphilis, and a simple tumour can be undoubtedly excluded, presents no difficulty. Such a growth has been and is frequently removed by operation without a pre-operative diagnostic section. On the other hand, the atypical case or the hidden subglottic growth requires considerable ingenuity in diagnosis. The differential diagnosis may be obscure, and tuberculosis is more often mistaken for cancer than any other condition. A chronic tuberculous ulcer of the vocal cord in a man of cancer age, or a healed scarred tuberculous lesion which has broken down with a recrudescence of the disease, closely resembles cancer.

Lupus, owing to its distribution in the soft tissues, is more readily distinguished; but in both cases tubercle bacilli cannot always be found in the sputum. In a few cases of tuberculosis it is almost impossible to make an accurate diagnosis without microscopy. The guinea-pig test by inoculation from the piece removed unfortunately takes six weeks to mature, but it is so conclusive that it might be useful. Chronic laryngitis with localized pachydermia renders diagnosis difficult, but the long history, the type of patient, the slow progress with a tendency to a wide distribution, gives the clue.

Syphilis is readily differentiated from cancer. The presence of other syphilitic lesions, the history, and the Wassermann reaction indicate the correct diagnosis; but when cancer is grafted on an old syphilitic lesion, the diagnosis may be difficult and can only be settled by the aid of the microscope.

Benign or simple tumours are recognized by their appearance, their situation, and slow growth. These tumours are usually removed by intra-laryngeal methods, and if the section for microscopy in any way suggests malignancy a more extensive operation can be done. Occasionally a malignant tumour has been labelled by the section to be benign, or a papilloma has recurred and has become locally malignant.

Sarcoma of the larynx is fortunately very rare. I have only seen two cases, both in young adults. The tumours were smooth, sessile, globular, and vascular, and occupied

* Read in opening a discussion in the Section of Laryngology and Otology at the Annual Meeting of the British Medical Association, Winnipeg, 1930.