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The KINGSTON MEDICAL QUARTERLY is presented to the Medical Profession with the compliments of the Editorial Staff. Contributions will be gladly received from members of the Profession and willingly published. JOHN HERALD, Editor

THE ONTARIO MEDICAL COUNCIL AND DOMINION REGISTRATION.

WE learn by the public press that Dr. Roddick was present at the meeting of the Ontario Medical Council in June last, and that he addressed the members on the subject of Dominion Medical Registration with special reference to the provisions of the Bill which he introduced into the Dominion Parliament last session. A discussion followed Dr. Roddick's address. Certain suggestions were made by members of Council whereby it was thought that the Bill would be made more fair to the various bodies whose interests are to be affected by the proposed legislation. Some of these suggestions, we understand, Dr. Roddick accepted and agreed that they would be incorporated in his Bill when he re-introduced it to the House next session. We wish to say a few words regarding these proposed amendments and again to call attention to what we still consider defects in the Bill. Our present criticism is based upon the reports of the meeting contained in the public prints. If we have in any particular formed a wrong conception of what the Council proposed and Dr. Roddick agreed to incorporate in the Bill we shall make the correction in our next issue. As we understand these proposed amendments at present, however, we must say that they are along the right line. In the QUARTERLY of January, 1900, we made a plea for representation of the profession on the basis of population, not absolute, it is true, but partial. It is a matter of satisfaction to us to find that not only the Ontario Medical Council, but Dr. Roddick as well, have come round to our views. The incorporation of this principle in the Bill will make its passage much more easy because it makes it much more fair and equitable.

Another amendment which it was agreed to make to the Bill is to have those Universities that have Medical Faculties actually engaged in teaching represented on the proposed Dominion Council. This is another concession which we advocated in the QUARTERLY of January, 1900. We must confess that when we find both the Ontario Medical Council and Dr. Roddick coming round to our views on these two important points we have a feeling of what might be called pardonable pride. It is always pleasing to have one's views endorsed, and especially by those who were previously in opposition. Our success in these two particulars encourages us to persist in our advocacy of other changes which we have already pointed out should be made in the Bill. The regulation regarding examinations must be altered before Dr. Roddick can succeed in passing his Bill. At present partiality is shown to Montreal and Toronto, and injustice to Winnipeg, London, Kingston and Halifax. Now, in our opinion, a bill which gives favours to one or two Universities and at the same time is unjust to all others can not and will not be endorsed by Parliament. Even if the Bill should receive the sanction of Parliament with this objectionable feature retained, the Dominion Council would always have to contend against the Universities which the Bill discriminates against as well as the opposition of all the graduates of these Universities scattered throughout the length and breadth of the Dominion. Thus the success of the proposed Dominion Council would be in jeopardy and its continued existence a matter of doubt. In the January (1900) issue of the QUARTERLY we gave expression to our views as follows:—"All written examinations should be held simultaneously, and all practical and oral examinations in succession at those centres at which there are Medical Schools. The papers could be sent to those centres under seal, and a presiding examiner could be appointed at each place. For the practical and clinical examinations examiners would have to go to the various centres." In the *Canadian Practitioner and Review* for March, 1900, the editor, referring to the above proposal, says: "The proposal to hold the annual examination at Montreal and Toronto would be strongly opposed, and we think the plan proposed by our contemporary would be much more satisfactory." With this endorsation we are determined to persist in our advo-

cacy of our position, and to oppose the passage of the Bill until justice is done to Winnipeg, London, Kingston and Halifax."

Later on we will take occasion to refer to some other defects in the Bill before it can be allowed to become law.

QUEEN'S NEW MEDICAL BUILDING.

FOR some time past the attendance at the Medical Department of Queen's has been increasing so greatly that the Faculty felt the absolute necessity of having more accomodation. During the past session the over-crowding was so great that the work of enlarging the building could be delayed no longer. Plans were prepared, the contracts were awarded and the work of construction commenced as soon as the classes closed. The enlarged and improved buildings will be ready for occupation at the opening of College next session. The new buildings will have a capacity one third greater than the old building. On the ground floor there will be a physiological and bacteriological laboratory, a private room for research in physiology and histology, a lecture room and a students' cloak room. The first floor will contain a pathological and bacteriological laboratory, a room for the preparation of the work in these departments, a lecture room, a library, a museum, a students' reading room and a private retiring room for the professors. On the third floor there will be the dissecting room, a room for the demonstrators in which to conduct their grind classes, a private room for the professors and two lecture rooms. The building will be furnished with all modern improvements. We congratulate Queen's Medical Faculty upon their success in the past which has rendered these enlargements and improvements necessary and would express the hope that their prosperity in the future will be commensurate with their enterprise.

The new building will, we understand, be formally opened on or about Oct. 2nd next. A public meeting will be held in the new dissecting room, which is capable of holding 400 people, and addresses will be given by graduates from New York, Ottawa, Toronto and other centres. We trust that many graduates will come in and take part in the ceremony.

THE EYE IN GENERAL DISEASES.*

*Read before the Orange Mountain Medical Society, Orange, N.J., May 31, 1901.

MR. PRESIDENT AND GENTLEMEN,—I fear it is not an original observation, but it is none the less true, that one of the most marked characteristics of the progress of medical science and practice during the century just ended has been the development of what we are accustomed to call the "Specialties." And in that development none of the branches of special medicine, it seems to me, have shared more fully or completely than Ophthalmology. It has been well said that the specialist must—to be a good specialist—have a knowledge of general medicine and surgery; otherwise he is almost certain to become narrow in his point of view, and to imagine that in his particular study lies the *fons et origo* of practically all the ills that afflict us. This mental astigmatism—if you will permit me to so term it—is not unknown, I regret to say, in the ranks of the Ophthalmologists, and there are those who would have you believe that a dislocation of the head of the femur is due to an unoperated upon exophoria, or that a pair of sphero-cylindrical lenses will banish for all time almost any of the discomforts to which we have become accustomed by long experience. Nevertheless, Ophthalmology is intimately interwoven with general medicine, and it is my desire this evening to bring to your notice some of the more commonly recognized ocular symptoms occurring in those diseases which as practitioners of medicine you are constantly called upon to treat. This I will be compelled to do with considerable brevity, as the subject is a large one; and it would consume altogether too much of your time to dilate, even in a small way, upon what might otherwise be matter of very great interest to both of us.

Let us begin, then, by considering the more important of the ocular symptoms occurring in *diseases of the Nervous System*, probably the most instructive of all. These may be divided into (1) affections of the eyes occurring in *diseases of the Brain*, of which tumors, abscesses. hemorrhages and meningeal inflammations are the most important. Intense optic neuritis—the

"Choked disc" of the Germans—occurs in about two-thirds of all cases of tumor of the brain. It is not an early symptom, is most common in cerebellar tumors, and is usually binocular. If unilateral, the tumor is as a rule in the anterior lobe upon the same side as the neuritis. Vision is usually affected, but it may not be.

In cerebral abscess there is often severe optic neuritis, more marked upon the side of the lesion, impairment of vision in any degree, contraction of the visual field, disturbances of color vision, and conjugate deviation of the head and eyes towards the side affected. In rapidly advancing suppurations, however, the diagnostic value of the eye symptoms is apt to be impaired by the appearance of remote symptoms from the opposite hemisphere.

In cerebral hemorrhages there is usually a loss of one half of the visual fields—either fleeting or permanent. Conjugate deviation of the head and eyes towards the side affected is common, and there may be optic neuritis, especially if the hemorrhage has been into the sheath of the optic nerve.

In meningitis the eyes take part in the general hyperæsthesia and there is great sensitiveness to light. Meningitis of the convexity may cause cortical blindness without ophthalmoscopic signs, while basilar meningitis carries in its train paralysis of the ocular muscles, optic neuritis with great congestion of the nerve head, nystagmus (frequently), anæsthesia of the cornea with its attendant neuro-paralytic keratitis, and atrophy of the optic nerve. Epidemic cerebro-spinal meningitis is frequently characterized by œdema of the conjunctiva, infiltrations into the cornea, and destructive keratitis.

In disseminated sclerosis, the ocular symptoms are of considerable diagnostic value. We find, for instance, (in about one-half of all cases) true nystagmus—a very rare symptom in other forms of general nervous disease. Defects in the central portion of the fields of vision—scotomata—often only for colors, are very common in disseminated sclerosis and very uncommon in hysteria, between which and disseminated sclerosis the differential diagnosis may have to be made. Moreover, paralyses of the ocular muscles are very common in disseminated sclerosis—very uncommon in hysteria, in which, however, we often see spasms of those muscles.

A peculiar and singularly interesting symptom is not infrequently noticed in general paralysis of the insane, viz. :—a reflex rigidity of the pupil, described by Argyll-Robertson, and called by his name. The symptom consists in the gradual loss of the pupillary reaction to the light stimulus, and subsequently also to that of convergence and accommodation. Paralyses of the ocular muscles are also very common in Paretics.

