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Original Communications.

ON SO-CALLED FUNCTIONAL HEART MURMURS.[1]

BY

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(From the Medical Clinic of the Royal Victoria Hospital, Montreal.)

Among the signs of organic disease which perplex the diagnostician by appearing when the organism is in health, or at least upon its borderland, cardiac systolic murmurs take a prominent place. Their occurrence as significant of a purely functional disturbance without any underlying valvular lesion, is of course well recognised, but their frequency in this connection is perhaps scarcely appreciated by the profession. In the discussion on "The Prognosis of Cardiac Disease in its Bearings upon Life Insurance," led by Sir Wm. Gairdner at the recent meeting of the British Medical Association[2] systolic murmurs are barely mentioned among all the doubtful signs of cardiac disease complicating an "estimation of life." Yet it is chiefly in relation to such questions as those of life insurance that the subject is of special interest and assumes some proportions as a difficult problem demanding solution. Indicative on the one hand, of that form of organic disease which most seriously threatens longevity, yet, on the other, consistent with nothing more serious than a temporary lowering of the general tonus, a well marked systolic murmur is capable of plunging the conscientious medical examiner into most uncomfortable doubt. For the subject is unfortunately as obscure as it is important.

Not only is the murmur subject to all the variations governing the principles of sound, but further, one's conclusions can have but a relative worth, for they are rarely verified by autopsy, and can only be

[1] Read before the Montreal Medico-Chirurgical Society, Nov. 21st, 1898.

[2] British Medical Journal, Sept. 17th, 1898.

formed by continued observation of cases and by amassing an abundant material.

Some months ago, Dr. C. F. Martin suggested to me a statistical study of systolic murmurs as they occurred in the medical records of the Royal Victoria Hospital. I am indebted to the authorities of the hospital for their kind permission to carry out this suggestion and I have to thank Dr. Martin for much assistance in the arrangement and revision of the work.

It must be admitted that the term "Functional Murmur" as at present usually employed, is a misnomer. On the one hand, *all* murmurs are functional, whether due to organic disease of the heart or to a malady of the blood itself, inasmuch as they depend on an impairment of function of the valves, or the parts in their immediate vicinity, or else to eddies abnormally carried in the course of the circulation. On the other hand, what are usually called functional murmurs, are often, indeed very often, due to a degeneration of the heart muscle, be it in the cells of the wall itself or in the cells of the papillæ or trabeculæ.

It is difficult to define functional murmurs in any other way than as temporary murmurs occurring in a heart with no other signs of valvular disease, and which ultimately disappear. This will exclude the organic murmurs which from time to time cannot be heard at all even when extensive valvular vegetations occur, and will admit of the term being applied where chlorosis, typhoid fever, etc., occasion murmurs through the altered conditions of blood or heart muscle. Hence we would say that temporary parenchymatous degeneration of the myocardium produces functional (not organic) murmurs.

Leube[4] has recently analysed the various conditions under which systolic murmurs occur and the requirements for a differential diagnosis. His classification is, briefly, somewhat as follows:

1. *Accidental Murmurs:* Systolic, heard most strongly at the base; may or may not be transmitted; no pulmonary accentuation; no increase of cardiac area.

2. *Relative Mitral Insufficiency:* Pure systolic moderately loud murmur; a weak impulse; moderate pulmonary accentuation; moderately increased cardiac area to right and left; relatively small often irregular pulse (myocarditis or myo-asthenia); history.

3. *Acute Mitral Endocarditis:* Soft systolic murmur at apex; cardiac area slightly increased to the left; pulmonary second moderately accentuated; pulse and heart impulse relatively strong, coexistence of fever or of some infectious disease.

[4] Zur Diagnose der Systolischen Herzgeraüschen, Deut. Arch. f. Klin. Med. Nov. 5th, 1896.

4. *Chronic Disease of Mitral Valve:* Systolic murmur; generally louder and harsher; increased cardiac area; marked pulmonary accentuation; impulse moderately increased; pulse relatively strong and regular; frequent association with the signs of mitral stenosis; history of adequate cause for assuming organic cardiac disease.

Pure systolic murmurs then may be regarded as:

(a) *Valvular*, depending on an organic deformity of the mitral valve, or upon its relative incompetence due to so-called cardiac myoasthenia or to a myocarditis; and

(b) *Non-valvular*, accidental, or hæmic, heard best over the base of the heart and produced, according to most authorities, in the great vessels by lack of tone in their walls, or by lowered peripheral blood pressure. Such pure accidental murmurs when typical are supposed to present little difficulty in diagnosis for they are basal and unaccompanied by pulmonary accentuation or increase of the cardiac area, and claim differentiation only from aortic and pulmonary stenosis.

These latter are not easily confounded with basal functional murmurs. But with apical murmurs the matter is quite different and presents constant perplexity. Here it is necessary for prognosis to differentiate not only a relative incompetency from the true insufficiency of a diseased mitral valve; it is also most important to distinguish the permanent relative insufficiency of organic cardiac disease (primary dilatation with hypertrophy, myocarditis, etc.), from the temporary relative insufficiency of anæmic or febrile conditions where the valves fail to close simply from weakness of the papillary muscles and trabeculæ, or from dilatation due to lack of tone of the cardiac muscle itself.

From Jan. 1895, to Sept. 14th, 1898, the number of cases admitted to the wards of the Royal Victoria Hospital, were some 3,302; of these, I have examined the case reports of 2,780. All cases were rejected whose histories showed pure systolic murmurs known to be due to organic cardiac disease, whether pericarditis, myocarditis or endocarditis, and, as possibly organic, all cases where there was a record of previous or concomitant chorea, tonsillitis, acute and chronic rheumatism, arterio-sclerosis and acute and chronic Bright's disease. I retained for study 589 cases in which pure systolic murmurs occurred (21 per cent). After eliminating cases giving a previous history of rheumatism, nephritis, etc., or showing arterio-sclerosis from this number, there remained a total of 466 cases, or not quite 17 per cent. of pure systolic murmurs occurring in patients in whom there was nothing either in the previous history or in the general condition to suggest organic cardiac disease; with the excep-

tion of a possible old endocarditis based on a previous history of scarlatina which occurred in a certain proportion of cases, and which I noted but did not eliminate.

This (17 per cent.) is a high percentage, and it will be objected to it, that without doubt many of these are really cases of organic cardiac disease of insidious onset; but when one considers that in some of the many cases rejected because there was a bare suggestion of etiology, the murmurs may have been functional, and further, that, present as this sign often is when the patient is suffering from a malady that would never suggest it, the less marked murmurs must, even in these carefully kept records, have sometimes escaped observation, it is evident that this percentage of 17 per cent. must be pretty close upon the facts.

In the subjoined classification, various plans are adopted and the effort has been made to seek a diagnosis of the underlying physical condition. In functional murmurs, this is generally admitted to be an anæmia or a toxæmia leading, possibly through the nerve centres, to lowered vascular tonus and to consequent irregularities in the blood current.

I have divided all the conditions as follows:

1. Murmurs occurring in febrile and afebrile conditions.
2. Murmurs occurring in anæmic and non-anæmic conditions.
3. Murmurs occurring in pulmonary tuberculosis. This being kept separately as here the condition includes to a marked degree, fever with cachexia and anæmia.
4. Classification according to the nature of the sound with special reference to:

(*a*) Site of murmur.
(*b*) Transmission.
(*c*) Condition of pulmonary second sound.
(*d*) Rhythm.
(*e*) Cardiac enlargement.

With reference to the *statistics* it is of note that:

In 466 cases with undoubted functional murmurs, 269 were in afebrile, and 163 in febrile cases.

Of the 269 afebrile cases, 185 showed anæmia; 74 none.

Of the 163 febrile cases, 85 showed anæmia; 78 none.

In 44 cases of pulmonary phthsis, 35 showed anæmia: 9 none.

The following *table* will indicate the main features it is desired to illustrate as suggested in a *few of the diseased conditions* with functional murmurs and the characteristics associated therewith. The relative frequency of murmurs in febrile and afebrile conditions and in pulmonary phthisis is also shown.

DISEASES.	Totals	Sub-totals.		Systolic Murmur at Apex.		Systolic Murmur at Pulmonary Area.		Systolic Murmur at Apex and Base.		Systolic Murmur at Aortic Cartilage.		Systolic Murmur Irregularly Situated.		Diastolic.	
		Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia	No Anæmia.	Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia.	No Anæmia.	Anæmia	No Anæmia.
Anæmias	87	87	....	21	....	21	....	36	....	5	....	1	....	4	....
Pernicious Anæmia	16	16	....	9	....	1	....	5	....	1	....	....	....	....	....
Chlorosis	46	46	....	7	...	15	....	19	....	3	....	....	....	2	....
Leuchæmia	4	4	....	2	....	....	....	1	....	....	....	....	....	1	....
Hodgkins	3	3	....	1	....	1	....	1	....	....	....	....	....	....	....
Secondary to Hæmorrhage or C—a.	22	22	....	5	....	4	....	10	....	1	...	1	....	1	....
Typhoid Fever	78	31	47	9	18	12	9	5	15	3	3	2	2	...	....
Hysteria and Functional Nervous Disorders	61	30	31	13	14	11	8	5	5	....	4	1	1	....	2
Exophthalmic Goitre	12	4	7	1*	2	2	1	1	3	....	1	....	....	....	1
Pulmonary Tuberculosis	44	35	9	11	4	12	4	8	....	1	1	2	....	1	....
Febrile Conditions	163	85	78	20	32	37	10	22	26	4	7	3	3	....	....
Afebrile Conditions	239	185	74	67	36	47	16	49	7	9	6	7	5	6	4
Total number of Murmurs	466	305	161	98	72	96	30	79	33	14	14	12	8	7	4

* Tachycardia only.

It will be seen from the foregoing how frequently functional murmurs have been found when no apparent cause was discovered; neither anæmia, fever or other usually recognised cause; and further, that in site and rhythm of murmur some unusual conditions occur.

In the *true anæmias* are included, pernicious anæmia, chlorosis, Hodgkin's disease and anæmia secondary to hæmorrhage or carcinoma. These form 87 cases of which 24 have apex murmurs; 36 have murmurs at apex and base, while in only 21 were the murmurs mainly at the pulmonary cartilage.

The *origin* of the murmur cannot always be read from its situation; Dr. Heitler, in an article on "The Localisation of Systolic Mitral Murmurs"[4] makes an exhaustive study of this point. He states that his researches lead him to disbelieve the dogma that murmurs of different *timbre* are necessarily of different origin, even though they be heard less loudly in the interspace. He argues that murmurs are formed of a mixture of sounds which are not transmitted as a whole in any direction, but that certain parts are heard best where the conditions for transmission are most favorable, and he cites a case, seen post-mortem to be one of pure mitral regurgitation, where there was a loud musical murmur at the apex growing fainter towards the base and a loud murmur at the aortic cartilage of the same quality as that at the apex.

This is confirmed by other authorities in the case of regurgitant murmurs due to endocarditis where roughened surfaces combine with altered blood currents to produce a mixture of sounds, but in functional cases there are probably less complicated conditions. In these anæmias where the double murmurs which occurred, are often described as of different *timbre*, rough at one orifice, blowing at the other, they are frequently transmitted in two directions: into the axilla from the apex, and upwards from the pulmonary orifice, indicating that they really are the double murmurs of a relative mitral insufficiency and of a physiological pulmonary stenosis. Such at all events seems quite as plausible an explanation as any other.

Leube, in his article lays much stress on the site of the murmur. He states that in lesser degrees of blood alteration, accidental murmurs are produced in the great vessels at the base from lowered tonus of their walls through the action of impoverished nerve centres, while the more severe grades of anæmia and intoxication lead to a myo-asthenia or even a myodegeneratio cordis, and to the *apical* murmurs of relative mitral insufficiency.

The anæmic cases I have studied tend emphatically to confirm this

[4] Wiener Klin. Woch., No. 17, 1897.

dictum. In pernicious anæmia, representing the most extreme degree, we know that there do exist often extensive degenerative changes in the myocardium leading to weakening of the heart and the papillary muscles and to consequent dilatation. Here then are the conditions for a relative insufficiency, and here every murmur is apical. Of the 16 cases, 10 occurred alone at the apex and pulmonary cartilage, and the only one heard best at the pulmonary is transmitted into the axilla, indicating that it too is an apical murmur. In chlorosis, on the other hand, where there is a lesser degree of blood alteration, there are only seven cases at the apex against 15 at the pulmonary area, and 19 heard with equal force both at the pulmonary cartilage and apex. Of the seven cases with a murmur at the apex, five showed only slight chlorosis, scarcely sufficient one would think to produce a relative insufficiency; the thought is suggested that although mitral valvular murmurs are heard best at the apex, all apical murmurs are not necessarily valvular.

The apical murmurs of pernicious anæmia, however, do not always present the other cardiac conditions that we would expect to find in a relative mitral incompetency of some duration. The murmur is generally transmitted, but in only ten cases is there cardiac enlargement or pulmonary accentuation; but the absence of these signs is explained by the physical facts. We cannot expect much increase in area where the heart though frequently dilated, is atrophied rather than hypertrophied, and the right ventricle must often be too weak to give rise to pulmonary accentuation. In pernicious anæmia where pulmonary accentuation does occur (as in most advanced cases) it is probably dependent on the dyspnœa, due to the imperfect aëration of the tissues by the diminished number and power of the oxygen-carrying elements of the blood.

The cases of *exophthalmic goitre* illustrate the same point as do the anæmias. It is known that in this disease in cases where there is much vasomotor disturbance, there is apt to be dilatation of the over worked heart resulting in a relative mitral insufficiency. In the ten cases before me there is only one that does not show marked implication of the vasomotor system; in this one there is but a slight anæmia (erythrocytes 4,000,000, hæmoglobin (Fleischl) 70 per cent.) and the murmur is hæmic in its character being heard only at the pulmonary cartilage without pulmonary accentuation or cardiac enlargement.

In all the other nine cases there is vasomotor disturbance and the murmur is at the apex, generally well transmitted into the axilla. In five of the cases another murmur is also described at the pulmonary,

in three of these, this second murmur is probably hæmic in character for there is marked anæmia.

Typhoid fever may be taken as an example of a condition in which functional murmurs are extremely frequent, which contrasts strongly with anæmia. Here the more rapidly acting heart, the low tension pulse, the increased metabolism and evident intoxication of the system explain much more readily the strain on the cardiac system, of which the murmur gives evidence, than do the changes associated with a moderate anæmia. But, under such widely differing clinical circumstances, a common symptom makes one look for a common cause and the inference lies near, that in anæmia as in typhoid, a toxæmia rather than a hydræmia is at work, in the one case acting slowly and insidiously, in the other, suddenly, acutely, and poisoning rather than impoverishing the nerve centres. Other facts suggest this idea also. Everyone knows that one meets with systolic functional murmurs often in apparent health, and in 78 of these 466 cases, there is present neither anæmia nor fever, but often a condition which suggests some form of intoxication. Instances of such conditions included among these murmurs are: 43 diseases of the digestive system (including six cases of cirrhosis), gout, acute alcoholism, morphinism, etc.

Out of about 298 cases of typhoid examined, a pure systolic murmur was noted in 78, making something over 29 per cent.

In this disease as in all infectious fevers, an apical murmur always suggests the possibility of an acute endocarditis; this is however, rare; Osler states that he did not find it in any of his cases and that it was present in only 11 of the 2,000 Munich autopsies. Parenchymatous degeneration on the other hand is undoubtedly common, and yet though this is naturally often the main underlying cause for these murmurs, they often declare themselves functional in other ways, by disappearing during the active course of the disease.

Since the year 1896, minute daily records have been kept of the typhoid cases in the hospital. These "typhoid charts" reveal some interesting facts as regards the cardiac condition. When a murmur develops, although it frequently has a course parallel to the height of the fever, it often, too, appears and disappears quite irregularly without any apparent reference to this. In no condition of high continued fever are the variations in the cardiac condition more striking than in typhoid. The sounds vary in character and relative intensity; murmurs appear and disappear; signs of cardiac dilatation come and go, as the struggle between the reactive powers of the individual and the invading toxins of disease goes on in the organism.