In insanity, there are, as is well recognized, all kinds of visual hallucinations ; in neurasthenia, we frequently find concentric contraction of the visual fields ; and in epilepsy there are various color anomalies, which may constitute the "Aura." Of the connection between errors of refraction and epilepsy I shall presently speak.

Of the accidents which may occur to the brain, none is so productive of mischief in the eyes as fracture of the base of the skull, which so frequently involves the optic foramen. From pressure of bone or blood we often get complete blindness. Atrophy of the optic nerve is also one of the results.

(2) Affections of the eyes occurring in diseases of the *Spinal Cord.* Of these diseases tabes is undoubtedly the most important, and in this affection the eye symptoms are of great value. Primary gray atrophy of the optic nerve and the Argyll-Robertson pupil are the chief symptoms, although many other eye complications have been observed and described. Atrophy of the optic nerve may be the first sign of the disease ; it is almost always pre-ataxic, and it is a curious fact that its progress during the early stages of the disease has often a beneficial effect upon the pathological process in the cord, it being not uncommon to find blind tabetic patients who never become ataxic. The Argyll-Robertson pupil may be found in general paralysis, as has already been noted, but it never occurs in peripheral neuritis—a disease sometimes mistaken for tabes.

In various forms of myelitis, hereditary ataxia (Friederich's disease), ascending paralysis, caries of the spine and tumors of the cord there are ocular symptoms, but they are not sufficiently typical to be of diagnostic value.

Let us now consider the ocular lesions and symptoms in diseases of the urinary and reproductive systems, and first of all

in albumenuria. You scarcely need to be told that the ophthalmologist is frequently called upon to make the diagnosis in this disease, and the eye changes are found not only in chronic interstitial nephritis (rarely in the acute forms of the disease), but also in the albumenuria of pregnant women and that following scarlet fever. We find in these cases inflammation of the retina, either alone or associated with inflammation of the optic nerve head, hemorrhages into the retina, and exudations into its substance. Most remarkable recoveries of vision are seen in the albumenuric retinitis of pregnancy, a case of my own having recently advanced to normal acuteness of vision after complete blindness and an ophthalmoscopic picture which revealed not one spot of normal retina as far as I was able to discover. Before dismissing the subject of albumenuria I might mention that in victims of uræmic poisoning sudden blindness—the so-called "uræmic amaurosis"—occurring usually after a convulsion and lasting from twelve hours to two or three days, is one of the acute symptoms.

In diabetes there are many ocular complications. A diminution in the range of accommodation, together with the onset of myopia in individuals of advanced age, are frequently noticed; partial or complete paralysis of the ocular muscles may occur; but exudations and hemorrhages into the retina and cataract are the most characteristic changes found, the retinitis being one of the late manifestations.

Here, as well as elsewhere, might be mentioned gonorrhœal ophthalmia, that most destructive of all conjunctival diseases, as well as its younger associate, blenorrhœa neonatorum, happily growing more rare year by year; hemorrhages into the vitreous humor in disorders of menstruation; puerperal retinal hemorrhages; syncope in difficult labors with the usual eye symptoms; and embolic panophthalmitis of the puerperal period with destruction of the eye.

Diseases of the Respiratory Organs have no characteristic eye symptoms, and *Diseases of the Digestive Organs* few, if we except the consequences of poisoning by various substances—to be referred to shortly—and reflex asthenopia from affections of the teeth or errors of refraction.

Diseases of the Skin affect the eyes generally by continuity. Erysipelatous inflammation may be the diagnosis in cases of acute inflammation of the lachrymal sac; but erysipelas may of course attack the lids, and it is worth noting that an intercurrent attack of erysipelas has a most beneficial effect upon the course of such diseases as trachoma, iritis or choroiditis already existing. Herpes and eczema of the lids, xanthelasma, and, finally, phthiriasis ciliorum, complete pretty well the list of skin diseases involving the eye or its appendages.

Diseases of the Circulatory System produce effects in the eye which might be expected. Small hemorrhages occur in the retina from changes in the blood or vessel walls. Severe loss of blood may cause partial or complete blindness, rarely entirely restored. Embolism of the retinal arteries is important on account of the fact that they are terminal vessels; and pulsating exophthalmos from aneurism of the internal carotid may be mentioned. In addition to these we sometimes have retinal hemorrhages and oedema of the retina in leukæmia and pernicious anæmia.

The effects of *various poisonous substances* upon the eyes are important, and I will refer to them briefly.

Alcohol, in acute poisoning, is said to produce diplopia—occasionally polyopia. In the chronic form, there are visual hallucinations, as in Delirium Tremens, and the well known toxic amblyopia with central defects in the fields of vision due to an interstitial inflammation of the axial bundles of the optic nerve. This form of amblyopia is, however, more common when the alcoholic is at the same time a smoker, and it does occur from tobacco alone; indeed, it is sometimes called "tobacco amblyopia" and is most commonly found in those who smoke constantly, particularly strong tobacco in a pipe.

Atropine produces the dilatation of pupils, 'though not necessarily maximal in poisonous doses; eczema of the lids in those predisposed, acute glaucoma and visual hallucinations. In connection with the fact just mentioned, that Atropine sometimes precipitates an attack of acute glaucoma, I would like to point out the danger involved in the somewhat indiscriminate

use of this drug in all attacks of superficial inflammation of the eyes. There are not a few physicians who, the minute they see a case of sore eyes attended with congestion of the conjunctiva, drop a few drops of a solution of Atropine into the conjunctival sac—almost as a routine procedure. This is a most dangerous practice and one which cannot be too strongly condemned ; and I would lay down a rule to be followed by all who are not Ophthalmologists : viz. :—never to put Atropine into any eye unless absolutely certain that it is suffering from Iritis—certainly never to put Atropine into the eye of anyone over thirty-five years of age ; and better still, not to use it at all, if an Ophthalmologist is available.

Chloral causes marked contraction of the pupils in poisonous doses ; *Cocaine* has been held responsible for attacks of glaucoma in cases where it has been applied to the nasal mucous membrane for a length of time ; *Chlorate of Potash* has brought on attacks of uræmic amaurosis through hemorrhagic nephritis ; *Ergotine* is said to cause cataract from interfering with the nutrition of the lens ; *Iodine and Iodoide of Potassium* cause conjunctivitis and increase the lachrymal secretion ; *Iodoform* is said by Hutchison to cause toxic amblyopia ; *Morphine* contracts the pupils, though in habitués it has been known to do the reverse ; chronic *Lead* poisoning produces optic neuritis, sometimes hemorrhages, hemianopia, visual aphasia and hallucinations from the peri-arteritic changes, and nephritis with its characteristic accompaniments. *Phosphorous and Carbonic Oxide* cause retinal hemorrhages and fatty degeneration of the vessels ; *Quinine*, arterial spasm, accounting for the blindness which sometimes accompanies the administration of this drug, also exudations into the retina, and atrophy of the optic nerve. *Salicylate of Sodium* has been known to cause similar symptoms ; and *Sulphide of Carbon* produces various paralyses, hemianopia, contraction of the visual fields, and even, in chronic cases, toxic amblyopia.

Passing on to *Fevers, Microbic Diseases and Constitutional Affections*, the albumenuric retinitis of scarlet fever is well known ; in *Measles* we find conjunctivitis ; in *Typhoid*, optic neuritis, inflammation of the iris and the whole uveal tract, and paralysis of accommodation with dilated pupil from the general weakness of

convalescence; in *Typhus*, similar symptoms; in *Cholera*, the loss of the serum of the blood causes the lids to shrink and they cannot be closed fully. Thus the exposed cornea often becomes denuded of epithelium. Corté says that as long as the pupillary reaction to light remains in Cholera the prognosis is good, and vice-versa. In *Diphtheria*, we have a peculiar form of conjunctivitis characterized by the formation of a membrane, partial paralysis of the accommodotion, commencing three weeks after the disease has run its course and lasting from four to eight weeks before disappearing spontaneously, paralysis of the other muscles occasionally. In this paralysis of the ciliary muscle, strange to say, the pupil is unaffected, and it is accordingly argued that there must be a ptomaine which has a peculiar predilection for that branch of the 3rd nerve which governs the action of the ciliary muscle, and that the paralysis is peripheral and not nuclear. In *Influenza*, there are ocular neuralgias, outbreaks of acute glaucoma, optic neuritis, and atrophy. I have now under my care a case of monocular optic neuritis undoubtedly due to Influenza.

In *Whooping Cough* have been noticed blindness of both eyes without any ophthalmoscopic appearances, and retinal and subconjunctival hemorrhages; in *Mumps* we occasionally find enlargement of the lachrymal glands; in *Malaria* there are ocular symptoms of great variety, but supra-orbital neuralgia is most noted, with occasional paralyses of the muscles, and attacks of blindness without intra-ocular changes. *Septicaemia* has had credited to it retinal hemorrhages and degenerative spots in the retina; in *Pyaemia* we may have orbital abscess or focal development in the globe, leading to destruction of the eye. *Rheumatism* is well known as a causative agent in the minority of the cases of iritis, in most of the inflammations of the sclera and Tenon's capsule, and is half suspected of having something to do with the causation of acute glaucoma.

Sufferers from *Ex-Ophthalmic Goitre* not infrequently seek advice first from the ophthalmologist on account of the protrusion of the eyeballs, which deformity goes on to complete luxation of the globe. Examination of the eyes in many cases reveals the spasm of Mueller's muscle, which goes by the name of Graefe's symptom, and the decreased sensibility of the cornea,

which is accountable for the infrequent winking (Stellwag's symptom).

Tuberculosis may involve the eyes through the meninges, producing the ocular symptoms of meningitis, or it may attack them locally, causing tubercular ulceration of the conjunctiva, tubercles of the iris, which, when primary, have been successfully removed by iridectomy with preservation of the sight and no return of the lesion; and tubercles of the choroid, which form a very late incident in the life of the consumptive.

In Syphilis, as might be expected, there are almost endless ocular lesions. In the acquired disease we may have chancre of the lid or conjunctiva, usually on the caruncle of the right eye, from the fact that most people are right-handed and most people have the habit of rubbing the eyes. Three-quarters of all cases of iritis are syphilitic, and either by inflammations or specific new formations every structure in the eye may be attacked in this protean disease. Cyclitis, choroiditis, specific arteritis with destruction of the small vessels of the retina, gummata, gummatous periostitis of the orbit and the various paralyses of muscles due to cerebral syphilis, are some of the forms which the syphilitic attack may take.

In the inherited disease we have an interstitial keratitis which is entirely typical, and every possible kind of congenital intra-ocular lesion.

In conclusion, Mr. President and Gentlemen, let us pay some short consideration to the part which errors of refraction play in the development or aggravation of disorders of the general health. It is held by many that errors of refraction and abnormalities of the muscular balance of the eyes form an important link in the causation of various distressing ailments: headaches of all kinds, certain disorders of digestion, vertiginous attacks, neurasthenia, chorea and epilepsy; and undoubtedly with good cause. There are few ophthalmologists who cannot refer to many cases of severe headache cured by the wearing of proper glasses, and to at least a few cases of each of the other troubles which I have mentioned to you either relieved or cured in the same manner. With regard to epilepsy, the most important of these affections, I will only refer to the work of a conservative

investigator who has examined a large number of epileptic patients and published his results. He found that the correction of errors of refraction will, in a very considerable proportion of cases, cure the disease, especially in school children and young adults who are making large demands upon their accommodative apparatus; and that it will relieve the symptoms in a fair percentage more. If the balance of the ocular muscles is at fault, after the refractive abnormality has been corrected, then the appropriate treatment (optical or operative) must be adopted; and in this way, not only visual comfort but also a degree of mental and bodily comfort until then unknown will be brought to many who have hitherto suffered helplessly. And this is one of the shining certainties of modern Ophthalmology.

JOHN R. SHANNON, New York.

ECTOPIC GESTATION.

Read at the Annual Meeting of the Ontario Medical Association, Toronto, June, 1901.

A STUDY of medical literature, past and present, goes to show that correct ideas of the causation and earlier changes consequent upon ectopic gestation, as well as a real working knowledge of its management, date back less than twenty-five years. Previous to that time deaths were reported from so-called accidental hemorrhage into the peritoneum, and from intraperitoneal and extraperitoneal hæmatoceles. Many cases were reported of fœtus found in the abdominal cavity, and of lithopedions discovered many years after the pregnancy from which they dated their origin. A few years ago the profession at large could not but regard as extraordinary the diagnostic acumen of the men who could make the diagnosis of tubal pregnancy on the occurrence of rupture. At the present time, with the increase of literature on the subject, and with our better knowledge of its pathology and symptomatology, every physician is expected to make a correct diagnosis on such occurrence, and, in a fairly large proportion of cases, to make a diagnosis before the occurrence of rupture.

Classification. Every pregnancy is the result of the impregnation of an ovum of the female by the spermatozoon of the male. The normal place for the development of the impregnated ovum is the cavity of the uterus. The channel through which the ovum must pass from the ovary, in order to gain the uterine cavity, is the Fallopian tube. Just where impregnation normally takes place is still an unsettled question. By some the situation is claimed for the uterus alone, by others for the ovary and sometimes for the tube.[2] On the one hand there is not much evidence for the belief that the seat of normal impregnation is limited to the cavity of the uterus; on the other hand facts are known concerning the invasion of the tubes by spermatozoa which unmistakeably point to the conclusion that normal fructification of the ovum may occur at any stage of its passage from the ovary to the uterus. It may thus be stated that an abnormal arrest, whether mechanical or special, of a fructified ovum in its progress toward the uterus is the determining factor of an extra-uterine, ectopic, or misplaced pregnancy. Theoretically this arrest may occur (1), in the ovary, (2), in the abdominal cavity between the ovary and tube, (3), within the tube, and (4), between the tube and the uterus. The first seems to be theoretical only. Many writers deny the possibility of the ovum becoming impregnated within the Graafian follicle and continuing to grow there, while others, quoting from various observers acknowledging the existence of such fecundation, freely admit that there are but few indubitable cases in record. Howard Kelly[3] describes it as "one of the greatest gynæcological rarities." Taylor[1] says "it is possible but absolute proof of such a pregnancy seems to be incomplete." For a practical study of the subject this variety may be dismissed. The second—arrest within the abdominal cavity between the ovary and tube—is probably almost immediately fatal to the unprotected ovum, owing to the digestive power of the peritoneum, and consequently may be eliminated from discussion. The fourth point of arrest—between the tube and uterus—may be quite correctly regarded as arrest in the uterine portion of the Fallopian tube. To all intents and purposes then we have, at the outset, but one kind only—arrest within the tube, or *tubal pregnancy*, and it is to this variety I shall mainly direct your attention. All other varieties are tub

later developments of tubal pregnancy owing to a secondary invasion from the Fallopian tube.

Etiology.—A careful and thorough consideration of the causation of ectopic gestation is indispensable for a proper explanation of many of the clinical symptoms observed, as well as for an accurate diagnosis at any early date.

Among the classical causes put forth in the various text-books, both large as well as small, one finds something like the following:—Diseases of the mucous membrane depriving it of its cilia; other inflammatory changes in the mucous membrane, contractions in the calibre of the tube, the result of chronic salpingitis or perisalpingitis; peritoneal adhesions constricting or distorting the tube; intratubal polypi; atresia of one tube with external migration of the fertilized ovum, or of the spermatozoa of the opposite side; all pointing, as they do, to a previous history of pelvic disease or decided deformity in some form. With these the etiology generally stops, a fact which is at least misleading to the clinical observer. With such etiological factors alone before his mind he will pass by as impossible an otherwise strongly suspected case.

A study of the development and structure of the tube and the means afforded by it for the transit of the ovum will serve to explain the occurrence of ectopic gestation in a woman with no history of pelvic disease, or of long sterility, or, in other words, the occurrence of ectopic gestation in a perfectly healthy woman with normal menstrual functions. The delicate plications of the mucous membrane of the tube, covered with innumerable cilia waving always toward the uterus, tend to sweep the ovum onward and outward, while the peristaltic action of the muscular fibres of the tube aids in the work done by the cilia. If from any want of activity on the part of the cilia, or if the action of the muscular coat be impaired, or both, owing to some nerve influence, it cannot fail to have its bearing on the progress of the ovum.

Slight congenital anomalies of the tube, the result of anomalies in early embryonic development of the Mullerian ducts, may produce an imperceptible stenosis in the calibre of the tube, yet sufficient to impede the progress of the ovum on its way to the uterus. Unusual hyperæmia, or marked menstrual changes in the tubal mucous membrane, renders possible an arrest of the

ovum within its folds. One can scarcely doubt that menstrual changes in the uterine mucosa prepares it for the reception and implantation of the impregnated ovum, and when the tubal mucous membrane undergoes unusual menstrual changes it not only diminishes the calibre of the tubal bore, but it becomes a soil in which an impregnated ovum may easily implant itself.'

A further study of the anatomy of the tube teaches us that it is not a straight tube, but a convoluted one, bound down at every bend by fibrous bands beneath the serous covering, and that its mucous membrane is arranged in plications, each forming elevations with recesses or depressions between them. From this it may readily be inferred that the journey of the ovum from the ovary to the uterus is naturally a slow one, being retarded by the convolutions of the tube and the irregularities which the plications afford. Next we must remember that the growth of an ovum once fructified is rapid in the extreme, reaching in size, at the end of the second week, from three to six millimetres in diameter. Now if there be any diminution in the propulsive power of the cilia, or failure in the peristaltic action of the muscular coat of the tube, or any diminution in the calibre of its bore the result of menstrual changes in the mucous membrane, or of congenital anomalies, it necessarily follows that the ovum will be further impeded in its journey to the uterus. If to these impediments we add the further difficulties which a fructified ovum, rapidly increasing in size, presents to a canal already crippled in its efforts to perform its function, it will readily be seen that a point within the tube may be reached when the fructified ovum can no longer be propelled on its journey, arrest must take place, and an ectopic gestation initiated at that point.