The seemingly casual occurrence of these murmurs at the apex replacing those at the pulmonary and again a few days later giving place to them, differing too in no way from the basal murmurs in quality, makes one question Leube's statement that the hæmic murmur is always at the base and that a murmur at the apex is always due to a relative mitral insufficiency.

The transient murmurs of typhoid are best illustrated by a glance at two or three of the cases:

CASE I.—(Murmur corresponds with course of fever). A. B., admitted on 8th day of disease. High temperature until 25th day when defervescence began; normal temperature 41st day.

On admission, faint blowing systolic murmur at apex, not transmitted; louder murmur at pulmonary cartilage; faint murmur at aortic, accentuation of pulmonary 2nd sound. These conditions noted daily until 17th day when the basal murmur disappeared; that at the apex persisted; on the 24th day (commencing defervescence) this also disappeared. The heart was examined daily until the 62nd day and there was no recurrence of the murmur.

CASE II.—(Murmur ended with pyrexia but disappeared and reappeared again during its course.) C. D., admitted on 22nd day, temperature 105⅘°; high pyrexia until 42nd day; temperature slightly lower (102°) until 45th day, when defervescence began; temperature not normal until 95th day.

On entrance a blowing systolic murmur was heard at apex and pulmonary cartilage, noted for three days; on 27th day, no murmur at pulmonary nor at apex, but here lengthening of the 1st sound; on 33rd day, a faint blowing systolic murmur developed at apex, noted daily until 65th day when it disappeared; daily examination of the heart until 95th day revealed no return of murmur.

CASE III.—(Murmur at pulmonary cartilage during height of fever: interval with no murmurs; murmur developed at apex during convalescence). E. F., admitted on 27th day, defervescence began on 42nd day, temperature normal on 48th, no recrudescence.

On entrance, systolic murmur at pulmonary with pulmonary accentuation; this was noted every day until its disappearance on 36th day; on the 61st day (temperature normal; patient doing well in every way; still quiet in bed), a systolic murmur developed at the apex, heard as a rougher sound at the pulmonary; noted daily until the 69th day when it disappeared. On the 71st day (two days interval with daily note of no murmurs) murmurs were again heard at apex and pulmonary which persisted (daily note) until the 84th day. Patient discharged 87th day; pulmonary 2nd sound remained accentuated at exit.

Hysteria and functional nervous disorders.—In these, out of 62 murmurs 32 occurred in subjects who showed no anæmia; 14 of these murmurs occurred at the apex; eight at the pulmonary cartilage; five as double murmurs at both apex and base while one is heard at the aortic cartilage. Some of them bear all the characters of functional murmurs, being unaccompanied by other signs of cardiac disturbance, either enlargement or pulmonary accentuation. With no anæmia and no intoxication present, how are we to explain the temporary disturbance of the vasomotor system which the systolic murmur indicates? Is it possible that here, not poisoned nor impoverished, but disordered nerve centres are acting, and doing their part as inefficiently as do the higher inhibitory centres?

Lastly, with reference to the *nature of the sounds*, the following features may be of interest:

As regards the *site* of the murmur, it occurs at the apex in 170 cases, in 98 of which there is anæmia, in 72 none. It is at the pulmonary area in 126 cases. in 96 there is anæmia, in 30 no anæmia.

In 112 cases it is heard both at apex and base, sometimes with equal force, sometimes as a double murmur; in 79 of these there is anæmia, in 33 none.

In 48 cases the site is irregular (at the aortic or ensiform cartilages, etc.), 26 of these occur in anæmic and 22 in non-anæmic subjects.

In *character* the murmur is described as "soft, low, blowing," often "faint," sometimes "short" and "long," about 20 times; in three cases it is described as "musical," twice at the apex and once at the pulmonary area. It is described 42 times as "rough" or "harsh," and in all but 15 cases this applies to a murmur situated at the pulmonary cartilage.

Intensity is a point of some importance, for the murmur is in general low as opposed to the usually louder and harsher organic murmurs, but no hard and fast rule applies to anything about a functional murmur. "Loud" is applied to some 16 cases.

As regards *transmission*, murmurs heard at the apex are transmitted "towards the axilla" in 43 cases, and "into the axilla" in 73 cases. In 7 cases the murmur is described as heard "in the back" and "at the angle of the scapula." These cases are, 4 of chlorosis, 1 of lymphatic leukæmia, 1 of incipient exophthalmic goitre, and 1 of tuberculous meningitis. In none of these, except perhaps the latter, is there reason to suspect organic cardiac disease.

Pulmonary accentuation occurred in 191 cases, in 127 of which the pulmonary system was diseased. In about one-third of the cases, it was noted as "slight," more often it was present in anæmic subjects.

The *rhythm* of the functional murmur, with more special reference to the diastolic sound. Among the cases studied I have found 10 diastolic murmurs which I have not been able to reject as organic by any of the criteria I have used. They occur in the following diseases:

Chlorosis, splenic leukæmia, functional and cardiac disturbance, exophthalmic goitre, catarrhal jaundice, cerebral thrombosis, chronic bronchitis, and dementia.

So distinguished an authority as Strümpell states positively that he himself has observed a case of undoubted functional murmur which was diastolic in rhythm; and Sahli explains this (when occurring at the base) as a transmission of the *bruit de diable* from the neck. Leube, on the other hand, considers them so uncertain as to be better left aside in making any critical enquiry into the subject. He himself has never heard a diastolic murmur which he believes to be functional. Without discussing this disputed point, I quote a few of the cases:—

1. *Exophthalmic Goitre:* Blowing systolic murmur heard best at apex, heard also over præcordium and at aortic and pulmonary cartilages. Faint blowing *diastolic* murmur at pulmonary cartilage, heard down along left border of the sternum; pulmonary 2nd sound heard louder than aortic 2nd; dulness normal; apex beat normal position; 1st sound of heart heard at apex; pulse compressible.

2. *Phthisis:* Patient died 13 days after admission. Heart normal on entrance except for accentuated pulmonary 2nd sound, five days ante-mortem, systolic and *diastolic* murmurs were heard at 3rd right interspace, transmitted down right border of sternum; persisted until death.

3. *Splenic Leukæmia:* (Hæmoglobin 14 per cent.). Blowing systolic murmur at apex, transmitted to axilla; blowing systolic murmur at pulmonary transmitted upward; systolic and *diastolic* murmurs at aortic cartilage; pulmonary accentuation and 2nd sound at apex much accentuated; apex beat and dulness in left mammary line; pulse regular, tension low.

4. *Dementia:* Patient was a boy of 15, small and undeveloped; *diastolic* murmur at pulmonary cartilage; no enlargement of cardiac area or accentuation of pulmonary second sound.

Some *cardiac enlargement* was noted in 66 cases, and seemed gen-

erally to be consequent on a relative mitral insufficiency. In ten cases, relative dulness began at the right sternal border, in 30 cases it was noted at the midsternum.

General remarks: A functional murmur is usually, though not always, systolic in rhythm; while frequently at the base, it is very commonly situated at the apex; in the more severe degrees of anæmia or intoxication it is more common at the apex, in milder disturbances at the base. In intensity it is generally "low" and "faint"; in quality, "soft" and "blowing," especially when at the apex. Rough, harsh, functional murmurs are generally situated at the pulmonary cartilage. The murmur is often transmitted from the apex to the axilla, and, as due to a relative mitral insufficiency, may even be heard in the back. Moderate pulmonary accentuation is frequent. In those cases where the murmur is basal and appears to be accidental, it is generally associated with anæmia. Moderate enlargement of the cardiac area is fairly common, and points to a relative mitral insufficiency.

The cases studied illustrate especially the following points:

1 In cases of anæmia, pulmonary accentuation is often associated with a pure accidental murmur.

2. Functional murmurs frequently occur where there is neither anæmia nor fever. They are then often associated with some other condition suggesting intoxication.

3. Diastolic murmurs have been noted which do not appear to have an organic origin.

4. Although accidental murmurs are generally heard at the base and those of relative mitral insufficiency at the apex, accidental murmurs are probably sometimes heard at the apex; (as in moderate anæmias where the murmur may occur at the apex unaccompanied by pulmonary accentuation or cardiac enlargement and disappear after a short time; or in high fevers where a murmur at the apex is replaced after a few days by one at the pulmonary cartilage of the same character.) On the other hand; murmurs produced at the mitral valve are occasionally, though rarely, heard best at the pulmonary cartilage.

Two conditions which it may be quite impossible to distinguish from each other by physical signs are:

A functional murmur at the apex with signs of moderate dilatation (relative mitral insufficiency), and an organic mitral murmur with signs of compensatory change. A decision can often only be reached

by considering the patient's general condition and the *persistency* of the murmur. This last is the clinching point and is the final criterion to which uncertain cases must be brought. In plain terms, *we must wait to diagnose the murmur until it is no longer there to diagnose.*

In this short paper such a large number of case reports have necessarily been dealt with in the most superficial way. Many points,—such as the persistency of the first sound of the heart—have not even been mentioned; and there remain the 123 cases where the previous history or present condition suggests organic cardiac disease; a comparison of these with the 466 cases glanced at in this paper where the murmurs were all apparently functional, would be very interesting. But a closer study of a subject such as this, is better combined with work at the bedside. The examination of these cases yields results which are useful chiefly as a basis for furthur study, and which become valuable when the conclusions drawn from them are confirmed by the prolonged observation of individual cases by a single observer. I hope at some future date, through the continued kindness of the authorities of the Royal Victoria Hospital, to be able to make further use of the material before me in a more thorough investigation of the subject.

PUERPERAL SEPTICÆMIA.[1]

BY

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Among those whom I have the honor of addressing this evening, there are a few who, although comparatively young, have still like myself, lived long enough to have been the contemporaries of that dreadful disease which we used to call *puerperal fever*. The remembrance of this multiform plague which, at times, seemed to cling exclusively to the clothes of one *accoucheur* in particular, I am sure, is still fresh in their memory. All the parturients with whom he would come in contact would be stricken by the disease, and with such deadly results that he would be compelled to give up obstetrical practice until time would allow of his getting rid of the virulent atmosphere which appeared to surround him individually.

The oldest among us have witnessed these terrific cases which used to kill in a few days, as they have also known those epidemics of erysipelas, hospital gangrene, as well as those wounds flooded with pus which we used to call "*laudable*," so proud were we to establish a distinction between it and the fetid sania which in every case was rightly considered of such a bad omen. It was the pre-antiseptic epoch, and at that time the true character of puerperal fever was enshrouded in the same deep darkness as all the other infectious diseases.

At the time, already very remote, when I began my medical studies, we were far from possessing the richness of means that clinical investigations afford us to-day. Wunderlich had not yet armed us with the thermometer which enables us to follow and register with such a precision the oscillations of animal heat during illness. *Tactus eruditus* and other phenomena would, however, easily permit us to ascertain the existence of fever. The cause of this fever was carefully looked for in every case, and generally it was found to be due to inflammatory action going on in some parts of the organism. At one time the lung was inflamed, at another the pleura, the peritoneum, the cellular tissue, and so on; the elevation of temperature was the consequence of that inflammation and the fever was called *symptomatic*. But in other cases, no organs would show sign of suffering, nowhere would the most careful examination detect the least inflammatory process, a certain amount of mere functional disturbance only would

[1] Read before the Medical Society, Ottawa.

accompany the sometimes considerable elevation of temperature of the body. In short, no appreciable anatomical lesions whatever could account for the fever which therefore was declared to be *essential*. Such were: scarlet fever, measles, variola, enteric fever, and at last, puerperal fever. The morbid phenomena which characterized these diseases were consigned to a special corner of the nosological frame and constituted a group to which were applied the denominations of infectious, virulent, zymotic affections. The etymology of these various names very well showed the tendency towards the right direction, and the terms virus, infection, ferments, seemed to foretell, as it were, the dawn of the greatest discovery of this century. Still the vagueness of those purely hypothetical conceptions brought but little satisfaction to the numerous observers deeply absorbed by the anatomical researches and who had at their disposal, means of investigations improving more and more every day. These indefatigable workers, digging with utmost eagerness the pathological tissues down to the last fibres, were incessantly in hope of finding in the ultimate revelations of post-mortem examinations the anatomical explanation of morbid phenomena, the etiology of which had escaped their predecessors. It is especially on the ground of puerperal fever that the conflict assumed the most gigantic proportions. Two opposed camps were in presence, and the combatants on either side made, at that time, the halls of scientific societies resound with the expression of their respective opinions. On one side puerperal fever was considered as a morbid entity, a disease "*sui generis*," produced by an invisible agent of an unknown nature, giving rise to manifestations, the explanation of which could not be found at the post mortem examination of the organs whose integrity would generally seem perfect. Those who fostered that opinion were called *Essentialists*. The others refused to accept the essentiality of puerperal fever. To them, there existed, after labour, a wound in the uterus. This wound, like any other traumatic lesion, remained exposed to inflammatory complications which would spread by continuity or contiguity of tissues up to the abdominal cavity, or end in the formation of pus which, absorbed by the blood-vessels or the lymphatics, would cause disorders in parts more or less remote from the initial point. And, they would add, if other observers had not found the presence of material lesions at the autopsy, it is because they did not know how to look for them, for they existed in every case and would account for the clinical manifestations offered during life. The latter were called "*Anatomicists*," and what a masterly description have they not left us of the course and symptoms of puerperal fever! They acknowledged three prin-

cipal forms of the disease : the first, characterized by pain and tumefaction of the appendages, appreciable by external palpation, cephalalgia, tympanites, diarrhœa ; all symptoms spontaneously terminating within five or twelve days. They were but mere puerperal disorders, simply due to an inflammatic having its starting point in the uterine wound, and thence propagating more or less to the peritoneum or to the cellular tissue of the pelvis.

The second form would begin by a chill, fever, abdominal pain, spontaneous or on pressure. Then signs of biliousness would supervene, great thirst, altered features, meteorism, typhoid condition, subdelirium, cold sweats, scanty and fetid lochiæ and often sudden death without agony. Here there existed lesions of the veins; the uterine sinuses, pampiniform plexus, and ovarian veins contained pus. It was a purulent infection due to uterine phlebitis. This phlebitis would often extend to neighbouring veins, and then would appear *phlegmasia alba dolens*, followed by obliteration of the vessels. This group had received the name of "*puerperal phlebitis.*"

Now, if we combine this latter form with the first, that is, given a puerperal phlebitis associated to peritoneal inflammation, if, besides, other symptoms of a more serious character supervene, such as sloughing of the genital parts, or especially hospital gangrene, then we would have the third form which was called, "*true puerperal fever*," offering the following symptoms : Fever, rapid pulse, cephalalgia, rigors, coated tongue, fetid lochiæ, meteorism, vomiting, extreme prostration. altered features, cerebral symptoms, ending in death with or without agony. In one word, it was purulent infection, complicated with peritonitis and gangrene.

As you see, gentlemen, the puerpera for the anatomist was but a patient bearing a large wound, and in their opinion, all the puerperal pathological phenomena were nothing else than mere complications originating on the site of placental insertion. Very well, but it was with regard to that uterine wound that there existed the missing link. What was the primordial cause which determined in that wound the modification from which all subsequent trouble would proceed ? The essentialists were keeping closer to the truth, and as early as 1718, Strother contended that puerperal fever was due to the etiological influence of atmospheric miasms causing the putridity of lochiæ. Nearer our time, Trousseau, in a flash of genius, expressed the idea that something specific was certainly added to the placental wound to produce the puerperal disturbance.

The modern doctrine, owing to the marvellous discoveries of the immortal Pasteur, has shed a light upon this chaos, and we are well

aware to-day that puerperal fever of former times is a more or less intense infection caused by the absorption through the genital organs of microscopical germs, These micro-organisms, by the mere fact of their presence, or the diffusion into the blood of their chemical products, poison the whole system, determining morbid phenomena, the form and gravity of which vary according to the nature of the pathogenic germs, their degree of virulence and the receptivity of the soil in which they develop.