Once the ovum has become arrested within the tube it is there surrounded by a zone of mucous membrane within which the chorionic villi develop, and to which the ovum becomes attached, but it is exceedingly doubtful whether there be any true decidual tissue or not. A point which is worthy of careful consideration is the rapidity with which the local blood vessels become enlarged and dilated, vessels which ordinarily are quite small become doubled, and even trebled, at a remarkably early period of tubal growth. This is the source of the greatest danger, and accounts for the violent hemorrhage and rapid death after early rupture.

The formation of a decidua within the uterine cavity, while the early changes are taking place in the tube, is also a matter of considerable importance from a diagnostic standpoint. Death of the ovum, in whatever way brought about, is often associated with the shedding of the decidua which may be cast off in its entirety, or more usually in small pieces or as a shreddy like material.

With the growth of the ovum the tube distends, but from its structure and anatomical relations it is very evident that it cannot long keep pace with the growth within it, and consequently most tubal pregnancies end in abortion through rupture of the tube. Many, however, become abortive by hemorrhage from their own blood vessels and the formation of a tubal mole within the tube. In only a very small proportion of cases does the fœtus, after rupture of the tube, go on to full term within the maternal organism. A pregnant tube may rupture very early—within the first four or five weeks—or it may continue to distend for several weeks longer, three months being about the outside limit.

Very early rupture has only recently received the attention the great dangers attending it deserve. It not infrequently occurs in those cases in which ectopic gestation has not even been suspected, or in which, from the paucity of the symptoms, the physician has had no material to guide him in that line of thought. There has been no history of ill health or of pelvic disease; no early signs of pregnancy, unless possibly some ill defined irregularities at the last menstruation.

I take the liberty of briefly quoting a case which came under my observation some three years or more ago, as a means of picturing the clinical history of such cases.

Mrs. B. aged about twenty, a fine healthy looking girl, without history of previous illness of any kind, and married only a few months, was on her return home from a short trip on the steamer seized with rather sharp pains in the abdomen, similar to an attack of intestinal colic or acute indigestion. Nausea and vomiting soon followed, the vomited matter being made up of a full dinner which had been partaken of an hour or two before. The general history of the patient elicited nothing special, she had menstruated twelve days before quite naturally as far as she knew. She was given a hypodermic of morphia by her regular

physician who was summoned and advised to remain quietly in bed until his return a little later. Not long after the husband visited the family physician and announced that his wife was better, was resting comfortably and that there was no necessity for his return. Early in the evening she complained of feeling weak and faint, and, on the arrival of a friend, the physician was again sent for, who found an anxious if not an alarming condition of affairs. I saw her at once in consultation. The patient was faint and collapsed, the pulse small, weak and thready, the temperature subnormal, the extremities cold, and a cold gray appearance had spread over the countenance. A careful enquiry elicited that the pain had become localized in the left iliac region. A vaginal examination revealed nothing which would aid in making a diagnosis—*no tumor could be felt.* After a hurried preparation she was taken to the General Hospital, where we at once opened the abdomen. The cavity was full of blood, I did not think it possible that the abdomen could hold so much, or that a person could lose so much blood and yet be alive. The left tube was first examined and a rent from which blood was still oozing was discovered in the isthmial portion about three-quarters of an inch from the uterine cornu. The ovum—about the size of a bean—was found on the anterior surface of the broad ligament between that structure and the bladder. After removal of the tube the abdomen was filled with salt solution and closed. Intravenous saline solutions were freely used, but she was too exsanguinated to react and died some four or five hours later. After operation an examination of the tube was made. There was no swelling except at the seat of the pregnancy. The part enclosing the pregnancy was thinner than usual, but without any evidence of compensating growth. The tube seemed to be fully developed, the opening through which the pregnancy escaped had the appearance as if a small pistol bullet had pierced it from within outward.

Instead of early rupture there is another cause, and if the most recent microscopical investigations into the early pathology of tubal pregnancy be correct, is the most frequent primary cause of the interruption of such forms of pregnancy, viz. the formation of what has been termed "tubal mole."

The ovum during its first few weeks of growth, depending

as it does for life upon very delicate chorionic villi lightly attached, is in constant danger. Hemorrhage from the tube [illegible] or gestation sac, into the intervillous spaces, even though very slight, is apt to detach and crush a number of villi, and in course of time will generally cause the death of the embryo. In more severe hemorrhages the chorion is more or less completely detached from the decidua, and at once death of the embryo takes place, forming in the tube what is known in uterine pregnancy as "blighted ovum," and may be here termed "tubal mole." The blighted ovum now acts as a continuous irritant to the tube, producing hyperæmia, followed by increasing vascularity and thickening of its walls. Repeated hemorrhages occur, some of which, if the ostium abdominale be pervious, passes into the abdominal cavity, other portions of the blood form lamellated clots within the tube, which in their turn increase its size and weight. The overburdened tube now falls over backward and reaches the floor of the pelvis on its own side, dragging with it the ovary and mesosalpinx. With this displacement of the tube there is consequent derangement of the blood return. Torsion of the blood vessels increases the difficulty, and there is, as a result, increased bleeding, often very abundant, into the tube and pelvic cavity. With repeated hemorrhages there is soon formed in the pelvis a well defined tumor of varying size, composed of tube ovary and blood clot, pushing the uterus to the opposite side, and an intraperitoneal hematocele is now formed. This by repeated hemorrhages may go on and increase to a large size, filling up the pouch of Douglas and probably rising up into the iliac fossa, or filling the whole of the lower part of the abdominal cavity.

A considerable number of ectopic gestations have a longer tubal existence than that described. The period to which the pregnancy may advance without rupture will depend much upon the direction in which the growth is greatest. The tube, subject to slowly increasing pressure from within, becomes stretched and thin, and, as it enlarges, may open up the layers of the mesosalpinx, by which space is gained in which pregnancy may develop further without interruption. But the time comes, at the furthermost about the third month, when the space is altogether insufficient for the growing tumor. In this case either

the layers forming the mesosalpinx must be still further displaced and the pregnancy burrow downward into the cellular tissue beneath it, or the upper layer will become thinner and thinner until rupture takes place, with partial or complete extrusion of the pregnancy into the abdomen. The amount of hemorrhage in this form of rupture is variable, depending much upon the placental site. Should the placental site be distant from the rupture it may be slight, as the portion of the tube in which rupture has taken place may have been almost entirely deprived of its blood supply by its conversion into a thin membrane. If the placental site be torn through undoubtedly the bleeding will be severe, and at times fatal. In some cases as the blood pressure falls hemorrhage ceases, and an intraperitoneal hematocele of varying proportions, according to the amount of blood lost, is formed. The patient will be, however, further subject to repeated hemorrhages from increasing detachment and protusion of the placenta, and in this way the hematocele will increase in size, assuming at times large proportions.

We might now with some degree of profit draw attention to the different formation of the two varieties of intraperitoneal hematocele which when fully formed would appear to be identical. In the first variety—that which is formed as the outcome of a tubal mole—it will be noticed that the early formation was the result of a constant dribbling from the tube, partly consequent upon the irritation produced in the tube by the presence of the mole and partly by the distorted blood vessels, varied by an occasional free bleeding at irregular intervals. In the second the hemorrhage is sudden, relatively copious and arises directly from the tear in the tube or from separation of the placenta.[1]

Should the gestation sac in its growth separate the layers of the mesosalpinx, especially if the site be near the centre of the tube, it will ultimately give way in that direction and the fœtus is extruded into the connective tissue space between the layers of the broad ligament, forming what is usually termed "tubo-ligamentary" or "broad ligament pregnancy." With this rupture there is usually considerable hemorrhage, but it is limited in amount by the attachment of its dense and unyielding walls, and consequently cannot attain any very great size, or be fatal from

the amount of blood lost. In this way is formed an extra-peritoneal, or broad ligament, hematoma. This brings us to the consideration of the third form of hematocele, and the oft repeated statement that every hematocele is the outcome of an ectopic gestation, and that when no fœtus has been discovered in it, nor any remnants of a previous gestation, it is no evidence to the contrary. While an intra-peritoneal hematocele may be said to be almost always due to an ectopic gestation, the existence of a broad ligament pregnancy is not always to be considered as having existed when a hematoma is discovered in it. On the contrary, I believe that they are so formed in a minority of cases, and that the majority of them are owing to menstrual irregularities, arrest of menstruation, or to chronic pelvic inflammatory diseases.

The fourth subdivision in the classification of ectopic gestation, although it cannot be said to be extra-uterine, deserves some slight separate consideration. In tubo-uterine or interstitial pregnancy the impregnated ovum develops in the portion of the tube which lies within the uterine wall. It is recognized by all observers as being exceedingly rare. In a collection of 1324 cases[6] but 40 were said to have been interstitial. The cause of this form will in all cases be found to be owing to contraction of the ostium uterinum, either permanent or muscular, so that it refuses to admit the passage of the fertilized ovum. On account of the situation primary rupture may be delayed as far as the fourth month, or even longer. When rupture takes place it may be into the uterus and will then become, if we follow up the classification initiated, secondary intra-uterine pregnancy. This classification seems to be largely theoretical, as I am unable to find any positive demonstration of its ever having taken place. The only rupture that is known to have taken place is into the abdomen. Because of the thicker wall and the greater vascularity of the sac, intra-peritoneal rupture is usually more rapidly fatal in this variety than in the ordinary tubal pregnancy. Taylor says "hitherto this has always proved fatal in a very few hours." This form of pregnancy is apt to be confounded with pregnancy of the rudimentary horn. The diagnosis is said to be exceedingly difficult, if not impossible, previous to opening the abdominal cavity.

Symptoms. In early rupture—the most fatal form if we take frequency into consideration when comparing it with interstitial pregnancy—there may be no pelvic or abdominal signs of definite importance. Very rarely is there any evidence to be obtained from the condition of the breasts. Often the earliest and only symptom is sudden abdominal pain, confined for the most part to one or other iliac region, and associated with symptoms of shock and hemorrhage. While many cases are of this sudden and wholly unexpected type a large proportion of ectopic gestations have well defined symptoms, if carefully and diligently sought for. There are four links in the chain of symptoms which should receive the most earnest consideration, and which I think, if properly followed up, will aid in no small degree in arriving at an early diagnosis. They are:—

1. The pre-pregnant history.
2. The menstrual history.
3. Uterine hemorrhage and the nature of it.
4. Localized pain in the inguinal region.

1. *The pre-pregnant history.* In a large proportion of cases there is a history of several years having elapsed since the last pregnancy, or the patient has been married a number of years without conception. In a moderate proportion of such cases there accompanies this history one of pelvic disturbances, it may be simply of dysmenorrhœa in some form; or it may be of a more serious or constant type, pointing to tubal or ovarian inflammatory disease. But whether one or both of these be present, a point that may often be elicited is that for a short time at least there has been a lull in these symptoms, the patient expressing herself as feeling better for some time past than she had perhaps for years before. This point is well to remember, for it will aid materially in making a differential diagnosis, in that there is a history of illness rather than of improvement in health immediately preceding the formation of a tumor with which the condition might be confounded.

2. *The menstrual history.* This link in the history possesses two distinct types. (*a*) The patient gives an unquestionable history of amenorrhœa, she declaring that she has exceeded her normal time by one or two months. Such cases facilitate diagnosis, in that our suspicions are at once aroused to the strong

possibility of pregnancy, and accordingly we are put on our guard. (*b*) The second type is that in which the patient gives a history of menstrual regularity, "she has never missed a term." Such a history is naturally misleading, and, unlike the other, throws us off our guard. If, however, we inquire very closely into the menstrual history of the last one or two periods, we will find a change in their character. Previous to that there was a certain type for her which she always looked upon as being natural and which she always expected. Now she remembers on thinking carefully that her "monthlies" had not been the same. The first period had been delayed somewhat, it had not come on as it should, or it had been rather scanty. Perhaps the second one had come earlier than expected, and perhaps more profuse or unusually protracted. In fact she may say that she has not yet *quite got over her last monthly*, and that she is unwell at the present.

3. *Uterine hemorrhage.* Whether there be a history of amenorrhœa or irregularity in a suspected case a period arrives when uterine hemorrhage is a symptom. In the case where there is a history of amenorrhœa it will likely be considered by the individual as a return of her delayed monthlies, or it may be regarded as an early abortion. In the cases without such history the menstrual flow, instead of stopping as it should, continues for an indefinite period. An examination of the character of the hemorrhage is of the greatest importance. The blood will be found to be almost invariably dark in colour, moderate in amount, steady in the rate of flow and decidedly thickish.[8] Gushes of bright blood occasionally occur, but they are exceptional and small in amount. The hemorrhage as a rule arises from the partial or complete separation of the decidua, consequently shreds or portions of decidual membrane, rarely the membrane in its entirety, may be found in the vaginal discharges. At this period there arises the possibility that it is an early abortion. I would particularly draw attention to the characteristics of the uterine hemorrhage. *It is dark in colour, moderate in amount, with occasional small gushes of bright blood.* These facts will aid largely in diagnosing it from the reappearance of a delayed, or from a prolonged menstruation, in that there is rarely, if ever, gushes of bright red blood; and second,

from an early abortion, as the blood at first is bright, often very profuse and coming away in large clots. Later, in cases of incomplete abortion, the color of the blood is dark and moderate in amount, but there is usually an odor of decomposition about it. The consistence of the discharge is an important point. *It is thickish*, in fact it has a mucous tenacity, which is not the case with menstrual blood, or in incomplete abortion. There is still another point to be gained from an investigation of the discharges, viz., *the odor*. There is accompanying abortion an odor which can always be recognized in its highest intensity in labor at full term, but which is not recognized in the uterine discharges of an ectopic gestation. In ectopic gestation the odor does not differ from that of the ordinary vaginal secretion.

In making a physical examination I shall pass by those early symptoms of normal pregnancy, and which may be present in ectopic gestation, such as changes in the breast and the color of the vaginal mucous membrane. Sometimes they are scarcely recognizable, sometimes absent altogether. Much stress is often laid upon enlargement of the uterus "bearing," as some writers say "a strong resemblance to that of the subinvoluted uterus."[1] The symptom is often misleading. Not infrequently the uterus is scarcely perceptibly enlarged even when measured by the uterine sound. In what is known as the Jessop case[1] the uterus at a full term abdominal pregnancy is described as feeling somewhat enlarged, and on measurement by Simpson's sound its cavity was found to be two and a half inches in length. The uterus will, however, always be found softer and more rounded off than an unimpregnated one. The condition of the cervix is also an important means of diagnosis. The os has a velvety feel, the cervix is softer than normal, and when there is uterine hemorrhage the cervical canal somewhat expanded, thus differing from a normal menstruation. I leave out intentionally those forms of dysmenorrhœa which are described as mechanical and membraneous, in that on careful enquiry there will be in such cases a history of one or other form of dysmenorrhœa on many previous occasions. Again, while it is noticed that the cervical canal is somewhat dilated and the cervix softened, the extent of dilation is never very marked, nor is the angle between the cervix and body of the uterus obliterated. This affords another point in differentiating ectopic gestation from early abortion.

The presence of a tumor in one or other fornix of the vaginal vault, and lying by the side of the uterus, but separate from it, is another important symptom. In the earlier stages, before the growth has assumed any size, and particularly when the gestation sac is in the isthmial portion of the tube, such will not be readily felt. If anything is discovered bimanually it will be in the form of a small spindle-shaped enlargement in the tube. Later, and especially when the gestation is ampullar, or the pregnancy has terminated in the formation of a tubal mole, this mass will topple over, after the manner already described, and will be found in the vaginal vault, either as a rounded or elongated tumor according to whether the dilated ampullar end of the tube lies vertical or horizontal on the pelvic floor. This point it is well to remember, as we are told to expect to find on examination an elongated tumor. Such will not be the case when the rounded out ampullar extremity stands, as it were, upon its head upon the pelvic floor, the isthmial end extending somewhat obliquely towards the uterus. The tumor will then have a rounded off feel resembling a cystic ovary.

Much too has been said about pulsating vessels being present in the vaginal vault. This sign when present must be a very valuable one, but it is not always present. It cannot be perceived before prolapse takes place, unless one has a touch sufficiently sensitive to distinguish the increased volume in the pulsation of one uterine artery when compared with that of the other. After prolapse it is not always possible to distinguish increased pulsation until adhesions have taken place and collateral circulation supplied.

I have selected the following case for illustration because of its very recent occurrence and characteristic history. Not the least interesting point about the case is the remarkable coincidence of its presentation at the office at the time when the section of the paper just under review was in preparation.