I will spare you the chronological enumeration of the researches which, within the last twenty years, have permitted the numerous investigators to arrive at that conclusion. The important point for us to know is that it is to-day almost universally admitted that the principal factor which has to do with the origin of puerperal infection is the streptococcus pyogenes, the same organism which is found in erysipelas and phlegmonous abscesses. Generally it is it alone which is the cause of the whole trouble. Still, several observers have found it sometimes associated with the staphylococcus aureus, and even with the bacterium coli. In some cases, by exception, it proved to be absent and the staphylococcus or bacterium coli only were found, but then the puerperal disorders were greatly mitigated and spontaneously disappeared or yielded rapidly to ordinary therapeutic measures.

In almost every case these pathogenic germs come from without and are carried in, owing to insufficient antiseptic precautions before, during, or after labor. I say *almost* every case and not exclusively. I certainly do not believe in spontaneous generation, and puerperal septicæmia has always a heterogenous origin ; but it would be erroneous to think that it cannot occur outside of contamination produced by the negligence or ignorance of the *accoucheur* or some of the attendants. Numerous observations have shown the possibility of an apparent auto-infection on a puerpera who, before confinement, carried microbes, dormant as it were, and awaiting but fortuitous and favourable circumstances to awaken from their torpor. For instance, often the cervix is the seat of old endocervicitis where the staphylococcus, the gonococcus and even the streptococcus having lost the greater part of their virulence, have been residing as it were in a latent condition. And again, old purulent collections may at times exist in or about the appendages without manifesting their presence until the day when the obstetrical traumatism resuscitates their nefarious power. The genital system of the woman being then placed by parturition in a special state of receptivity, becomes a soil favourable to their growth and further invasion.

However, I repeat it, in most cases it is by germs brought from without that infection takes place, whether these micro-organisms have found their way into the genital organs more or less anteriorly to the confinement, or whether they have been carried in during or after labor by the hands or instruments of the *accoucheur*. As long as the mucous membrane preserves its integrity, their entrance into the system does not take place, but we must not forget that there always exists in the uterus after delivery a door wide open, the spot laid bare by the expulsion of the placenta. Besides, it is of very rare occurrence that the traumatism of labor does not produce in the cervix or along the vaginal tract an erosion, a minute abrasion, perhaps imperceptible, but still sufficient to permit septic absorption. And when once these germs have found an entrance into the tissues, is there anything astonishing in the fact that their symptomatic manifestations vary in their form as well as in their intensity without it being necessary to attribute the varied clinical phenomena which we observe to micro-organisms of different nature? The greater or less severity of the disease, and the various clinical forms it presents are due to numerous causes which are easily accounted for. The modern conception of the microbic etiology of puerperal sepsis has brought no change whatever to the admirable clinical picture left us by the old masters; moreover, it gives us a satisfactory explanation of all the different pathological symptoms observed during puerperium. Only, here as well as in all other surgical traumatism, certain complications have almost totally disappeared and belong hereafter to the domain of history. Thanks to the discovery of antisepsis, those epidemics of erysipelas, those everlasting suppurations, and also those putrid and so rapidly fatal forms of puerperal fever are but very exceptionally met with in the hospitals and even in private practice. Still puerperal infection, to-day as formerly, shows itself in different forms which, outside of the degree of virulence possessed by the infecting agent and the individual resistance of the constitution, depend upon the anatomical lesions of the various tissues which are affected. Thus, to-day as before we must expect a less alarming symptomatic manifestation where septic absorption exhausts itself in a mere inflammation of the pelvic cellular tissue than when the morbid process spreads to the appendages or to the peritoneum. Should the vascular system, which irrigates the uterus and the neighbouring territories, participate in the infection, the clinical picture will be more gloomy still and, lastly, if these varied pathological conditions are associated, if above all, gangrene of the tissues takes place, oh! then the reaction of the organism will show itself in a most solemn and desperate manner.

If I offer these few remarks to your consideration, gentlemen, it is because I want to refute beforehand the opinion of those who might be tempted to make of the severest forms of puerperal septicæmia a morbid entity entirely different from the other less intense puerperal manifestations, trying to explain it by the hypothetical existence of a special and still unknown micro-organism.

But enough on this subject. I long to arrive at what must particularly interest us, I mean the preventive and curative treatment of puerperal septicæmia.

I confess, gentlemen, that I am a most fervent believer in the germ theory, and that I am thoroughly convinced of the truth of its teachings. Must I add that I believe with all my soul in the justness of the modern etiological conceptions of puerperal septicæmia. I earnestly believe that in order to ward off this disease, we must behave during an obstetrical case as we do in presence of all other surgical acts. We are bound to take before, during, and after labor all the usual antiseptic precautions which are now universally known, having been so often and everywhere repeated. The lying-in-hospitals of the United States give us, with this respect, an example deserving our most careful meditation, and it suffices to have had the good fortune of visiting some of them to form an idea of the perfection with which the attendants of these model institutions carry out the principles of asepsis and antisepsis in which they have the most absolute faith. There, they proceed to the conduct of a midwifery case exactly as if it were a major gynæcological operation, and should they be suddenly compelled to open the abdomen during labor, they would be thoroughly prepared to do it, the parturient: the assistants, the nurses, the *accoucheur* having been entirely aseptisized beforehand.

It would be utterly ridiculous to pretend that we ought to surround ourselves with similar precautions in ordinary practice; this ideal condition can be realized but in institutions especially designed for obstetrical purposes, it is nevertheless our duty to try and do all we can with the means at our disposal. It is needless to speak of the preparation of the bed, the bedroom or the surroundings of the parturient; these things usually escape our control and generally cannot be put in the favourable condition required by all thoroughly aseptic confinement. But what we must never forget is to see, at least, that the external genital organs of the woman are carefully cleansed with soap and water and afterwards bathed with some antiseptic solution before any digital examination is made.

Is it necessary to use vaginal douches before labor?

We do it no more at the Ottawa Maternity Hospital, unless the

woman is affected with leucorrhœal discharge somewhat suspicious by its abundance or its character. Such has also been the practice of several *accoucheurs* who lately have thought best to discard those antepartum vaginal injections for the following reasons: They contend that physiological vaginal secretions possess germicidal properties upon the micro-organisms concealed in the vaginal tract. The douche in diluting and carrying away these salutary secretions would leave the soil unarmed against the microbes hereafter masters of the place. Kronig has lately published on that question very interesting statistics. He demonstrated that puerperal complications have been observed just as frequently on women who had received antepartum vaginal injections as on those who had not been previously douched. I will spare you the relation of his numerous experiments and you can take his opinion for what it is worth; however, according to his figures, it remains indisputable that these injections are, at the least, unnecessary when the vaginal cavity is in its physiological condition. Still, personally, I humbly confess that I fail to understand how antiseptic solutions, such as for instance a solution of corrosive sublimate, could possess a less valuable germicidal action than that attributed to the vaginal secretions which they expel in taking their place.

It is during or after labor that the puerpera is ordinarily infected, and in the majority of cases, contamination takes place through the ignorance of some of the attendants or the carelessness of the *accoucheur* himself. Labor, although a physiological phenomenon, must be likened to a real surgical traumatism; presently, there will be in the uterus a vast surgical wound near which none must approach without previously taking the most careful precautions.

No one, I am sure, would dream of attending or assisting at the opening of the abdominal cavity after having been in more or less immediate contact with an infectious disease, or even any case of suppuration whatever, and still less after taking part in a post-mortem examination. Still, how many physicians, overwrought by an extensive practice either forget or neglect to pay attention to these elementary notions! The mere mention of this fact is sufficient to indicate the duties we have to fulfil.

But aside from these general indications, I must particularly insist upon the necessity of submitting the hands which will be used in making vaginal examinations, to a careful cleansing and to a sterilization as complete as possible. How many errors are not daily committed in this respect, especially by those who, little conversant with the usual surgical practices, underrate the importance of watching over the faithful execution of the most minute details! The super-

ficial ablutions used by a certain number are entirely insufficient from an aseptic standpoint; the hands may be clean "socially speaking," but *surgically*, they are far from being pure. To dislodge the germs which have sought refuge in the folds of the epidermis and under the nails, more than a simple *lick and a promise* is required, and without conscientious and prolonged scrubbing, the action of the strongest antiseptics themselves is totally delusive, because these antiseptics cannot come in contact then with the germs protected by the layer of dust and grease in which they are imbedded. In order to show how this act of thoroughly disinfecting the hands, although apparently simple, is nevertheless difficult to realize, allow me to cite the experiments made by Fürbringer; they will permit your drawing the most practical and salutary conclusions.

The author begged several of his friends to sterilize their hands according to their own ordinary process and then to immerse them in a culture-medium.

Dr. A. —— Nails of medium length. Scrapes them with matches. Washes his hands in a 3 per cent. solution of carbolic acid and wipes them lightly. Scrapings from under the nails thrown in the culture-medium. Result, 3,000 colonies.

Dr. B. —— Nails longer. Cleanses them with a knife. Washes his hands with soap and water, scrubs them with sublimate solution and then wipes them. Duration of the process, 4 minutes. Result, 2,000 colonies.

Dr. C. —— Same method, but nails very short. Result, 2 colonies.

Fürbringer himself: Nails, medium length. Cleansed with a knife. Washing with soap and warm water during five minutes; washes them afterwards during the same length of time with sublimate solution. Result, culture medium remains sterile.

The left hand of another was simply washed with lukewarm water, then two fingers were washed with carbolized water, and the three others with a sublimate solution. Result, colonies appeared by hundreds.

The right hand was bathed one quarter of an hour in *warm water*, two fingers washed in carbolized water and the others in sublimate. Result, culture-medium sterile.

In comparing these various experiments, it is easy to see that the number of germs developed in the culture-medium increases with the length of the nails, and also, that the results obtained with antiseptics are not to be compared with the far better results obtained by vigorous scrubbing with soap and warm water.

May I now be allowed to suggest the following method of sterili-

zation which I have adopted, before beginning any surgical operation: 1. The nails are cut short and cleaned, when dry, with a knife or any instrument suitable for that purpose. 2. The hands are scrubbed during five minutes in warm water frequently changed and with Johnson's fluid soap, which, owing to the ether it contains, helps to dissolve the fatty substances which cover the epidermis and gather under the nails. 3. Hands then thoroughly bathed in alcohol and rinsed in sterilized water. 4. Finally, they are immersed for one minute in a solution of sublimate. I beg your pardon for laying so much stress upon these details, irksome as they may appear to some, but which I persist in considering of the greatest importance.

Now come the forceps, which may also claim a good deal of the many disorders observed during puerperium. I do not mean their awkward, irregular or untimely application; this goes without saying, but I allude to the lack of sterilization, which is a fault more frequently committed than some seem to think. Every time they have been used, these instruments should be disinfected by dipping them during at least five minutes in boiling water, wiping them dry with a sterilized towel, in which they remain wrapped up until the next application. It is an easy and simple thing to do and I generally employ, to this effect, one of those long and narrow vessels used for boiling fish and which are found in all hardware stores.

A third source of infection to which I want to call your attention is the retention of the secundines during the third stage of labor. I do not mean the retention of parts of the placenta itself, but some portion of the membranes which, remaining in the uterus, become infected and are often the starting point of septicæmia. I would not have made this observation, which may seem puerile, had I not witnessed this accident in the hands of even skilful physicians. The placenta is often suddenly expelled, by the pressure made upon the fundus of the uterus or merely by contractions of that organ. Thus, the secundines are turned up like an umbrella by the wind, becoming slender and very fragile. They may unexpectedly tear up, a considerable portion being left behind. This possible occurrence should always be carefully looked for, the placenta being grasped and twisted upon itself in order to make a rope of the membranes, which, without the least traction, will slowly slide down and out as if by their own weight.

I shall have ended what I intended to say about the prophylaxis of puerperal infection when I recommend you to supply the obstetrical bag with the following articles: A bottle of catgut, a few strands of silk-worm gut and needles for the repair of laceration, a few strips

of iodoform gauze, some creolin, Johnson's fluid soap, a nail brush and a small package of sterilized absorbent cotton. You may add, if you like, a Bozeman's sound or any other suitable instrument for intra-uterine douches; however, except when the hand has been introduced into the uterus, as in cases of turning or adherent placenta, I think it entirely unnecessary to make intra-uterine irrigation after labor. But what must be most carefully looked after is the laceration of the pelvic floor, however small the lesion may be. In every case, these doors opened to septic invasion must be closed as soon as possible. The repair of a cervical laceration is often difficult to perform for one who is not in the habit of doing this operation and who, therefore, is certainly excusable in not attempting what he is not in a position to do in a suitable manner, but the immediate suture of a vaginal or perineal laceration is within the reach of everybody: two or three silkworm gut sutures being all that is required, provided care is taken to protect the line of reunion with a small strip of iodoform gauze which shall be renewed every twenty-four hours for a few days.

In spite of all the precautions which may have been taken, and all the more if they should have been neglected, puerperal septicæmia will nevertheless occur and far too often, alas! I refer to your own experience. What shall we do then?

It is not my intention to dwell upon the symptoms and the course of all the complications which may happen during the puerperium, nor to study in detail the curative treatment which must be opposed to these various pathological conditions. I shall confine myself to mention briefly the symptoms which announce the commencement of the infection, indicating the chief therapeutic means whose efficacy we have been taught by experience, provided they have been employed in the very first days of the disease. We must remember, gentlemen, that is is of the utmost importance that we act promptly without the least delay at the very bursting out of the fire. In a few days it may be too late; the conflagration will have become beyond control, and we shall then only be reduced to a general save him who can. Therefore, I shall intentionally leave aside the varied symptomatic manifestations which may supervene later on, such as peritonitis, phlebitis, pelvic cellulitis, salpingitis, and so forth. Whatever may be the form assumed later on by puerperal sepsis, we must bear in mind that the etiological starting point is the same in every case, that is: absorption of pathogenic germs rendered possible by a solution of continuity in the genital system. The initial symptoms differ very little at the outset and it is then that we must act, that is, at the very moment when the infection, still localized, is within reach of the means

at our disposal to check it at its origin. We must, therefore, be vigilant and be able to recognize its earliest manifestations.

It is generally about the second or the third day after delivery that the declaration of war takes place, and the first gun-shot fired by the organism is the elevation of temperature. The enemy is still far, it is true, but woe to the one who, in every case of puerperium, does not listen with an attentive ear, in order to catch these still distant reports. Every night and morning after labor, temperature must be taken by the nurse or the physician himself, whatever may be the apparent good condition of the patient. Should the thermometer indicate the least increase in the temperature, then is the time to prepare our means of defence and try at once and discover the issues through which the foe may have penetrated. There is fever; be sure, then, that there is septic absorption somewhere. We must guard against any false security and I hope that no one among you is still believing in the old pretended milk-fever. At the most, might we be justified to a certain extent, in incriminating in some cases, stercoræmia due to a more or less prolonged constipation. At all events, it is but wise to prescribe some laxative which will soon clear the scene. In almost every case of elevation of temperature at the beginning of the puerperal state, septic absorption has taken place either in the placental wound or along the genital tract, through some abrasions which, owing to their exiguity, often escape the most careful search. As soon as fever appears, an antiseptic vaginal douche should be given, and if some suspicious spot is detected on the pelvic floor, it must be touched lightly with some caustic substance. I generally use in these cases a solution of one part of creosote in three of glycerine, leaving afterwards in contact with the mucous membrane, a strip of iodoform gauze; the next day, very often, the temperature falls to normal. But should we fail to discover anything suspicious along the vagina and should the temperature continue to rise, in spite of repeated antiseptic injections, then it is a sign that the enemy is proceeding forward more threateningly. To the artillery it has added its skirmishers and very soon its cavalry, followed later on by a few columns of infantry, and if we do not hasten to check its invading progress, we shall soon be overcome, and a pitched battle against its total forces means, for us, a sure defeat. Therefore, should frequency of the pulse increase while temperature continues to rise or even remains stationary, but above normal; if the conditions of the tongue indicate gastric disturbance, whether there has been a chill or not, and whether the abdomen shows or not distention or tenderness, let the lochiæ be sweet or fetid, we must at once use intra-uterine douches. But then we must bear in mind that the

instrument will get in contact with an open wound and the antiseptic precautions must be carried out with the utmost care. The hands are carefully disinfected, the vagina cleansed and irrigated with a creolin solution. A Bozeman's sound, thoroughly sterilized, is introduced in the uterus and the uterine cavity is washed out with creolin, taking care to hold the reservoir not more than one or two feet above the patient's bed.

If the next day the symptoms, instead of amending, become more intense, if the temperature keeps on increasing, reaching 100° or more, if the pulse beats 115, 120, whether the lochiæ are fœtid or not, the uterus must be curetted. But before proceeding any further, I want to say one word with regard to the fetor of lochiæ. It is an error to believe that the offensive character of puerperal vaginal discharge is an ordinary sign of septicæmia. It is erroneous also to attribute to this sign a gravity which it does not always possess, and finally more erroneous still to think that fetor of lochiæ is the exclusive indication of post-partum curettage. Many, many cases go through all their phases and even kill without the lochial discharge offering the least odor, and others terminate favorably and often without having presented any other symptoms than precisely that fetor, accompanied with some elevation of temperature and perhaps an increased frequency of the pulse. The offensive odor detected in the lochial discharge, especially during the first days of puerperium, generally means that saprophytes, that is, non-pathogenic germs, have found access into the uterus and produced the decomposition of the secretions or of some remnants of membranes forgotten after delivery. Of course this sapræmia must not be entirely trifled with, owing to the toxins produced and the fever which is the consequence of their presence in the system, and above all, on account of the favorable conditions impressed on the soil which then becomes more suitable to the growth of pathogenic germs which will engender septicæmia. Fetor of the lochiæ being a sign of the presence of decomposing material in the uterus, surely constitutes a new indication for curettage, but it is not a *sine quâ non* of the necessity of this operation, and whether it exists or not, and I insist upon that point, as soon as progressive or persistent elevation of temperature, acceleration of the pulse, gastric disturbance, cephalalgia, etc., etc., have convinced us of the beginning of septic absorption, if these symptoms have resisted the influence of intra-uterine irrigation, we must at once curette the uterus.

To do this operation in a satisfactory manner I prefer anæsthetizing the patient, although it is not absolutely necessary; Pinard, at the Baudelocque Clinique, never does it. He says that curetting can be

done with hardly any pain, if care be taken to hold horizontally and not vertically the vulsellum with which the uterus is drawn down, and always to avoid bringing the instrument in contact with the superior part of the vulva. He contends that the pressure of the vulsellum upon the soft and tender tissues situated at the entrance of the vagina is what generally causes the greatest pain. The hands are disinfected, the instruments sterilized, the vagina irrigated and finally an intra-uterine douche given. The curette must have a long handle and a large dull wire loop. The fundus is first carefully scraped, then the lateral, and at last the anterior and posterior walls of the uterus. Two dangers must be avoided in performing this operation, to go too far or not far enough. During puerperium, and after miscarriage, we must always bear in mind that the uterine tissue is exceedingly soft, and should the instrument be carelessly pushed forward with too much force, it is liable to perforate the organ. This has occurred already and the woman died. On the other hand, should we proceed with two great timidity, in the fear of perforating the uterine walls, the fundus may not be reached and the results will be null. I was inquiring one day from a physician if he thought it possible that, after an abortion or normal labor, a considerable portion of the placenta could be left behind in the uterus after curetting. He answered that a similar accident could not happen but in the hands of an operator who did not know his business. I believe he was entirely wrong and such a thing may occur even with a very attentive operator, if he is not aware of what is liable to happen in certain cases. After miscarriage or normal labor, the uterus sometimes is ante or retro-flected. A portion of the placenta may remain adherent to the fundus and shut out of the rest of the cavity by a kind of hour-glass contraction. The constricting ring constitutes a sort of resisting ceiling which the curette may scrape as well as the rest of the lower segment. The operator will not dare force a resistance into what he believes to be the fundus of the uterus itself, fearing to produce a perforation. I can assure you that, in some cases, this error is more easily committed than one would think, still, I fancy that it suffices to remember the possibility of its occurrence to avoid it. We must always carefully find our way with the uterine sound if necessary, the free hand being at the same time, applied to the fundus through the abdominal walls. After curetting, another intra-uterine irrigation is given to carry out the clots and debris which might have escaped the curette, and the whole cavity of the uterus is to be touched with an applicator wound with a piece of absorbent cotton soaked in a solution of creosote in glycerine. Finally a

strip of iodoform gauze is carried up to the fundus and removed the next day.

Now, I imagine that I hear buzzing around my ears a multitude of voices whispering: "But we have done all those things; we have given intra-uterine douches, we have curetted and still our patients became worse and worse and finally died."

I know it, and I think I can point out some of the causes of our failure. Let us examine our conscience. The majority of *accoucheurs* practice amidst what very frequently is far from being all that is desirable with regard to hygiene and perhaps cleanliness. Are we sure that some fault has not been committed against strict asepsis, before, during or after curetting? Have all the requisite antiseptic precautions been taken in the act of disinfecting our hands? After washing them, as I have seen it done, perhaps we have wiped them conscientiously with the towel, very clean I believe it, but still covered with germs, that was tendered us by some graceful attendant. And our instruments: the vulsellum, the sound, the curette, the nozzle of the irrigator, were they always properly sterilized? Have they not been, unknown to us, in contact with some non-sterilized objects before they were introduced in the genital tract? Have we not forgotten to give a vaginal douche before using the curette which then in traversing the vagina, may have carried into the uterus germs which had not yet found entrance to that organ?

How long after labor or the beginning of the symptoms have we made up our mind to act? Were we not too late, and when, therefore, the infection had ceased to be localized and had become systemic? And who knows, perhaps, did we perform curetting, trying to reach in the uterus, a foc which was encamped elsewhere, in a cervical or vaginal tear for instance.

At last, I grant that we have fulfilled all the conditions required by a thorough aseptic curetting, which besides was correctly indicated by the intra-uterine localization of the disease; we have not omitted the previous vaginal douches and uterine irrigation; the cavity has been carefully swabbed with creosote and glycerine, a strip of gauze has been left *in situ*. The next day fever persisted, or the temperature reached a higher degree still, all the symptoms seemed aggravated . . . . and we stopped there. It remains to say what I think we should do in this occurrence.

1. If, after curetting and removing the uterine gauze, the next day the temperature falls and the symptoms decrease, there is generally nothing else to do and the patient will soon get well.

2. Should fever persist and the other symptoms remain what they

were the day before, but without aggravation, then intra-uterine irrigation should be done twice daily with creoline, permanganate of potash, sublimate or carbolised water.

3. If the patient gets worse, if the temperature reaches the next day a higher degree than before the operation, then we must repeat the curetting.

4. At last, if after the second curetting the results are nil and the disease continues its course, it is useless to insist upon the local treatment; the infection, in spite of us, has spread abroad, the whole system is infected and the prognosis has become very gloomy indeed. We are reduced to the means at our disposal to relieve special symptoms, and above all, to strengthen the organism and help it to resist the general intoxication which we could not prevent. At the head of the list, I will mention alcohol, wine and other stimulants. Quinine may be administered by those who have confidence in it; ice-bags on the abdomen if the symptoms of peritonitis seem prominent; subcutaneous injections of normal salt solution which, of late, have appeared to possess an effect considered by some as almost specific; and, at last, hypodermic administration of Marmorek's antistreptococcic serum, which has just arrived full of the most brilliant promises.

Since the measures just enumerated are supposed to have some therapeutic influence upon confirmed systemic infection, why not, may I ask, gentlemen, why not use them all, simultaneously with the local treatment, at the very onset of the affection? Why not, after curretting, for instance, at once prescribe stimulants, tonics, quinine and, above all, antistreptococcic serum? I know perfectly well that there exists, as I have already said, infections where the staphylococcus and even bacterium coli commune are alone at the bottom of the trouble, but we know also that it is the streptococcus which is the etiological factor of the serious forms of puerperal septicæmia, and, every day, new observations are brought forward showing the beneficial influence of Marmorek's serum upon the infections produced by this microorganism. Why not, then, attack the enemy at the very moment when we suspect its presence and before its growth has caused irreparable disorders in the whole system?

Conclusion.

Puerperal septicæmia is an infectious disease caused by the absorption of pathogenic germs, usually the streptococcus pyogenes, rendered possible owing to a lesion produced along the genital tract by the obstetrical traumatism.

Almost in every case the germs come from without and are brought into the genital organs before, during or after labor.

Whatever may be the form and intensity of further clinical manifestations during puerperium, the disease is always localized at the beginning and liable to be reached and checked by surgical means at our disposal.

The first symptoms generally appear within thirty-six hours after labor and are characterized by elevation of temperature, frequency of pulse, cephalalgia, insomnia and more or less abdominal tenderness.

The preventive treatment consists in having the uterus thoroughly empty after delivery and in surrounding ourselves before, during and after labor with the most scrupulous antiseptic precautions, endeavoring also to detect and immediately treat the least solution of continuity along the vaginal tract.

The curative treatment must be employed at the very onset of the symptoms and consists in vaginal antiseptic douches, successively followed, should there be no improvement in the symptoms, by intrauterine irrigation and a single or repeated curettage of the uterus.

Later on, if we have been called too late or if we have neglected to intervene opportunely, curetting and antiseptic cleaning of the uterine cavity may be tried, but the results are then doubtful. Infection has become totally systemic, general therapeutic measures must be resorted to, the prominent indication being to sustain the organism in its struggle against the effects of septic absorption. The treatment which must be directed against the various complications arising then in the different organs, such as peritonitis, pelvic abcess, phlebitis, cellulitis, being usually the same as the treatment required by the surgical affections of these organs outside of puerperium.

A LECTURE ON THE CAUSES AND TREATMENT OF HARE-LIP DELIVERED IN THE POST-GRADUATE COURSE, JUNE, 1898.

BY

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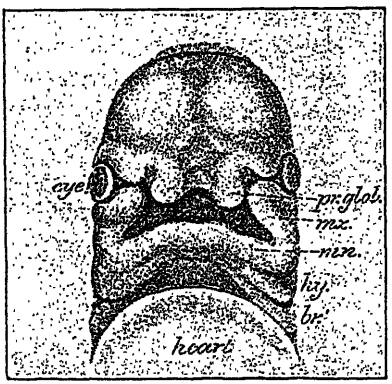
GENTLEMEN:—Before describing the treatment of the deformity which I am to speak of to-night, I should like to give you a short account of the development of the face, for all the congenital deformities met with are due to an arrest of this development at an early period of fœtal life. A study of embryology is of great assistance to the surgeon in enabling him to account for many of the congenital conditions he not infrequently meets with.



Pr. Glob.—Globular extremity of the mesial nasal process.
Mx.—Maxillary arch.
Mn.—Mandibular arch.
Hy.—Hyoidean arch.
Br.—First branchial arch.

Development.—At a very early period of fœtal life a series of clefts (branchial) appear on the side of the cephalic extremity, separated by rods of tissue called *branchial* or *gill arches*. The clefts communicate with the alimentary canal. The first branchial cleft is between the mandibular and hyoid arches. The mandibular arch which is afterwards developed largely into bone is divided into the superior and inferior maxillary portions. The two sides of the inferior maxillary portion early unite to form the lower jaw, but interposed between the two superior maxillary portions is the fronto-nasal pro-

cess. The space between the superior and inferior maxillary portions is called the buccal cleft. This is closed early except where the aperture remains for the mouth which is larger or smaller according as the cleft is more (macrostoma) or less (microstoma) closed. Sometimes the buccal cleft remains open from ear to ear. Now as to the nasal processes, these are divided into *mesial* and *lateral;* the mesial processes are united at their base by a depressed median part the *fronto-nasal process,* but below they are separated and each ends in a globular process (diagram and slides exhibited). These nasal processes, as development proceeds, extend backwards and along the embryonic roof of the mouth forming the nasal laminæ. Eventually, the nasal processes coalesce in the middle line and form the intermaxillary process and the middle part of the upper lip, the depression between forms the septum of the nose and by a coalescence of the nasal laminæ the rest of the nasal septum is formed. In rodents the notch between the globular processes persists and there is a fissure leading through the upper lip to the mouth Above the depression is a triangular space forming an angle with it, this forms the tip of the nose and the triangular surface above it, the bridge. The lateral nasal processes form the alæ nasi, these are not so prominent as the mesial. Between the lateral nasal processes and the maxillary process the lachrymal groove passes from the eye to the nose. Where the maxillary process of one side does not coalesce with the globular process then single hare-lip results, and if the union fails in the bony part as well, cleft palate is then seen. When both maxillary processes fail to unite with the globular processes, double hare-lip results, in this case the cleft usually goes through the line of union between the intermaxillary and superior maxillary bones. The middle part of the lip thus floats free and has attached to it the two intermaxillary bones, and is itself hanging from the septum of the nose. A failure of the two globular processes to unite is very rare though from time to time cases are reported. The mesial or septal part of the nose is developed from the junction of the globular processes. The septum is at first broad and depressed and the nostrils are widely separated, as seen in the lower races of mankind and monkeys.

The median union of the palate is completed about the 10th week of fœtal life and the globular processes unite with the maxillary also very early, the incisor foramen only remains to mark the junction of these structures. The fact that the arrest of union of these processes results in hare-lip and that the union takes place so early, rather discredits the many stories one hears of hare-lip and other deformities being produced by maternal impressions. In many cases the tenden-

cies to such deformities runs in families and it is not uncommon for two children in one family to suffer from hare-lip and cleft palate.

Hare-lip then is a congenital affection and often is due to heredity. There are various forms of this deformity :

1. Simplest, merely a notch in the red edge of the lip.
2. Through the soft parts only and not going through to the nostril.
3. The cleft through the lip and nostril and accompanied by cleft palate.
4. Double hare-lip, with a floating intermaxillary bone and cleft palate, occurs in $\frac{1}{10}$ of all cases.

There are other forms of deformity connected with arrest of development of face, such as enlargement of mouth, a persistence of the lachrymal groove, &c. (Slides shown of these deformities.)

Single hare-lip is usually on the left side, and is always to one side in the line of the junction of the intermaxillary with the maxillary bone. The child who suffers from this deformity, as a rule, cannot suck and has to be fed with a spoon. The mother's milk should be drawn and used as food for the child. Some advocate injecting the milk into the pharynx with a glass syringe, to which is attached a piece of rubber tubing. Sometimes a stoppered bottle with a large teat, having the aperature below, is useful. Rubbing the child with codliver oil or olive oil if it is puny, may help to keep it in condition until old enough for operation. Artificial foods should not be given unless under dire necessity. The child should be kept warm in flannel.

Best Age for Operation.—This depends on the condition of the child and the character of the deformity ; should the deformity involve only the soft parts and the child be healthy, operate at once for the mother's sake and in order that the child may suckle. In simple cases the earlier the operation the better. The only danger in early operations is from hæmorrhage, young children do not stand the loss of blood well. On the other hand they soon make up lost blood. Should the child be weakly, or the fissure be double and extend through the hard parts, then the operation ought to be postponed some weeks or even months. From six weeks to three months is probably the proper time for operating. I prefer the age of six weeks, this is well before dentition has commenced. Some advise waiting in the difficult cases until the child is weaned, but this is keeping a deformity before the family too long, and furthermore it renders the success of the operation more difficult.