Mrs. B., aged 31, a strong, vigorous, healthy looking lady, came to my office on May the 6th last, complaining that she had been "unwell" for the last month or more. She had been married thirteen years, and was the mother of three children, the youngest three years and a half old. Her "monthlies" had always been regular and painless, except some six years ago, when

she had been irregular as to time for many months. She last menstruated January 30th, missed in February, and again at the end of March. On April 2nd she commenced to lose some blood, having, as she said, all the characteristics of her ordinary menstruation. This discharge continued in varying quantity throughout the whole of the month of April and up to the time of her appearance at the office. There never was at any time an immoderate flow. No clots had been passed, sometimes the blood seemed quite bright or brighter than usual. She was quite certain that nothing had come away that would attract attention as unusual. She had none of the early signs of pregnancy so characteristic of her former pregnancies, and accordingly felt quite certain she was not pregnant. About three weeks before she had rather a sharp pain in the left iliac region, which lasted an hour or two and was accompanied by vomiting. On examination I found in the vagina, and exuding from the cervical canal, a brownish-black discharge, small in quantity and with a thick mucus-like tenacity. There was no shreddy material that I could detect with the eye. The cervix was soft and the canal somewhat patulous. The uterus was not enlarged. A small, smooth, globular tumor, freely moveable, and about the size of a Tangarine orange, could be felt in the left vaginal fornix. The tumor was only perceptibly tender, and unusual vascularity in or about it could not be detected. On May 8th, two days following, she returned to the office saying she had been seized with a severe pain in the left iliac region late in the forenoon, and that she was still suffering from it. I ordered her at once to the hospital, to be kept there under strict observation. Late in the evening I again examined her and found the same condition as previously, but without symptoms which might cause anxiety as to a possible rupture. Early the following morning I operated. The pregnant tube—the left one and which I now present to you for examination—was readily lifted from the floor of the pelvis. There were no adhesions, and its removal was completed without event. Dr. W. T. Connell, pathologist, has kindly furnished the following report:—"The ovary is small, measuring 3cm. x 2.2 cm. x 1 cm., and contains a corpus luteum measuring 1.3 cm. x 1 cm. x 1 cm. The tube shows towards its abdominal ostium a globular enlargement 5 cm. x 4 cm. x 3 cm. The ostium of the

tube is open, but the fimbriæ are partly inverted. On cutting into the enlargement it is found to consist almost completely of clot, a few shreds of tissue are visible but cannot be recognized without microscopic investigation. The corpus luteum is that of pregnancy, and the enlargement of the tube a tubal mole."

Enticing as the subject may be, time will not permit a study of the further development, mode of growth or the physical symptoms of the various kinds of hematocele, the outcome of ectopic gestation, and which have been so well illustrated in a remarkable series of lectures on ectopic gestation by John W. Taylor, of Birmingham[1]; nor of the final termination of the secondary broad ligament and abdominal pregnancies, the outcome of primary tubal pregnancy. The very interesting discussions which have been in progress of late years as to the fate of the fœtus after rupture into the abdominal cavity, whether it lies naked there or is still enveloped in amniotic sac, is an inviting field to enter. It has generally been conceded that a continuation of the gestation is impossible on account of the digestive power of the peritoneum, and accordingly various explanations have been advanced for the occurrence of the seemingly impossible. Tait[10] advanced the theory that all such pregnancies which had survived intraperitoneal rupture were originally broad ligament pregnancies which had remained till the seventh or eighth month, and when rupture took place the vitality of the fœtus was maintained, "its tissues having arrived at a period of development by that time which enabled them to resist the efforts of digestion which, doubtless, would be directed towards them." Taylor, in his clear and comprehensive way, defines the more generally accepted theory on the subject. "When a fœtus which has already formed within the Fallopian tube escapes into the abdomen of the mother, enclosed in its own *unruptured membranes*, pregnancy becomes 'abdominal.' If the placenta retains its attachment to the tube and receives sufficient blood supply from the maternal blood vessels, the pregnancy may pursue an uninterrupted course to term and both child and placenta attain mature development within the peritoneal cavity of the mother. The protection of the *unruptured amnion*, however, appears to be absolutely indispensable for this development." The point claimed is that every abdominal pregnancy which has maintained

a prolonged growth, or reached full term, has done so within its own sac and which has separated it from the general peritoneal cavity, that this sac may exist as a scarcely perceptible membrane, or thin veil, and perhaps spread out from organ to organ, and intestine to intestine, nevertheless, it can on careful observation always be demonstrated. In cases reported of the child lying naked in the peritoneal cavity he asserts that a careful analysis of official reports always gave evidence that such a membrane or sac existed. Mordecai Price, [12] in an article on this subject, is still more sweeping. He says :—" The only way an extraperitoneal pregnancy can come to term is encapsulated in the amniotic sac. The sac is a foreign body in the peritoneum and adheres to everything coming in contact with it. The adherent viscera protect as well as nourish the displaced ovum. The peritoneum would digest the fœtus at any age not protected in some way from its influence."

The further formation and growth of the placenta after primary rupture is another interesting field for investigation and study for the obstetric surgeon. What to do with the placenta is one of vital importance. A mis-step in its management at the time of operation may cost the patient her life. Sutton and Giles [20] write :—" A uterine placenta consists of fœtal and maternal elements, but a tubal placenta possesses fœtal elements only, for in a tubal pregnancy a decidua forms in the uterus not in the tube, further the tubal mucous membrane takes very little share in the formation of the placenta." This is probably true in the early period of ectopic gestation, but as the placenta increases in size, and particularly after rupture, it not only takes up and changes into its own tissue that of the Fallopian tube but often also spreads out and becomes attached to organs and structures in the immediate neighborhood the back of the uterus, the broad ligament and the pelvic walls being favorite sites for such extension of attachment.

A third interesting field that might be presented for study is the management of full term ectopic gestations. So satisfactory have been the recent efforts of surgeons to take into consideration the life of the child as well as the mother that definite rules are being laid down for their guidance. Edwin B. Cragin in an article on " The treatment of full term ectopic gestation " asks and answers three very vital questions :—

1. Is the viable ectopic gestation worth saving ?

2. Do the attempts to save the child seriously increase the mortality or morbidity of the mother ?

3. What is the best procedure at the time of operation ?

As to the first question the writer replies in the affirmative and shows photographs of three fœtus which were in a good state of maturity. The only one of three born alive is reported as leaving the hospital a vigorous healthy child.

Taylor says in regard to this question, "So far as my own experience goes the extra-uterine child at term, in size weight and nutrition, is in no way behind the average. The child is often locally defective or malformed. These deformities are most commonly met with in children of abdominal pregnancies. The children of ligamentary pregnancies, and particularly those of the posterior variety, may be free from any visible defect whatever."

As to the second question Cragin says :—" Maternal mortality, when the operation is performed during the viability of the fœtus, will always depend very largely upon the judgement and skill of the individual operator in his decision as to the treatment of the placenta, whether it can be removed without great danger of fatal hemorrhage, or whether it is wiser to leave it to be removed or to come away later."

The third question is subdivided into a discussion on the best time to operate and the technique of operation. It is argued, and apparently with much reason, that while "any additional growth and development which can be given the child is a thing to be desired," it must be admitted "that at the completion of the full period of gestation, and during the spurious labor, there is some danger of rupture of the gestation sac and fatal hemorrhage," and accordingly, "when the fœtus is alive, and with the mother under careful consideration, and in the absence of unfavorable symptoms on her part, at about eight and one half months of gestation is the most desirable time for operation in the interests of both mother and child."

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EFFECTS OF POLLUTED WATER ON FISH LIFE.

(Extracted from Dr. Knight's report to the Minister of Marine and Fisheries.)

THE inquiry detailed in the following pages aimed at eliciting as accurate information as possible in regard to the effects of polluting our waters with (*a*) saw-dust; (*b*) nail waste from nail factories; (*c*) waste water from pulp mills; and (*d*) waste water from gas works.

The general method of investigation consisted in adding varying percentages of the waste water to fresh water or salt water, according to the kind of fish experimented with, and then immersing the living fish in the mixture and noting the effects upon them.

A "control" experiment was usually carried on along with those in waste water. This "control" consisted in placing a normal, vigorous animal in unpolluted water, so that observations on the fish immersed in the polluted water could be simultaneously compared with observations upon the animal in normal water.

Some preliminary experiments were undertaken for the purpose of determining (1) the shape of the vessel in which the fish

should be placed, and (2) the volume of water to be used in proportion to the weight of the fish. Should the dishes used be broad and shallow, or should they be tall and narrow? Should large quantities of water be used in proportion to the bulk of the fish, or should comparatively small quantities be used?

The answer to the first question was obtained by repeating the following experiment a number of times. Two rock bass (*ambloplites rupestris*) of equal weight were placed in separate vessels, each vessel containing 3½ litres of lake water. One vessel was an ordinary agate-ware baking pan, 13¼ inches long, 9½ inches broad, and 1¾ inches deep. The other vessel was a tall, cylindrical museum jar (external diameter 6 inches), the water in which stood 8¾ inches high. The experiment began at 10 a.m. At 5 p.m. the one in the tall vessel was lying on its side in a dying condition. The next morning at 10 o'clock it was dead, while the fish in the shallow pan was quite lively. The same result occurred whenever this experiment was repeated.

Such experiments evidently show that ventilation or aeration of water is as important in fish respiration, as ventilation of air is in mammalian respiration. They show that ventilation goes on naturally and easily in the shallow water of a broad, flat vessel. In such a vessel a large surface of water is exposed to the air. As the oxygen dissolved in the water gets used up by the fish, fresh oxygen is absorbed from the air, the absorption being facilitated by the movements of the fish, which agitates the water and exposes a fresh surface to the air.