Operation.—The number of operations devised for the relief of this deformity are many and varied. The ingenuity of surgeons is taxed

more by these plastic operations than by any others, and the number of methods is only equalled by the great variety of procedures advocated by the gynæcologist in sewing up the abdominal wound. Chloroform is the best anæsthetic in these cases. The child should be wrapped in a sheet or large towel, so that the arms may be confined, and then held in the arms of a strong nurse. A good light is essential. Sitting in front of the patient, the operator should first cut through the mucous membrane attaching the lip to the gum, and freely separate it so that the lips hang loosely ; the edges of the cleft are now freely pared by using a narrow-bladed knife and transfixing the edge of the cleft well up to the nostril, the flap is cut free above but below it is left on each side attached to the edge. As the two edges of the cleft are seldom the same length, one being usually distinctly longer than the other, on the longer side the soft parts should be more freely freshened ; both flaps should be cut as far as the red line of the lips. Some advise cutting the flap of the shorter side quite away and only leaving the long one, and then bringing this flap across the middle line to fill the deficiency of the shorter side. Any redundancy can be cut off without trouble. It is my custom not to separate the flaps from the edges of the cleft below until several sutures have been placed in the lip above and the fastened edges of the cleft accurately adjusted near the nose. Now the paring from the shorter side is cut away and more or less, as occasion requires, of the tissue at the red portion of the lip removed, the flap of the long side is brought over as before and adjusted as accurately as possible. By this means there is less hæmorrhage and no mistake of taking too much or too little away is made, Of course, during the operation an assistant compresses the sides of the cleft with his fingers, and thus loss of blood is prevented. Should any blood get into the mouth it must be at once removed with sponges on handles. Now as to sutures : formerly wire and hare-lip pins were always used. At present we employ nothing but silk-worm gut and horse hair. For years I have used nothing else and with the best results. Care should be taken not to go through the lip whilst suturing, but to dip down to the mucous membrane only ; the stitches should range on each side at least one-eighth of an inch from the edge. It has always been my custom, if the sutures have not been satisfactorily placed or seem to pull too much, or if perhaps there is a slight unevenness, to immediately take them out and re-introduce them. A little painstaking at this step of the operation is worth a good deal. After the main sutures of silkworm gut are placed, intermediate ones of horse hair may be employed, and afterwards the lip everted and the mucous membrane

sutured in the mouth, by this means the continuity of the surface is preserved and septic matter is prevented from entering the wound from the mouth. To recapitulate then. The most important points to be observed in the operation are :

1. Freeing the lip from the gum.
2. A free sacrifice of the edge of the cleft.
3. Accurate apposition of the parts.

The dressing should be simple. I usually apply an antiseptic paint (made of iodoform, resin, oil and alcohol) put on a piece of lint or cotton and nothing more. If the usual cheek straps are applied to preserve tension, they should be made of diachylon plaster, which is less irritating than the rubber adhesive, and the cheek parts cut broader than the part running across the lip, they should interlace in the middle line, the cheeks being well pulled forward. Before operation it is very important to know that the child has not been exposed to any fevers, as measles, or scarlatina. This is one cause of failure. Another is the inordinate crying of the child, and also the too early removal of the stitches. Sepsis, of course, is the great cause of failure and this is most likely to occur in badly nourished infants with poor resisting powers.

It is very important that sutures should not be removed too soon. In the old days of hare-lip pins they were removed in from 24 to 48 hours, because if left longer they would cut through the soft tissues of the infant's lip. Now we commonly leave silkworm gut in from 6 to 10 days. Should primary union not occur, wait until the inflammatory action has subsided and then freshen the edges and bring them together. Union now almost always occurs, because the parts have become, so to speak, immune. After the operation the child should be closely looked after. There is often great difficulty in breathing through the nostrils owing to tension on the upper lip and compression of the nostrils, and rubber tubes introduced are often a great aid and prevents collapse of the nostrils. After a time the parts get eased and the child will breathe easily through its nostrils.

The operation I have already described is that for single hare-lip. Double hare-lip is less common and must be somewhat differently dealt with.

Where there is no projecting intermaxillary process, the operation is not difficult, for then all the mucous membrane from the central portion is cut away and the flaps taken from the sides of the cleft as in single hare-lip. The central portion is sutured on each side to the lateral clefts and the lateral flaps run across to meet each other below the central portion, the lower part of which is freshened. What is in

excess is cut away. Sometimes the central portion may be cut into the shape of a V and the lateral flaps adjusted to it.

In those cases, however, where the intermaxillary bone projects the case is rendered much more difficult. In some cases, such as where the bone grows from the tip of the nose it must be sacrificed, but usually it can be broken back and forced into the cleft. Sometimes it is necessary to pare the edges of the gums, and I have been obliged in some cases to keep the bone in position with wire or silk sutures, It has been objected that the incisor teeth which belong to this premaxillary portion grow in crooked, if so they can be afterwards straightened by a dentist, or the teeth may be pulled out. It is also objected that the retention of the intermaxillary keeps open the palatal cleft. Always try and save the intermaxillary bone and so prevent a gap in the solid jaw. In cases where I have had to remove this bone, however, there was remarkably little deformity. Sometimes there is a double hare-lip and only a single cleft in the bone. In such cases the bony cleft of one side projects and has to be forced back with the thumb. In severe cases of operation in very weak infants where much paring has to be done, and the bleeding is excessive, the final stages of the operation may have to be postponed until recovery from shock takes place. In very young children bleeding is a factor which must be considered. (The different methods of operating were then described, such as Malgaigne's, Nelatons, Mirault's, Giralde's, Rose's and many others. All were illustrated by lantern slides.)

CLINICAL REMARKS ON THREE CASES OF DIFFERENT VARIETIES OF NUCLEAR PARALYSIS—BEING THE SUBSTANCE OF TWO CLINICAL LECTURES DELIVERED IN THE ROYAL VICTORIA HOSPITAL, MONTREAL, DURING DECEMBER, 1898.

BY

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CASE I.

Chronic Progressive Ophthalmoplegia Externa.

The patient before you entered the Ophthalmological Department some time ago, and through Dr. Buller's kindness he was transferred to the Medical Department.

He came complaining of double vision, which he says has troubled him at intervals for nearly three years. The intervals of freedom varied much in duration, from days to months. For some time he was annoyed with drooping of the right upper eyelid. He is a fairly well-developed, healthy looking man, 36 years of age, and is able to carry on his work, which is that of a machinist. Sixteen years ago he had a chancre which was followed by a rash over his body. He has used both alcohol and tobacco in excess. He makes no complaint of any pain in the head, nor does percussion over the scalp elicit any pain or tenderness.

With the exception of the ocular paralysis nothing abnormal is noticeable.

Dr. Buller found, when he first examined him some weeks ago, a nearly complete ophthalmoplegia externa. The inferior recti still retained some power, especially that of the right eyeball.

Vision of right eye $\frac{6}{12}$—L. $\frac{6}{6}$.

At present it will be noticed the paralytic state of the ocular muscles remains the same.

All the structures supplied by the 3rd nerve are paralyzed, except the inferior recti, the levator palpebræ, and the pupillary fibres. There is an appreciable difference between the right and left upper lids, the former presenting a slight droop, while there is complete power over the left lid. He is able to accommodate, but there is

diminution of the normal contraction to light. The external recti and the superior oblique are almost completely paralyzed.

The distribution of the paralysis renders it almost certain that the morbid process is situated in the nuclei of origin and not in the root fibres or in the nerves in their course. The involvement of certain parts of the 3rd, while others have escaped, cannot be readily explained in any other way.

The third nerve arises from a series of nuclei, the most anterior of which presides over the mechanism of accommodation, while behind this we have the nucleus which has to do with the contraction to light. The two nuclei are near and form what is called the anterior group of third nerve nuclei.

Whether there are separate nuclei for each external muscle supplied by the 3rd, is a question. Very exact descriptions of such are usually described in the text-books, since Kahler and Pick published their observations. We, however, have no anatomical proof that each muscle has its own definite nucleus. All that can be said is that there is a posterior group of nuclei belonging to the 3rd nerve, but in our present state of knowledge these cannot be arranged in a definite order. In close proximity to the posterior group of the 3rd nerve nuclei, we have the nucleus for the fourth nerve, and still further back the nuclei for the 6th nerve, both of which are here involved.

We have bilateral disease of the nuclei of the fourth, sixth, and those nuclei of the third which supply the external muscles of the eyeball, except the inferior recti and levator palpebræ of one side.

There are no symptoms pointing to any further cerebral changes. The bulbor nuclei are normal as far as can be made out. There are no symptoms of tabes or any other spinal affection. Nuclear ophthalmoplegia is sometimes a part of tabes and sometimes of general paralysis, but here it appears to be the only evidence of disease in the central nervous system. It is quite possible that tabes may eventually develop, but at present there is no evidence of a morbid process other than that situated in the third, fourth and sixth nerve nuclei.

It is instructive to observe how the degeneration has involved structures which have similar functions, although separated, while more contiguous tissues of a similar nature, but different in function, have escaped. The fifth and seventh nuclei, for instance are intact, while lying nearer the third and fourth nuclei than do the sixth nuclei.

What has been the exciting cause of this progressive degeneration

in the nerve nuclei? We have a history of both syphilis and alcoholism. It is recognized that alcoholism is an exciting cause of an acute form of nuclear-ophthalmoplegia, a very rare disease, which runs a rapid and usually a lethal course, but I am not aware of any evidence to show that it gives rise to a slowly progressive form of ophthalmoplegia. The after effects of the syphilitic poison is the greatest of all factors in bringing about chronic degeneration of nerve nuclei, an eminent example being the degeneration of tabes. It is reasonable to conclude that here we have to do with post-syphilitic degeneration.

Seeing that the condition has been in existence for three years the prospects of even a stay in the progressive character of the downward tendency are not bright. It is not likely that an anti-syphilitic course will help to check the advance of the degeneration, judging from the effect of similar treatment in tabetic degeneration.

For several weeks iodide of potassium has been given and it is the intention to continue its use for some time.

CASE II.

Chronic Bulbar Paralysis.

Miss E. B., aged 44, a seamstress, has had good health, with the exception of an attack of diphtheria at 15 and of acute rheumatism at 20. The latter did not involve the heart, otherwise there is nothing to note in her past or in her family history.

She dates the onset of her present troubles to Feby. 1895, when a few minutes after exposure to a cold wind, she felt a chilly sensation at the back of the head, which was at once followed by some difficulty in speaking.

The following day, she had less trouble in articulating, but a few days after there was return of the difficulty in speech and she now noticed for the first time a difficulty in swallowing both solids and liquids. Since the onset of the above symptoms, both the patient and her friends have noticed a great variability in their intensity, at times she appeared to articulate well and have but little trouble in swallowing, but on the whole there appears to be progressive tendency, although very irregular in its progression, to a more marked disability in both speaking and in swallowing. For upwards of two years she has been annoyed with saliva drooling over the lips.

Her speech has a nasal twang. The chief difficulty appears to be with the labials.

Her tongue is atrophied, and she is unable to protrude it but very slightly beyond the lips. It is the seat of fibrillary twitchings. She has not complete control over the muscles of the lips. She is for

instance unable to press them firmly together. The palatal muscles are weak, fluids are often returned through the nose, and there is a very marked difficulty in not only swallowing liquids, but also solids.

There is a bilateral paresis of the laryngeal abductors, not sufficient however, to give rise to stridor or other symptoms of laryngeal stenosis.

We have then in this case the following changes:

1. A marked paresis of the tongue with wasting of its muscular fibres shown by inability to protrude it, and the imperfect articulation of the lingual consonants.

2. A paresis and wasting of the orbicularis oris muscles, shown by the imperfect closure of the mouth, and the inability to correctly pronounce letters requiring the action of this muscle.

3. Paresis of the muscles of the palate resulting in regurgitation of fluids through the nose and nasal twang to speech.

4. Paresis of the muscles of the pharynx, causing a great difficulty in swallowing both liquids and solids.

5. A paresis of the abductors of the larynx, which has not as yet caused any distressing symptoms of laryngeal stenosis.

The following nerves are involved:

1. Part of the 7th.
2. The whole of the 12th.
3. Part of the 11th.
4. Parts of the 9th and the 10th.

That part of the 7th nerve which innervates the orbicularis is involved. Some observers contend that this muscle receives its supply from a nucleus immediately in the neighbourhood of the 12th nucleus, or even from the latter itself, but recent observations tend to show that the lower facial muscles receive their motor supply from the main facial nucleus, while the upper facial muscles are innervated from a nucleus situated higher up.

The laryngeal and palatal paralysis arise from an involvement of the bulbar part of the 11th nerve. While the pharyngeal difficulty is due to involvement of either the 9th or 10th, or both. It is not definitely known from which nerve the pharyngeal muscles receive their motor nerve supply.

We have to do here with a bulbar paralysis. No doubt symptoms of a more or less similar character may arise from a morbid process at the base of the brain, damaging the nerve roots as they emerge from the pons and medulla. The nerve roots of the 10th, 11th and 12th are very close together as they emerge from the brain, but a growth

in such a situation would almost certainly damage also other structures, thus giving rise to symptoms distinctive of the situation.

The accompanying diagram shows the chief nerve nuclei situated in the medulla and pons. It will be seen that there is a common nucleus for the 9th, 10th and 11th nerves, the nucleus for the latter being situated at the lowest part of this common centre. The nucleus for the 12th is close to and parallel with the 11th nucleus, while the 7th nucleus is only a little above the upper end of the 12th nucleus.

The symptoms are dependent on loss of function in parts of the 7th, 9th, 10th, 11th and in the whole of the 12th.

What has brought about this loss of function, in the bulbar nuclei?

I. By far the most common cause of chronic bulbar paralysis is a slowly progressive degeneration of the nuclei.

This is a well recognized disease met with most frequently after the age of forty. Little is known about the causes that give rise to it. It is not infrequently a part of a more diffuse but similar morbid process in the ventral cells of the spinal cord.

II. Bulbar paralysis may arise suddenly from plugging of the vessels that supply this part of the brain with blood. In this form we, however, rarely meet with softening confined wholly to definite nuclei. Other parts are generally robbed of their blood supply, so that additional symptoms to bulbar paralysis are found to be present.

III. A rare form of bulbar paralysis from acute inflammation of the bulbar nuclei.

IV. Cases of bulbar paralysis have been described where no lesion or change of any kind has been found after death. The disease has an intermittent character and is at first chiefly characterized by a marked weakness of certain muscles after exertion, but which disappears after rest. This disease known by the name of asthenic bulbar paralysis, soon involves other nuclei than those innervated from the pons and medulla.

V. We may have a disseminated sclerosis only involving the bulbar nuclei.

VI. Finally, pseudo-bulbar paralysis from bilateral lesions of the tracts between the cortex and nuclei.

It is difficult to arrange the case before us in any one of the above classes. We have to do with a condition setting in suddenly, irregularly, but still slowly progressive and with the intensity of the symptoms varying much from day to day. Were it not for the fact that there are no symptoms other than bulbar it would conform more closely to the fifth variety (a limited disseminated sclerosis) than to any of the other forms.

We may exclude without discussion, the 2nd (hæmorrhagic, embolic or thrombotic bulbar paralysis); the 3rd (acute bulbar myelitis); the 4th (asthenic bulbar paralysis); and also the 6th (pseudo-bulbar paralysis).

The history of a sudden onset with very slow progress are against its being of the first mentioned or progressive degenerative variety. It is just possible that the patient is mistaken as to the suddenness of onset. How frequently we meet with histories of sudden onset of disease, when we know that there must have been symptoms in existence for some time! It is the effects of the *last straw* that makes the impression on the patient's mind. She may have had slight difficulty in articulation without its being noticed by either herself or friends. The fact that the trouble has been slowly progressive certainly points to a degenerative lesion, but whether this is a pure atrophy of the nuclei or a sclerotic process, limited to the nuclei or their immediate neighbourhood I don't think we have sufficient evidence to determine. The diagnosis as to the nature of the bulbar paresis rests I believe between these two varieties of lesion. The course in the future will determine the nature of the morbid process.

CASE III.

Chronic Progressive Spinal Muscular Atrophy involving parts of both the Cervical and Lumbar Cords.

The man whose case we will now consider is 36 years of age and a weaver by occupation. Six years ago his left forearm was injured in machinery, and an abscess developed on the ulnar side of the forearm, about midway between the wrist and elbow joints, which was opened. In the course of two months the wound had healed, but it was noticed that there was wasting of the muscles of the hand. The wasting steadily increased for two years, but he thinks there has been but little increase during the past four years. Five years ago he found that the muscles of the left leg were weak, his toes catching any object above the usual level. A few months ago he first noticed the weakness and wasting of the small muscles of the right hand. Although there is marked wasting of the muscles of the right thigh, the patient was not aware of this until his attention was directed to it after his admission into hospital.

At 21 years of age, and again at 27 years, he had attacks of pulmonary hæmorrhage. One brother and four sisters died from consumption. There is no history of either syphilis or alcoholism. With the exception of the muscular wasting described, he does not present any evidence of disease.

Left Upper Extremity.—There is extreme wasting of the thenar and hypothenar eminences, of the interossei and lumbricales. The hand is a typical example of the "Claw Hand." The muscles on both surfaces of the forearm are much wasted as compared with those of the right forearm, the circumference of the left forearm, a few inches above the wrist, being an inch less than the corresponding part of the right forearm. There is no disturbance of any form of sensibility in the left hand and forearm. The reactions to the faradic and galvanic currents are nil in the wasted muscles. There is no wasting in the upper arm or shoulder muscles.