On the other hand, the water in a tall narrow vessel has a comparatively small surface exposed to the air, and a fish, usually lying at the bottom, does not agitate the surface so as to promote aeration of the water.

This experiment evidently explains how trout can live so well in a very small trout stream in dry weather, and also explains how minnows can be kept alive by turning them loose in water in the bottom of a fishing boat.

The second question, should large quantities of water be used in our experiments, is not so easily answered. The proper quantity will depend upon the extent of ventilation. If artificial ventilation be applied to the water during the experiment, then a relatively small volume of water will do. If no artificial venti-

lation be applied, then, of course, a much larger quantity of water should be used, and it should be placed in a large shallow dish.

In connection with this subject a number of experiments were tried for the purpose of determining the length of time that unit weight of fish (1 gram) would live in unit volume (1 cubic centimetre) of unventilated water. Fish were weighed and placed separately in sealed jars, completely filled with a known volume of water and the length of time they lived was then carefully observed. The following was a typical experiment :—

Weight of fish, 76 grams; volume of water, 5530 c.c.; lived 6 hours. Therefore 1 gram weight of fish lived in 1 c.c. of ventilated water for about 5 minutes.

Ten similar experiments on rock bass of different sizes gave 7 minutes as the average time during which unit weight of fish could live in unit volume of unventilated water—the range being 5 minutes as the minimum, and 9 minutes as the maximum. The temperature of the tap water with which these experiments were conducted was 22° c. When the water was cooled down to 4° c. the fish lived for a shorter time. When the temperature was raised above 22° c. they lived for a *shorter* time.

These figures for duration of life in fish confined in a limited quantity of water are interesting when compared with those given by Paul Bert,* for mammals breathing a limited quantity of air. Five experiments by this observer gave 8 minutes as the average length of time during which unit weight of mammal (1 gram) lived in unit volume (1 c.c.) of confined or unventilated air. From these data it will follow that mammals use 5 or 6 times as much oxygen as fish in the same unit of time; life is more intense in the higher animals.

These experiments suggested the possibility of determining the smallest amount of water in which a fish of a given weight could breathe for many hours, or even several days, on the supposition that this minimum quantity could be kept perfectly ventilated. Of course a fish requires something more to maintain life than ventilated water. Free movement is essential, not to speak of food; but apart from these and similar considerations it seemed worth while to conduct an experiment or two on the respiration of fish in a minimum amount of water.

*"Lecons sur la phys. comp. de la respiration," Paris, 1870. Page 510, quoted in Schafers text book of physiology Vol. I., page 743.

With this object in view, a perch (Perca Americana, Jordan) was placed in 600 c.c. water in a jar, and arranged so that a continuous stream of air was bubbled through it. There was just enough water to cover the fish. Its position in the bottle tended to throw the animal on one side, in which position it seemed to stiffen, for at the end of 24 hours it was removed from its prison with its body slightly curved to one side. In 3 or 4 hours it could swim slowly about the aquarium, but for days afterwards it had a kink in its tail. This experiment shewed that unit mass of fish lived in unit volume of aerated water for 130 minutes.

In another experiment of a similar kind a small rock bass lived for 74 hours in 700 c.c. of aerated water.

RATE OF RESPIRATION.

A few observations were made upon the rate of respiration in fish confined in an aquarium. Four rock bass breathed at the rate of 44, 48, 52 and 56 per minute in water at 22 °c. Rate of respiration here means the rate at which the gill covers were raised. When the water was cooled down to 5 °c. the rate in one of the animals fell to 16 per second, and when warmed to 32 °c. the rate increased to 112. Warm water (32 °c.) had another peculiar effect on rock bass. It caused the pigment cells (chromatophores) of the skin to spread out and give a decidedly darker hue to the whole fish. This became particularly marked when the animal was returned to the aquarium where it could be compared with the other fish.

Muscular exertion also increased the rate of respiration.

EXPERIMENTS WITH SAW-DUST.

A box 3 ft. long, 2 ft. wide and 14 in. deep, lined with zinc, was used as a tank in which to confine the saw-dust and fish. The box was covered with mosquito netting, and over this wire gauze. A pailful of old, that is water-soaked, saw-dust and about a quart of fresh saw-dust were placed in the tank. A V shaped trough conveyed water (from a dam or a very small trout stream) down to the tank. The tank itself was immersed in a small pool, the water of which came up the sides of the tank to within 3 inches of the top. Temp. in shade 16.9 ° c, in the sun 11.3 ° c.

About two miles up James' brook, from where it empties into Chamcook harbor, near St. Andrew's, N.B., was the site

chosen for this experiment. The water was clear and cool, and runs over a gravelly and stony bottom—a typical trout stream, containing a fair number of *salvalino fontenalis*. Primitive forest, or second growth elder, balsam, cedar, and various kinds of hard-wood covered the district through which the stream runs.

An hour's fishing in the brook furnished four speckled trout and an eel (*Echelus conger*) for the experiment. Two of the trout had been badly injured in the eye by the fish hook. These along with a frog were placed in a tank about 5.30 p.m. of July 6th, 1900, and the water turned on. The flow was abundant and continuous, the descent from the dam being sufficient to stir up the saw-dust into a gruel-like mixture, as thick as in any mill stream, no matter how much saw-dust may have been thrown into it. The conditions were therefore, as much as possible, like those prevailing in a saw-dust polluted stream.

The tank was not visited until July 11th, when all the animals were found alive, active, and apparently healthy. The frog was lying at the bottom, as he could get no air at the top on account of the cover.

About half-a-pail more of saw-dust, some sand and gravel were added, and the tank again closed.

On July 14th the brook was again visited. All four trout were alive, active and apparently well. The eel escaped as the cover was removed; the frog was dead.

About a dozen earthworms were thrown into the tank, but the trout did not touch them while under observation.

On the 21st of July three-fourths of the water in the tank was emptied out, and the tank, containing the four trout, was brought to the laboratory, St. Andrew's, a distance of about three miles, in a wagon, part of the journey being over a very rough road. On examination the four trout were found to be very lively and so active that they were only captured after emptying out nearly all the water. This ended the experiment, the conclusion being that if fish so sensitive as the trout could live in such a mixture for a whole fortnight without apparent harm, in fact with recovery from severe injuries, then any fresh water fish could live in any mill stream, no matter how badly polluted with saw dust.

Dr. Stafford conducted a post-mortem examination on one

of these trout and found only two very small pieces of saw-dust on one of the gills. Neither piece seemed to have injured the gills. A few filaments were slightly damaged at the outer and fore arch, but there was no evidence that this condition of the filaments was due to the action of the saw-dust.

My own post-mortem examination of two other animals showed no trace of damage from saw-dust.

While the experiment seems conclusive as regards the fact that saw-dust does not directly injure adult fish life, it by no means follows that streams polluted by saw-dust are harmless to fish life. Water-soaked saw-dust may, and no doubt does, cover longer reaches of river beds. The breeding grounds of fish may thus be interfered with. Fish that spawn on sandy and gravelly bottoms are not likely to spawn on beds of saw-dust. The saw-dust may also interfere with the development of aquatic insects, and thus reduce the food on which some fish live. So that although saw-dust itself may not be hurtful to adult fish, indirectly it may interfere very seriously with the laying of their eggs and the development of the young. No one, however, can tell; further investigation is necessary.

WASTE WATER FROM PULP MILLS.

In experiments with waste water from pulp mills five kinds of fish were used, viz., stickleback (*gasterosteus aculeatus*), "white-perch" (*Rocus Americanus*), brook trout (*salvelinus fontenalis*), rock bass (*ambloplites rupestris*), sunfish (*lipomis pallidus*) and sea chub (*fundulus heteroclitus*). As is well known stickleback frequent brackish water, or fresh water near the sea. They are very hardy and can live in stagnant pools and ditches where no fish life would ordinarily be expected.

A stickleback and a fundulus were placed in equal parts of pulp waste and pond water. In less than an hour both were dead. The vessels used had a capacity of about five litres and were floated in a pond, so that the temperature of the water used in the experiment was the same as that of the pond from which the stickleback were taken.

In another experiment in which the waste water formed 25 per cent. of the mixture, two sticklebacks, placed in the vessel at 5.30 p.m. of July 14th, were found dead the next morning at 10 a.m.

Reducing the amount of waste water to 10 per cent., it was found that two stickleback placed in such a mixture on July 16th lived until July 27th, when the animals were liberated.

Trout are much more sensitive to this pollution. One placed in a 10 per cent. mixture of pulp water and spring water lived from 5 p.m. of July 21st to about , p.m. of the 22nd.

White perch from Bocabec lake lived in lake water polluted with 10 per cent. of pulp waste water for about 36 hours.

Rock bass and sunfish lived about 24 hours in a similar mixture; but fresh water clams (*anodon*) lived for two or three weeks in it without apparent inconvenience.