Right Upper Extremity.—There is wasting of the thenar eminences and of the first and second interossei. This was first noticed a few months ago. The wasting is progressive in character, but has not involved to any appreciable extent the muscles of the forearm. There is some wasting, however, in the upper arm, the difference in circumference between it and the corresponding part in the left upper arm being half an inch. The difference between the two upper arms is also distinctly to be made out on testing the power of the muscles. The faradic reaction of the muscles is lessened but very slightly. There is no disturbance of sensibility.

Left Lower Extremity.—The patient makes the complaint that when walking he is unable to lift the toes of the left foot as well as those of the right. This difficulty is very apparent when he is asked to walk across the room. There is slight wasting of the anterior group of muscles of the leg. There is no disturbance of sensibility.

Right Lower Extremity.—There is marked wasting of the muscles of the right thigh, the circumference at the middle being an inch and a quarter less than the circumference at the same part on the left side. There is a distinct diminution of both the faradic and galvanic reactions, but no objective or subjective sensory disturbance.

Reflexes.—The knee jerk cannot be brought out on the right side. It is, however, normal on the left. Ankle clonus is not noticeable in either limb, but a rectus clonus is present in the left limb. The superficial and organic reflexes are normal.

Fibrillary twitchings are to be seen in all the atrophied muscles, and also in those of the left thigh. There is no disturbance of co-ordination or special senses.

The condition present in this patient may be summarised as follows :

I. A progressive wasting of the muscles of the left hand and forearm extending over six years.

II. A slight wasting of the interossei and of the thenar and hypothenar groups of the right hand extending over a period of 6 months.

III. Wasting of the muscles of the anterior surface of the right thigh.

IV. Wasting of the muscles of the anterior surface of the left leg.

The state here differs considerably from the usual course met with in cases of progressive muscular atrophy.

1. In the prolonged course. The atrophy was marked in the left hand for some years, before there was any evidence of wasting elsewhere.

2. In the involvement of a limited but different set of muscles in each lower extremity. It is not usual for progressive muscular atrophy to involve the lumbar enlargement as is here present.

3. The different groups of muscles of the lower limbs which are the seat of the atrophy is likewise an unusual feature. The atrophy is generally symmetrical.

From the course and distribution of the atrophy and the entire absence of sensory disturbance we must conclude that the disease is spinal and not peripheral in origin. We have to do with a wasting of the anterior cornual cells in the lower cervical enlargement and at two different levels in the lumbar cord.

There is nothing that we can find in this man's past history that gives any clue as to a probable exciting cause, except the presence of an abscess in the ulnar border of the left forearm: shortly after its discharge, atrophy of the small muscles of the hand were noticed. Whether this is an accidental coincidence or not, it is impossible to say. A number of cases of spinal muscular atrophy have been reported following injury to a limb, the atrophy starting in the injured member and afterwards becoming general. It is not rare to meet with cases of other degenerative diseases of the central nervous system, where the starting point appears to have been a traumatism either general or local.

It would seem as if the nerve elements when injured suffer in their nutrition, and when once started, the degenerative process may extend far beyond the original influence exerted by the traumatism. There is no history of either alcoholism or syphilis. It is rare, I think, to meet with cases of spinal atrophy due to the syphilitic poison. Such cases do occur, but they are very rare as compared with the frequency with which the syphilitic poison attacks the sensory side (tabes) of the cord; rarer also than cases of ophthalmoplegia from syphilis.

We cannot tell why the poision of syphilis should select, as it were, more readily the nuclei of the ocular nerves, and rarely the bulbar and anterior cornual cells.

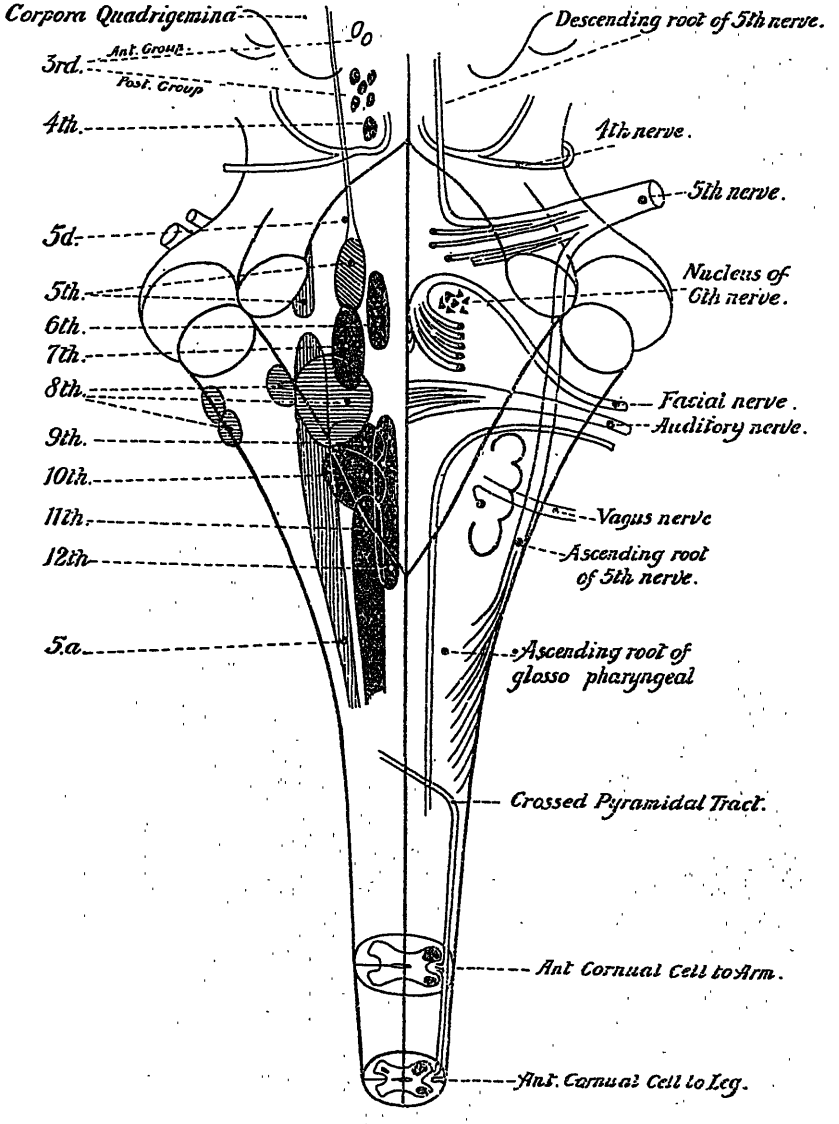
Corpora Quadrigemina
Ant. Group
3rd.
Post. Group
4th.
5d.
5th.
6th.
7th.
8th.
9th.
10th.
11th.
12th.
5.a
Descending root of 5th nerve.
4th nerve.
5th nerve.
Nucleus of 6th nerve.
Facial nerve.
Auditory nerve.
Vagus nerve
Ascending root of 5th nerve.
Ascending root of glosso pharyngeal
Crossed Pyramidal Tract.
Ant Cornual Cell to Arm.
Ant. Cornual Cell to Leg.

Diagram to illustrate:—

1. The relative positions of the 3rd, 4th and 6th nerve nuclei, the nuclei involved in the first case. The two groups of third nerve nuclei are shown, the posterior are deeply shaded to indicate their degeneration, while the anterior are shown in outline. The 4th and 6th nuclei are also shaded.

II. The various pontine and bulbar nuclei. The 7th, 9th, 10th, 11th and 12th are deeply shaded, to represent the supposed condition present in the second case (chronic bulbar paralysis). The whole of the main nucleus of the 7th nerve is shaded, which is not strictly correct. The same holds true with the 9th and 10th nuclei, which, like the 7th, can only be partially degenerated.

III. The course of the crossed pyramidal tract in the spinal cord and its relations to the anterior cornual cells in the cervical and lumbar enlargements. These cells are shaded to represent their degeneration, the condition of things as they are supposed to be in case No. III.

The nuclei are represented as if seen through transparent material.

Case Reports.

INTESTINAL OBSTRUCTION DUE TO BANDS, THE RESULT OF A FORMER APPENDECTOMY AND COMPLICATED BY A VOLVULUS OF THE MESENTERY.

BY

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Alex. M., æt. 27, was admitted into the Montreal General Hospital on May 9th, 1898, suffering from symptoms pointing to obstruction, as evidenced by vomiting, constipation, distension and tenderness of the abdomen.

Two days before he was suddenly seized with severe abdominal pain confined to a small area, two inches below the umbilicus. Soon after this he commenced vomiting and the vomiting has continued fairly constantly ever since. Purgatives of all kinds were administered, including castor oil and salts, without effect, so he was sent to the hospital. On admission he was found to have a subnormal temperature (97½°), a rapid, weak pulse (140), and a very anxious expression of countenance. His tongue was dry and brown ; he had contracted pupils. There was no rigidity of the abdomen, but it was somewhat distended and very tender below the umbilicus, and he complained of severe pain in this region. There had been no motion of the bowels or escape of flatus for two days. Although he was not vomiting he had a feeling of nausea. In the right iliac region was seen a scar due to an operation for appendicitis performed some three years before, in the centre of this was a small hernial protrusion. Liver and spleen dulness normal, and in the right flank a dull area was made out. Owing to this serious condition immediate operation was determined upon.

An incision, three inches long, was made in the median line below the umbilicus. On opening the peritoneum a quantity of reddish coloured serum escaped and distended dark coloured bowel presented at the opening. There were ecchymoses in spots. On introducing the fingers a band was felt in the right inguinal region near the site of the old wound, running to the mesentery and constricting the ileum. The band was divided between the ligatures ; this did not relieve the distension, and on further examination a large por-

tion of small intestine was seen hanging through another loop or band, but not acutely constricted by it. This was also divided, still there was no relief, the bowel seemed to be lifeless, and no peristalsis was present, so the incision was enlarged and a further examination revealed a twist to the right of the whole mesentery. There was now nothing for it but to turn ont the whole of the small intestines so as to untwist them. This was done, the intestines being covered by hot towels. They were then replaced, the wound sutured and the man returned to bed. I might mention that the wound was not completely closed, but iodoform gauze was introduced in various directions amongst the coils of intestines and a glass tube into the pelvis. The man rapidly improved and never had a bad symptom. The bowels moved next day and recovery was rapid.

In this case the volvulus was probably consequent on the constriction. These cases of intestinal obstruction are always a source of great anxiety to the surgeon and should be operated on as early as possible. The results from obstruction by bands, pressure of tumours, volvulus, &c., are not nearly as good as where the obstruction is due to some constriction within the lumen of the bowel or where resection is necessitated. Kocher has shown that any over distension of the bowel with gas or feces, produced by constriction and without any interference with the mesenteric circulation, will cause congestion of the bowel, blueness, and consequent venous stasis. Then follow ecchymoses in the mucous membrane, alteration and distension of epithelium leading to ulceration above the constriction. Now from this moment absorption of septic products commences from the fecal contents and your patient will die from general intoxication which will cause heart failure and collapse, or the bowel conditions may lead to necrosis and perforation.

Kocher advises in these cases an incision into the distended bowel and the washing out of as much as possible of its septic contents. This in a strong person will do no harm and perforations which are commencing will be detected, and if the person has a weak heart there is no better stimulant than removing the toxic products. Kocher advises emptying the bowel also in those cases where the patient's condition is such that a prolonged operation or resection would not be tolerated. In such cases washing out of the stomach should first be tried.

A CASE OF ABDOMINAL HYSTERECTOMY FOR FIBROID TUMOUR OF THE UTERUS AND DISEASE OF THE OVARIES AND TUBES—RECOVERY.[1]

BY

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This is a case of fibroid uterus with double hydrosalpinx which was removed successfully from a patient with the following history :

Mrs. P., 44, was admitted to the gynæcological ward of the Western Hospital, on the 12th September, 1898. She gave the following history : Menstruation had begun at the age of 13, and was normal until her marriage at the age of 24. She had seven children and five miscarriages, the last child four years ago. She enjoyed fairly good health until two years ago, since which time her periods have become very profuse, although regular and painless. About six months ago she noticed her abdomen enlarging, and on consulting her physician, the latter diagnosed uterine fibroid and sent her to me for operation.

She presented the appearance of anæmia from hæmorrhage, but all other organs, except the uterus, were fairly healthy. The urine was acid, s. g. 1020, and free from albumen and sugar. The uterus was very hard and uniformily enlarged to the size of a large cocoanut. The tubes were enlarged and contained fluid, and the ovaries could be felt but were adherent. Operation on the 17th September, 1898. An incision, five inches long, was made in the abdominal wall, through which the uterus was dragged, after separating dense adhesions, which bound down the ovaries and tubes. It was my intention to have left in one or both ovaries so as to prevent the artificial menopanse with its discomforts of hot flushes and chills, but on examination it was found impossible to do this without unduly risking the patient's life, and they were, therefore, removed. The method followed was to find and tie the two ovarian arteries individually and not *en masse*. Then the round ligaments were tied and a cut was made through the peritoneum across the front of the uterus from one round ligament to the other. The bladder was pushed away and a similar incision was made across the back of the umbilicus. It was then quite easy to

[1] Read before the Montreal Medico-Chirurgical Society, Nov. 6th, 1898.

push away the cellular tissue in the broad ligaments until the uterine artery could be felt when a ligature was placed around it, about half an inch from the uterus. As all the blood supply to the uterus was cut off, the uterus was removed at the level of the internal os without loosing an ounce of blood. The cervix was left in, but the anterior and posterior layers of the cervix were brought together with catgut, and then a running catgut suture, starting from the left ovarian pedicle right across the floor of the pelvis to the right ovarian pedicle, completely closed the opening in the peritoneum and covered over the cervical stump. The incision was closed with three layers of sutures; one of catgut for the peritoneum, one of buried silk-worm gut for the fascia and muscle, and a subcutaneous silk-worm gut for the skin. This method of suturing entirely does away with the risk of hernia and also with the unsightly scar left after the interrupted suture. The time occupied was nearly an hour.

The patient had very little pain, only requiring two one-quarter grain hypodermics of morphine and her temperature never rose over one-half a degree; she was up in three weeks and went home on the twenty-eighth day.

Examination of specimen.—On cutting through the anterior uterine wall to the depth of less than one-quarter of an inch, the hard round tumour is easily shelled out from its capsule, leaving a cavity, the walls of which quickly retracted so that it was difficult to put the tumour back again. The tubes are closed at both ends so that the secretion was retained sufficiently to distend the ovaries and buried in dense adhesions. The internal cavity is enlarged to about four times its normal area, but the tumour cavity does not communicate with it. It was placed in 2 per cent. formalin, which has preserved it perfectly.

Remarks.—If this had been a young woman, anxious to bear children, it would have been possible to have removed the tumour without removing the uterus, enough of the anterior wall only being cut out to enable us to obtain pressure on the tumour cavity. The tubes could have been dissected out and removed close to the cornua and their canal dilated and the ovaries could have been cleaned free from adhesions, but there was no object in doing this conservative work in a woman of 44, especially as it would have taken much more time and increased the risk of the operation. As regards time required, however, since my visit to Europe this summer this factor will not weigh so much in the future as it has in the past, as I saw some of the most successful men there take as much as a hundred minutes for an operation like this.

NOTES OF A FATAL CASE OF ACUTE ALCOHOLIC DELIRIUM WHERE THERE WAS AN ERUPTION OF ACUTE PEMPHIGUS.

(From the Medical Clinic of the Royal Victoria Hospital.)

BY

W. O. Rose, M.D.,

Senior House Physician to the Royal Victoria Hospital.

AND

D. McD. Robertson, M.D.

House Physician, Royal Victoria Hospital.

C. O'N., male, aged 31, was admitted to the Royal Victoria Hospital, Oct. 31st, 1898, complaining of swelling and pain in both legs, also of blisters on left forearm, right upper arm and both legs near the ankle. He gave a history of using alcohol, in moderation, till two years ago, but since then to excess. There is no previous history of delirium tremens. Had Pott's disease of the spine when a child.

Condition on entrance was that of a poorly developed and poorly nourished man, with marked right lateral scoliosis, and kyphosis, the sternum being pushed forward. He had not slept for some three days. There was a marked tremor all over the body, most marked in the hands.

Temp. 100°. Pulse 120. Resp. 24.

On both legs, about midway between ankle and knee, were excoriated areas about three inches in diameter, from which there was a slight serous exudation. These were preceded by blebs. A similar condition existed on right upper arm, and, on entrance, there was an unbroken bleb about three-fourths of an inch in diameter, full of a clear yellowish coloured fluid, on the left forearm near the wrist. The skin was warm and moist, with profuse perspiration.

Pulse was 120, fair tension, volume low. Heart dulness was not increased in size. At the apex both sounds were weak, the first being proportionately increased; at the base, sounds were normal.

Nothing abnormal was found in respiratory system, except the deformity noted above.

Tongue was coated, appetite was poor and had been so for two months. There has been no nausea or vomiting, except six months ago, when he had an attack of acute gastritis, lasting four or five days; some slight diarrhœa was present on entrance.

On admission he was quite rational, but the next day there developed a noisy, talkative delirium, with marked hallucinations, illusions and delusions. At times he had a few lucid intervals. The face became flushed, eyes bright; he could be roused from the delirium by direct questioning, but could not concentrate his thoughts for any time. All the muscles were in constant motion, tugging at the bed-clothes, trying to get out of bed, and throwing himself from side to side. The hands, tongue and lips were most markedly tremulous, while the head was retracted, the muscles of the neck being tense. The active motions were paroxysmal and accompanied by profuse perspiration.

Urine was largely passed involuntarily, and on examination was negative.

On Nov. 2nd and 3rd, the mental condition remained much the same, being somewhat more noisy. The pulse became weaker and albumen appeared in the urine. On the evening of Nov. 4th he became quieter, the pulse became rapid and weak, and there was a marked cyanosis of the extremities. About 2.15 a.m. of the 5th Nov. he began to have slight convulsions, lasting some 5 or 6 seconds, recurring every few minutes. At 2.25 a.m. he became comatose, the pulse became very weak, and he died at 2.45 a.m.

Autopsy.—Vessels of the pia all over were injected, without any evidence of meningitis. On section the brain showed acute hyperæmia.

There were old bilateral pleural adhesions, also of pleura to pericardium; partial collapse of both lobes of left lung, and acute œdema at extreme apex of right lung.

Commencing atheroma of aortic valves, and cloudy appearance of heart muscle, were noted.

Stomach was small, rugæ prominent, scattered petechiæ over mucous membrane, slight excess of mucus, but no marked gastritis. In duodenum, 1.5 cm. from pylorus, was a radiating scar of healed ulcer; duodenum was slightly congested in upper half; in jejunum some slight congestion of tips of rugæ; a more marked congestion of ileum, in some parts amounting to distinct hæmorrhagic effusion into mucous membrane.

Surface of liver was irregularly mottled with yellow; on section firm with congestion and fatty mottling.

Some old perisplenitis was present.

Left kidney was "hogbacked" with capsule slightly adherent; on section congested and firm. Right kidney on section showed uniformly dark color, the vessels of cortex and pyramids being distinctly injected as in opposite kidney.

RETROSPECT

OF

CURRENT LITERATURE.

Medicine.

UNDER THE CHARGE OF JAMES STEWART.

Acute Yellow Atrophy of the Liver.

DAVID ROBERTSON DOBIE. "Acute yellow atrophy of the liver."—*The Edinburgh Medical Journal*, December, 1898.

In a paper read before the Medico-Chirurgical Society of Edinburgh, Dr. Dobie reports a unique case, in which a diagnosis of acute yellow atrophy of the liver was made. The recovery of the patient throws some doubt upon the correctness of the diagnosis—thus running contrariwise to the teaching on the point of prognosis in such cases, and at the same time precluding the possibility of correcting or justifying the same by the evidence obtained at the autopsy table.

The patient, a farmer, aged 50 years, fell ill in such a way as to suggest something taken having disagreed with him, as headache and vomiting and abdominal tenderness were among the early manifestations of his illness. He became febrile and jaundiced. His urine was bile-stained albuminous, with blood in clots. Hæmorrhages from the throat, mouth and nose occurred and a profuse hæmorrhagic petechial eruption was observed over the forearms, upper part of the chest and later over the whole of the body.

He made a rapid recovery for from the onset of the symptoms until he sat in his chair beside his bed with brisk appetite, regular motions and with no albuminuria, was 31 days—2nd August to 2nd September. The yellowness persisted on the 18th of August. Two days previous to this date the petechiæ increased over the thorax and abdomen. The urine is reported to have contained leucin and tyrosin—and what is yet much more rare, cholesterin crystals in the presence of bile.

It is remarkable, too, to note that the percentage of urea was fully

up to the *normal*, 3 per cent. having been observed, Such a percentage of urea is regarded as stronger evidence against the diagnosis made in this case than the finding af leucin and tyrosin is in favour of such a condition.

We are not quite able to follow Dr. Dobie in his report from time to time upon the physical examination of the liver, for in the first record of his visit he says that after a careful examination of all the organs, he could not detect any abnormal condition in heart, liver, lungs or spleen. The following day the record shows no enlargement or diminution in the size of the liver. A little further on, under "remarks," he says, "in this case we had marked increase in the volume of the liver on the day I first saw the patient, and I am sure from the history of the great discomfort and rapidity of breathing during the onset that there had been tumefaction to a great extent." . . . the liver dulness had disappeared entirely.

On subsequent examinations this organ was found to be gradually regaining size. Thus in this brief space of time, from 5th to 13th of August, enlargement, diminution beyond detection of areas and then reappearance of liver dulness are recorded. It is not easy to see how this argues acute yellow atrophy of this organ.

Dr. Dobie strives to draw several lessons from this very interesting case :

1. That the jaundice in this case is of extra hepatic origin, *i.e.*, "the symptoms were due to suppression of the secretion of the bile" which "like many other excretions in a normal state is found in the blood and eliminated at the liver, therefore accumulating within the vascular system, when its separation at the liver had been rendered impossible and therefore temporarily suspended."

2. That jaundice may be induced by a very high and long continued high atmospheric temperature.

3. That acute yellow atrophy is not a primary liver affection.

4. That it admits of cure by a very simple remedy, the oil of turpentine.

The question naturally arises in ones mind—In what state are those liver cells now ?

The case corresponds to many cases recently reported under the terms purpura or purpura hæmorrhagica, or purpura with visceral complications. So little is known of the cause of purpura and the associated organism, that this case may possibly belong to the category of obscure purpuric cases.

Mixed Infection in Tuberculosis of the Lungs.

J. A. SHABAD. "Mischinfection bei Lungentuberculose."—*Zeitschrift für Klinische Medicin*, 1897.

During the last decade the subject of mixed infection in various diseases has been attracting much attention.

A secondary infection in pulmonary tuberculosis has been regarded by many observers as accounting for the so-called septic or hectic type of temperature, so common in the final stages of the disease, and as accounting also for the rapid destruction of lung tissue observed in the more acute infections. Dr. Shabad working in Obuchow Hospital, St. Petersburg, under the direction of Professor M. A. Afanassiew, examined thirty-one cases of pulmonary tuberculosis with a view of investigating the subject of mixed infection in this disease. In his method of examination of the sputum, all possible error was excluded; in several instances the blood was examined and the body also examined after death. He divides the cases observed into two groups.

1. Those examined during life and post-mortem.
2. Those examined only during life.

Seventeen cases comprised the first group. Of these, sixteen were cases of mixed infection, while one was uncomplicated tuberculosis. There were thirteen cases of simple mixed infection, twelve of these showing the pyogenic streptococcus and one the micrococcus tetragenus. Of double mixed infection there were three cases, two with streptococcus and pneumococcus, and one with streptococcus and staphylococcus. Twenty-one deaths occurred in the thirty-one cases; seventeen bodies were examined bacteriologically.

Concerning the cause of secondary infection in tuberculosis of the lung, Dr. Shabad remarks that a lung involved in a tuberculous process affords a favourable ground for the development of streptococci and a less favourable for the development of other micro-organisms.

From an analysis of the literature and his observations upon thirty-one cases of tuberculosis Dr. Shabad deduces the following conclusions:

1. Cases of mixed infection of pulmonary tuberculosis are those in which the accompanying organism may be found either in the lung-tissue or in the blood.

2. The finding of a pathogenic micro-organism in the sputum according to the method of Kitasato is not sufficient to establish mixed infection, for experience teaches that the micro-organisms observed in this way do not all come from the lungs. To this the genuine pyogenic streptococcus forms an exception.

3. It is very important not to confound the true pyogenic strepto-

coccus with the so called streptococcus of the mucous membrane, which is morphologically similar, but is distinguished by its biological peculiarities and its lack of pathogenity. While the streptococcus of the mucous membrane plays the part of a harmless parasite. the true pyogenic streptococcus has an important prognostic significance. The streptococcus mixed infection is found in a large majority of cases (15 out of 16 fatal cases.)

4. Less frequently than the infection from streptococcus, one meets the infection of tuberculosis of the lungs with the micrococcus tetragenus and pneumococcus and the double mixed infection with streptococcus and staphylococcus or with streptococcus and pneumococcus.

5. The secondary infection complicates mostly the last stages of tuberculosis of the lungs and leads rapidly to a lethal ending. It is so frequently met that almost all cases of tuberculosis of the lungs which come up for autopsy present the manifestation of mixed infection.

6. The importance of secondary infection in pathological processes consists in the fact that together with the tubercule bacillus it takes part in the etiology of tuberculosis of the lungs, or when it has not resulted in exudation it has at least participated in influencing the general condition of the patient and the fever through the toxines produced and also in the destruction of lung tissue—the formation of cavities.

7. There are undoubtedly cases of tuberculosis of the lungs which run that course with every appearance of the hectic state, and end fatally without the participation of other micro-organisms besides the bacillus of tuberculosis.

8. For the progressive, uncomplicated pulmonary tuberculosis the reverse type of hectic fever is characteristic. In mixed infection with streptococcus, typical streptococcus temperature curve was seldom observed. In most cases the temperature was remittent or approached the constant or continued type.

9. The normal temperature characterizes the stationary uncomplicated cases. Mixed infection with normal temperature is unlikely.

Tubercular Neuritis.

ALEXANDER JAMES, M.D., F.R.C.P. "Tubercular Neuritis." *The Scottish Medical and Surgical Journal*, Dec. 1898.

In this paper, read before the Edinburgh Medico-Chirurgical Society Dr. James reviewed five cases of tuberculosis in which tubercular neuritis of one form or another was present. By tubercular neuritis, Dr. James desires to be understood—"a disturbance in the nutritive

condition, not of the peripheral nerves only, but of any part of the nervous tissue as the result of the circulation through it of a toxin developed by tubercular disease."

The first case quoted is an example of what Dr. James regards as the commonest form of neuritis in which sensory, motor, reflex and trophic disturbances existed. The tubercular process was well advanced in the lungs, the nervous phenomena were manifest in impaired sensibility of the skin over both legs and feet, impaired motor power, especially in the left side, muscular wasting in the legs, and muscular tenderness. As the case advanced foot-drop occurred, power of extension disappeared, and cramps in the muscles of the calves developed. The reflexes, plantar, achilles and patellar, disappeared. The patient died, the condition of the legs and feet remaining unchanged up to death.

The second case is quoted as illustrating: 1. That the sensory tract may be the more affected. 2. The tendency of symptoms to disappear and reappear.

The third case shows that motion was mainly affected. Loss of power was complained of in the left arm, the side upon which the pulmonary lesion was observed. The integumentary system was practically normal, the left radio-carpal joint was involved, evidently with tubercular disease. The whole arm was smaller than its fellow, and the supra- and infra-spinati and the rhomboid were considerably atrophied. Muscular tenderness existed over the forearm and slightly increased irritability to the faradic current. The power to flex and extend at the elbow joint and to raise the arm from the side was gone. After treatment with a splint considerable power was regained, and muscular tenderness disappeared.

The fourth case was that of a boy who came complaining of soreness of the left foot, and weakness of the left leg, which came on rather suddenly about five months before his admission to the infirmary. On awakening one morning, at that time, he found the foot swollen and painful to walk upon, and although the swelling went down under treatment, the foot remained extremely tender. On admission there was found slight thickening over the inner side of the left tarsus. The pulse and temperature were normal, no signs of pulmonary disease. The left thigh and leg were considerably smaller than the right. After treatment in bed for some time slight, if indeed any, improvement was observed and amputation was performed with the result that the reflex irritability of the legs became less and less marked. "Six months after the amputation," Dr. James remarks, "the knee and ankle clonus could no longer be elicited on the right limb, although

the knee and ankle jerks were still increased. One year afterwards all that remained was a slightly increased knee-jerk in the left limb."

The disturbed condition of the lumbar cord in Dr. James' opinion was due to a combination of the toxin with peripheral irritation, while the extremely irritabl econdition of both lower limbs, and the wasting of the muscles of the left lower limb show that there is little doubt that the condition of the cord itself must have been a morbid one.

"Causeless hemiplegia" is grouped under the head of tubercular neuritis. Often post-mortem examination on hemiplegic cases reveals no cause for the paralysis and the supposition of the presence of œdema or some vaso-motor change explains the occurrence. Dr. James prefers to regard such cases as the result of the action of a tubercular toxin, manifesting itself in connection with a certain tract of the cerebro-spinal axis. Convulsive attacks and temporary paralysis in tubercular meningitis are probably largely due to the toxin.

The fifth case illustrates that class of cases in which tubercle is present in the lungs. It terminated suddenly with severe brain symptoms, convulsions and coma. Post-mortem examination of the brain revealed nothing beyond congestion, œdema and slight subarachnoid hæmorrhages on vertex of the left side. Dr. James thinks we are entitled to look upon this case as very probably an example of general nerve tissue-poisoning by the tubercle toxin. He remarks, further, that in tubercular disease we are very likely to have toxic effects from other toxins than the tubercle.

Relapse in Scarlatina.

RICHARD H. KENNAN, M.D. "Note on relapse in scarlatina."—*The Dublin Journal of Medical Science*, December, 1898.

This note containing evidence upon the rare occurrence of relapse in scarlatina has its foundation in observations made by Drs. Caiger and Kennan.

Dr. Caiger after eliminating such cases where any doubt surrounded the diagnosis of the first attack, as well as those of abortive second attacks occurring late in the period of convalescence, found in a large statistical account a true relapse or early second attack in .5 per cent. of all cases admitted to hospital. He further stated that the severity of the second attack was inversely proportioned to that of the first.

Dr. Kennan saw three undoubted cases showing relapse, while Dr. MacDowell Cosgrave is quoted as having reported upon two such cases.

W. F. Hamilton.

Hemiplegia following Diphtheria.

SLAWYK. "Zur Caseristik der halbseitigen Lähmungen nach Diphtherie," Von Dr. Slawyk (Asst. in Prof. Heubner's Klinik, University of Berlin).—*Charité Annalen*, 23.

In the above article, Dr. Slawyk gives an interesting account of three cases of hemiplegia following diphtheria which occurred in Prof. Heubner's Klinik. Although peripheral palsies are common after diphtheria, hemiplegia from cerebral changes is one of the rarest of events in the diphtheria of childhood.

Henoch, in the latest edition of his work on the diseases of children, says that he has only seen four cases of hemiplegia following diphtheria, and Baginski only saw three in 3,000 cases. Bokai, in a very extensive field in the children's hospital in Budapest, saw only five cases during nineteen years.

The three cases observed by Slawyk occurred in children during convalescence from severe attacks of diphtheria. In every case there were marked signs of cardiac weakness, and also of nephritis. In two cases there was paralysis of the palate. The hemiplegia set in on the 22nd, 19th and 18th days respectively, after the beginning of the diphtheria. Restlessness, headache and elevation of temperature accompanied the onset of the paralysis. Delirium, loss of consciousness or convulsions was not present in any of the cases.

The cause of the hemiplegia, which was left-sided in all three, was found to be embolism of the arteriæ fossæ sylvii in two cases, and a large thrombus in the right internal carotid where it gives off the sylvian artery in the third case.