These experiments indicate that river or brook water when mixed with 10 per cent. of waste water from pulp mills is decidedly poisonous to fish life. If, therefore, a large quantity of this waste is poured into a comparatively small stream, it must result in the destruction of fish; if into a large river, then it is difficult to see how any great harm could be done. The specific gravity of the pollution, 1.00005, being so very slightly greater than that of river water, shows that the water from a pulp mill would mingle readily with that of any stream into which it was discharged, and unless the pollution equalled or exceeded 10 per cent., no great harm could be done.

WASTE WATER FROM GAS WORKS.

This water is much more poisonous to fish life than the former and kills much more quickly. The very quickness with which fish succumb to its effects indicates that death results from poisoning with the sulphuretted hydrogen which the water contains. Confirmation of this view is afforded by the fact that if a fish does not die in the polluted water during the first 24 hours, it will usually live on in the pollution for several days. Besides, when a fish succumbs quickly, say, in 10 to 20 minutes, to the effects of this gas, it could usually be resuscitated by transference to pure water. Within 15 to 30 minutes after transference the fish was as lively as ever, especially if the water were agitated so as to increase the amount of oxygen dissolved in it.

The following were typical experiments: A *rocus Americanus* was immersed in a 5 per cent. solution of gas water, and in 20 minutes it was dead. Immersed in a 2 per cent. solution, the same kind of a fish survived about half an hour. In a ½ per cent. solution, the fish lived about half a day.

Stickleback endured the poison a much longer time. Of two sticklebacks, placed in solutions of this strength, one lived a day and a half, the other ten days, and was then liberated. I had reason for suspecting that the animal which died was not healthy when the experiment began. If so, its death was merely hastened by immersion in the pollution.

Trout are very sensitive to the effects of this poison. At 4.45 p.m. July 21st, I placed a trout in $\frac{1}{2}$ per cent. gas water-waste. In 10 minutes the animal was lying on its side on the bottom of the vessel. As it was evidently moribund it was removed so fresh water which I agitated by pouring water upon it from a height of a foot above the vessel. In 10 minutes more the animal had apparently recovered and lay quietly and comfortably at the bottom of the basin. In half-an-hour more it was very active and frightened if I approached.

A tom cod (*gadus tomcodus*) was placed in a $\frac{9}{10}$ per cent. solution of this waste in sea-water. In a few minutes it was lying on its back. When returned to sea water which I agitated vigorously, the animal revived.

Experiments with smelt gave exactly simiar results with $\frac{1}{2}$ per cent. solutions of this waste.

Fresh water forms, like the rock bass and sunfish, and salt water "chub" (*fundulus heteroclitus*) were much less affected. These forms were kept from two to three days in the pollution, some dying within 24 hours, and some surviving several days. The explanation would seem to be two-fold. In the first place these fish are constitutionally more resistant to pollutions of all kinds. In the second place, sulphuretted hydrogen in the mixture would largely diffuse into the air and decompose in the water, in an open vessel, during the first 24 hours. If the animal, therefore survived this period, it would probably die later on, through the poisonous effects of the other ingredients of the water, such as the sulphates and chlorides.

In estimating the poisonous effects of gas waste water, the extent of its dilution with river or lake water must be taken into account as well as its specific gravity 1.0023, and the volume of the stream or lake into which the waste is discharged.

WASTE WATER FROM NAIL WORKS, ST. JOHN'S, N.B.

This pollution was the most deadly one examined. In

many experiments one-tenth of one per cent. was sufficient to kill in a few hours. The most marked peculiarity in all experiments made with this waste was that in a few minutes after mixing it with either fresh or sea water a reddish brown precipitate began to form and continued forming for several hours. The suspicion that this was ferric hydroxide was confirmed by subsequent chemical analysis. Microscopic examination of the gill filaments of fish killed by this waste showed that death was caused by this adhesive precipitate sticking to the filaments and preventing respiration. With a coating of this rust-like substance covering the gills it is difficult to see how oxygen could pass into the blood and carbon dioxide could pass out.

Experiments began with solutions of 6 per ct, 2 per ct, and ½ per ct, all of which were found to be very poisonous, fish living from half an hour to an hour.

Reduction to ¼ per ct resulted in the death of the hardy stickleback in about five hours. They were able to survive for two or three days when the solution was reduced in strength to one-seventh per cent. In fact, when any of the hardier fish like fundulus, stickleback or rock bass, were able to survive the six or eight hours during which the ferric hydroxide was being precipitated, they usually lived on for several days or a week. More delicate fish, like smelt (*osmerus mordax*) and trout, however, succumbed to weak solutions of the poison in from ten minutes to half an hour. Repeated attempts to resuscitate these fish by artificial aeration in fresh water proved failures. In the case, therefore, of the more sensitive fish death, was apparently caused by the absorption of the free hydrochloric acid and ferrous chloride. That small quantities of ferrous chloride became absorbed was proved by treatment of the gill filaments with ferro-cyanide of potassium. This reagent stained the filaments a blue colour, and subsequent examination of sections of these under the microscope showed slight absorption of the iron compound along the surface cells. This proof was suggested by Dr. McCallum.

Attention is directed to the very high specific gravity of this pollution. The effect would be to cause the pollution to fall to the bottom of a stream into which it might be poured. This would result in the death of fish that frequent the deeper parts of a river, especially if the flow was sluggish. On the other hand, the great density would increase the rapidity of diffusion throughout the fresh water in accordance with the laws of diffusion of liquids of different densities.

A. P. KNIGHT.

QUEEN'S GRADUATES ABROAD.

FOR some time past the Graduates of Queen's who have made their home in New York have desired an organization to perpetuate and increase Queen's spirit. A fitting opportunity presented itself in the presence in New York of Principal Grant. A meeting of the graduates was arranged for at the residence of Dr. Geo. E. Hayunga, 581 Broome St., Manhattan.

After a few introductory remarks by Dr. Hayunga, the Principal was called upon and in a brilliant address outlined the growth of Queen's since he took up the duties of Principal and dwelt upon the benefits to be derived from the proposed social organization

Dr. Wolfred Nelson, a graduate of McGill, very kindly offered his assistance and that of McGill Society in the matter they had in hand and told of the benefits McGill's sons derived from a similar organization.

The ocffiers elected were :

Hon. Pres.—Principal G. M. Grant, D.D., LL.D.
President—James Douglas, M.A.
1st Vice-Pres.—Prof. F. Ferguson, M.D.
2nd Vice-Pres.—Dr. J. R. Shannon.
3rd Vice-Pres.—W. R. Givens, B.A.
Sec.-Treas.—Dr. Geo. E. Hayunga.

Executive Committee : Dr. S. H. Gardiner, Dr. W. G. Fralick, Robt. O'Loughlin, M.A., D. C. Porteous, B.A.

These delegates were elected to represent the Society at the October Ceremonies this fall, viz :—Jas. Douglas, M.A., Geo. E. Hayunga, M.D., and S. H. Gardiner, M.D.

Dr. Nelson invited the Society to join the other British Societies in New York, in presenting an address to the Duke and Duchess of Cornwall and York on their arrival in Canada, an invitation which was gladly accepted.

The executive is arranging for a dinner about Christmas at which there will be a re-union of many of the Faculty of Queen's and former students.

QUEEN'S STUDENTS AT THE ONTARIO MEDICAL COUNCIL.

At the recent Examination of the Ontario Medical Council we are pleased to note that the following Queen's men have passed :—

PRIMARY.

F. M. Bell, Kingston; F. W. Birkett, Ottawa; T. V. Curtin, Brockville; J. W. Edwards, G. C. Ferrier, Kingston; T. H. Houston, Belleville; L. W. Jones, Kingston; W. B. Kayler, Saginaw, Mich.; D. B. Kennedy, Pembroke; F. E. Mellow, Sillsville; T. O. McLaren, Lancaster; H. E. Paul, Newburg; W. J. Patterson, Peterboro; A. W. Richardson, Kingston; W. C. Redmond, Bethel; R. M. Reid, Renfrew; A. A. Staley, Wolfe Island: D. T. Smith, Ottawa; E. Sheffield, Peterboro; G. S. Sadler, Pakenham; W. G. Tyner, Kingston.

INTERMEDIATE.

I. G. Bogart, Borwick, Ont.; F. W. Birkett, Ottawa; F. F. Carr-Harris, J. W. Edwards, Kingston; G. S. Genge, Holleford; W. S. Grimshaw, Kingston; A. D. Macintyre, Glencoe; J. McCulloch, Port Perry; H. E. Paul, Newburgh; E. Richardson, Brockville; A. W. Richardson, Kingston; W. C. Redmond, Bethel; G. S. Sadler, Pakenham; W. G. Tyner, Kingston.

FINAL.

F. W. Birkett, Ottawa; J. W. Edwards, Kingston; W. B. Kayler, Saginaw, Mich.; A. F. McLaren, Lancaster; H. E. Paul, Newburgh; A. W. Richardson, Kingston; G. S. Sadler, Pakenham, W. G. Tyner, Kingston.

We extend our congratulations to these young men and wish them further success.