In all three cases, the hemiplegia was complete, involving the face as well as the extremities. In the treatment antitoxin serum was employed on the 5th, 7th and 8th days respectively, but it apparently had no influence in preventing the severe affection of both heart and kidneys which was present.

Slawyk has been able to find an account of 50 cases of hemiplegia following diphtheria in medical literature. In every instance were reference is made to it in the reports which he consulted, the paralysis set in during convalescence. Of the 50 cases, 32 recovered, 14 died, and in 4 no statement is made as to the final issue of the disease.

Complete recovery was not the rule, contractures, hemichorea, athetosis and idiocy being met with in the majority of the 32 who escaped with their lives.

Post-mortems were performed in 13 of the 14 fatal cases, when it was found that the cause of death was : Hæmorrhage (Mendel's case),

1; thrombosis, 1; embolism, 9; embolism and thrombosis, 1; sclerotic atrophy of a hemisphere (Baginski), 1.

The arteriæ fossæ sylvii was the seat of the cause 11 times, and the internal carotid, where it gives off the sylvian artery, once. In one case, in addition to the hemplegia there was embolism of the central artery of the retina. The great cause of the embolism is the heart weakness, and it seems that in the majority of cases, no endocardial changes are met with, but often myocardial which may be either interstitial or parenchymatous. *J. Stewart.*

Surgery.

UNDER THE CHARGE OF GEORGE E. ARMSTRONG.

Complete Gastrectomy.

SCHLATTER. "Complete gastrectomy."—*New York Medical Record*, December 25th, 1897.

RICHARDSON. "Complete gastrectomy."—*Boston Medical and Surgical Journal*, October 20th, 1898.

Although cases of so-called total extirpation of the stomach have been reported from time to time, since Langenbuch reported his two cases in 1894, yet a critical examination of the reports of these cases renders it probable that in all of them a small portion was left behind. Schlatter claims that his case, operated upon on September 6th, 1897, is really the first instance of the stomach being completely removed in the human being. His claim to have done a total extirpation is confirmed by the pathological report of Professor Ribbert, of the University of Zurich. Ribbert, who examined the stomach after its removal says that one end is unmistakably œsophageal in histological structure, and that the other end is unmistakably duodenal. It has been known for some time that dogs are able to survive the artificial obliteration of the stomach and the loss of its functions. The so-called "Czerny dog" survived the operation of gastric excision for five years and was then killed for purposes of anatomical study.

Schlatter's patient was a woman, fifty-six years of age, a silk weaver, who claimed that in her family cancer was hereditary.

Richardson's patient was also a woman, fifty-three years of age. The operation was performed on May 31st, 1898.

MacDonald and Brigham, of San Francisco have each performed total extirpation of the stomach.

Schlatter after removing the stomach was unable to approximate the duodenum to the œsophagus without undue tension and therefore closed the duodenal end and united with the œsophagus a loop of small intestine fifteen inches from the duodenal-jejunal fold which he brought up over the transverse colon. Richardson, however, by tying and cutting the restraining attachments, succeeded in approximating the cut end of the duodenum to the end of the œsophagus without dangerous tension. The abdomen was closed without drainage. This

woman three months after operation was going about the ward, in good health. She had a clean tongue, absence of all fœtor ex ore, a natural stool once in twenty-four hours, no pain, and had gained 2000 grams in weight. Richardson's patient had some trouble with sutures. After the wound had closed it reopened and several sutures came away with some pus, but on October 16th, 1898, three and one-half months after operation she was well and had gained a great deal in strength, but not in weight.

An important anatomical fact determined by these operations and confirmed by the examination of the cadaver is that the œsophagus may be drawn down considerably below the diaphragm, thus permitting the division being made quite beyond the limits of the stomach.

These cases raise a number of interesting questions concerning the obliteration of all gastric functions, It was found necessary to administer food in minute quantities. A quantity approaching ten ounces seemed to excite vomiting some weeks after operation. At regular intervals of two to three hours, in Schlatter's case, milk, eggs, thin gruel or pap, tea, meat, rolls, butter and Malaga wine were given. The daily quantity amounted to one quart of milk, two eggs, two or three ounces of pap or gruel, seven ounces of meat, seven ounces of oatmeal or barley water, one cup of tea, two rolls and half an ounce of butter. As was expected, for theoretical reasons, the addition of hydrochloric acid and pepsin was found valueless. The alkaline fluid of the intestine at once neutralized the acid and rendered the pepsin inert. Nevertheless proteids were readily assimilated in the intestinal tract.

The urine remained normal except in the diminished quantity of chloride of sodium. But this woman, asked to have her food prepared with less salt than that of the other ward patients.

The fæces were well formed, of normal consistency, and light yellow in colour. The microscope showed large numbers of fat globules and fatty crystals, some undigested vegetable fibres, but no undigested animal fibres or connective tissue. Large quantities of triple phosphates were observed. The number of micro-organisms was normal.

How can a person vomit without a stomach? This woman, under observation had repeated attacks of ordinary nausea, retching and vomiting, ejecting as much as thirty ounces at one time. It may be that the duodenum became distended into a sort of compensatory receptacle for food.

Dr. Wendt thinks the following conclusions are justifiable:

1. The human stomach is not a vital organ.

2. The digestive capacity of the human stomach has been considerably overrated.

3. The fluids and solids constituting an ordinary mixed diet are capable of complete digestion and assimilation without the aid of the human stomach.

4. A gain in the weight of the body may take place in spite of the total absence of gastric activity.

5. Typical vomiting may occur without a stomach.

6. The general health of a patient need not immediately deteriorate on account of the removal of the stomach.

7. The most important office of the human stomach is to act as a reservoir for the reception, preliminary preparation and propulsion of food and fluids. It also fulfils a useful purpose in regulating the temperature of swallowed solids and liquids.

8. The chemical functions of the human stomach may be completely and satisfactorily performed by other divisions of the alimentary canal.

9. Gastric juice is hostile to the development of many micro-organisms.

10. The free acid of normal gastric secretions has no power to arrest putrefactive changes in the intestinal tract. Its antiseptic and bactericidal potency has been overestimated.

Thyroidectomy.

KOCHER. "Thyroidectomy."—*Correspondenz-Blatt für Schweizer Aeoet*, Sept. 15th.

Prof. Kocher reports an additional 600 cases of thyroidectomy, bringing his total up to 1,600. All these cases with the exception of 150 of the last 600 were operated upon by Prof. Kocher himself. When it is known that only about 10 per cent. of the cases of enlarged thyroid treated at the Berne Clinic are operated upon, some idea is obtained of the enormous field for observation of this condition. The remainder, 90 per cent. of the cases are treated medically. The medical treatment consists in the administration of thyroid extract and preparations of iodine. The latter acts as efficiently as the former. Cases of solid tumour that do not improve under medical treatment, cases of cyst formations, cases in which there is embarrassment of respiration, and all cases in which there is a suspicion of malignancy are submitted to operation.

A curved incision with the convexity downward is preferred as leaving a less conspicuous scar. The muscles are separated and not

divided. Prof. Kocher now operates chiefly with local anæsthesia using a 1 per cent. solution of cocaine.

G. E. Armstrong.

Operative Wounds of the Thoracic Duct.

CUSHING, H. W. "Operative wounds of the thoracic duct."—*Annals of Surgery*, June, 1898.

Wounds of the thoracic duct are usually considered to be invariably fatal. Dr. Cushing reports two cases which have been successfully dealt with in the Johns Hopkins Hospital. The first case (Professor Halsted's) was one in which an extensive dissection of the glands of the axilla and supra-clavicular triangle was carried out during the removal of a scirrhus of the breast. The wound of the duct was not recognised at the time of operation, nor was any evidence of the injury observed for ten days, when, upon the wound being dressed for the second time, a slight fulness was noted in the supra-clavicular region. The cicatrix was opened allowing the escape of four or five ounces of milky fluid. The wound was packed with gauze, and after discharging profusely for eight days the flow ceased and the patient made a good recovery. The second case (Dr. Cushing's) also occurred during the removal of cancerous glands in the neck. The dissection had been carried back to the brachial plexus, when a small, thin walled vessel, the size of a straw, was seen upon the tissues being relaxed. A clear, serous fluid welled from a small wound in it about one centimetre above the subclavian vein. A fine black silk suture was passed which inverted the edges of the wound and controlled the flow. The patient made an uninterrupted recovery.

Seven other authentic cases of operative wound of the duct are recorded. In one of these, death occurred from shock within a few hours of operation. In the remaining six cases, recovery ensued; in two of these the wound was packed with gauze; in one, a small vessel was found and clamped; in one, the point of evacuation of the fluid was clamped for three days; and in the remaining two, the vessel was sutured. The condition was recognized in all these cases by the discharge of a more or less milky fluid. In one case the amount lost was estimated at three pints per diem.

E. J. Semple.

Obstetrics and Diseases of Infants.

Macerated Fœtus in Latter Half of Pregnancy.

NAU. "Macerated Fœtus in Latter Half of Pregnancy."—*Centralb. f. Gynak*, No. 25, 1898. (*B. M. Jour. Epitome*, Nov. 19, 1898.)

Nau collected 61 cases from one hospital in Marburg. In 28 per cent. there was absence of all symptoms raising suspicion of fœtal death on the part of the mother herself. In about 50 per cent. nothing more was noted than distinct cessation of fœtal movements; in 19.67 per cent. other symptoms were said to be present; but Nau urges the possibility that they were often suggested by leading questions. In the most nearly verified cases Klein and Baumeister were of the opinion that the constitutional disturbance was due to absorbtion of toxins due to maceration of the fœtus without the action of bacteria.

The dead fœtus was usually expelled in the eighth month. The average term of retention of the fœtus in the uterus after its death was fourteen days.

The labor was of average duration, but in 47.37 per cent. of the cases premature rupture of the membranes occurred.

In six cases only was any obstetrical operation required.

The placenta separated normally in all cases.

Forty-two out of the 61 mothers had no fever in child-bed, and in only three of the 19 temperature cases was the fever evidently due to the passage of a dead fœtus out of the genital canal.

Treatment of Abortion by Abdomino-Vaginal Expression of the Uterus.

BUDIN, P. "Treatment of Abortion by Abdomino-Vaginal Expression of the Uterus. *Progrès Med.*, Sept. 17th, 1898. (*B. M. J. Epitome*, p. 346, 1898.)

P. Budin records three cases of abortion treated by digital curettage and abdomino-vaginal expression of the uterus.

He considers that the curette and all forms of abortion forceps are dangerous.

The following procedure is then recommended: After chloroform

has been administered and the vagina and external genitals rendered aseptic, the cervix is manually dilated. Occasionally it may be necessary to use Hagar's dilatars.

The uterus is then fixed with one hand acting through the abdominal wall, and one or two fingers, according to the age of the pregnancy, inserted and the whole interior of the organ scraped out, care being taken to loosen all parts of the ovum. If traction then fails to remove the fragments, uterine expression may be resorted to, as follows: Two fingers are placed in the posterior vaginal fornix and pressed forwards, while the external hand makes firm pressure on the fundus and anterior wall of the uterus.

In this way the organ is compressed between the two hands and the contents are forced into the vagina.

The cavity of the uturus is then washed out with some antiseptic solution at a temperature of 110-120° F. If the organ fails to retract and hæmorrhage occurs, the cavity may be plugged with iodoform gauze, a small quantity of the same material being placed in the vagina.

A Speedy Method of Dilating a Rigid Os in Parturition.

FARRAR, J., M.D. "A Speedy Method of Dilating a Rigid Os in Parturition." *Brit. Med. Jour.*, Sept. 17th, 1898.

The author of this paper, read at the Edinburgh meeting, reports five cases in which he made use of the local application of a ten per cent. solution of cocaine to overcome rigidity of the os in labour.

All the cases reported were primipara, and all had been in hard labour for many hours. In all the condition disappeared within five minutes of the application, leaving the os widely dilated.

He recommended that in these cases a sterile 10 per cent. solution of cocaine be swabbed over the cervix by means of a piece of absorbent cotton, held in a pair of forceps. The solution must be held in contact for a moment or two to secure the best results.

In no case was any deleterious effect of the cocaine noted.

In the discussion which followed the reading of the paper Dr. R. Jardine, of Glasgow, stated that he had employed cocaine pessaries without result.

Several physicians have, since the publication of the paper, reported cases in the *B. M. J.*, where this drug, applied as Dr. Farrar suggested, has proved very effectual.

Forty Cases of Fever in the Puerperium.

WILLIAMS, WHITRIDGE J. "Forty cases of fever in the puerperium, with bacteriological examination of the uterine contents."—*Am. Jour. of Obstet.*, Sept. 1898.

Dr. Williams has adopted the rule of making a bacteriological examination of the uterine contents in every case where the temperature reaches 101° F., in hospital cases, and 102° F., in out-patient practice. After thoroughly cleansing the external genitals with soap and water, and a 1-1000 bichloride solution, the patient is placed in the Sims' position, and a Sims speculum inserted, the anterior lip of the cervix is seized with a bullet forceps, drawn down as far as possible and then the exterior of the cervix is wiped off with sterile cotton. A sterile glass tube 25 c.m. long and ⅘ millimeters in diameter with one end curved like a uterine sound, is then inserted and passed up to the fundus. A large syringe is attached to the free end of the tube and by suction a quantity of the lochia thus drawn up with it. Both ends of the tube are sealed after its withdrawal. Both cover glass and cultures are then made from the contents of the tube.

The cases reported were from the following sources: 22 from the obstetrical wards of the Johns Hopkins Hospital, eight from the out-patient department of the same hospital and 10 cases from consultation practice.

Streptococci were found in eight cases; staphylococci in three cases; colon bacilli in six cases; gonococci in two cases; anaërobic bacteria in four cases; unidentified aërobic bacteria in three cases; bacteria in cover glass, but cultures sterile in four cases; diphtheria bacilli in one case; typhoid bacilli in one case; gas bacilli (bacillus aërogenes capsulatus) in one case; cover glass cultures, but blood sterile in 11 cases; cover glass and cultures sterile, but with malarial plasmodia in blood, in one case. Total of 44 cases.

The apparent discrepancy is due to mixed infection in several of the cases.

In the eight cases where streptococci were found, they were demonstrated in pure culture in six, while in two there was a mixed infection. None of these cases terminated fatally. Staphylococci were found, once alone and twice associated with other organisms.

Colon bacillus present in six cases, were found in pure culture in four and mixed in two. Two of these cases were subjects of eclampsia, in which the bowels were acted upon by croton oil so the source of infection was not far to seek.

The practical value of bacteriological examination was specially

demonstrated in the fact that in eleven of the cases with marked rise of temperature, the examination showed the absence of infective bacteria in the lochia. Further observation of the above group of cases showed that in many instances the temperature was probably due to an auto-intoxication from the intestines as was indicated by the rapid fall of temperature after brisk purgation.

The Practical Significance of Bacteria Found in the Vagina.

WILLIAMS, WHITRIDGE J. "The practical significance of bacteria found in the vagina."—*Am. Jour. Obstet.*, Oct. 1898.

From the bacteriological examination of the vaginal secretion in ninety-two pregnant women, Dr. Williams comes to the following conclusions: That auto-infection is impossible, as he found in but two of the cases the staphylococcus epidermidis albus, and in none of them any of the usual pyogenic cocci. That the gonococcus is occasionally found in the vaginal secretion, and may extend into the uterus and tubes during the puerperal state. That it is possible, but not yet demonstrated, that in rare instances the vagina may contain bacteria, which may give rise to sapræmia and putrefactive endometritis by auto-infection. That death from puerperal infection is always due to infection from without, and usually follows the neglect of aseptic precautions by doctor or nurse.

He advises that vaginal examinations be made as infrequently as possible, and that before such, the external genitals should be cleansed and disinfected carefully, the examining hand being rendered as aseptic as for a laparotomy. He considers that vaginal douches are not necessary and are probably harmful.

D. J. Evans.

Reviews and Notices of Books.

A Text-Book of Obstetrics. By Barton Cooke Hirst, M.D., Professor of Obstetrics in the University of Pennsylvania. With 653 illustrations. Pp. 820. Philadelphia: W. B. Saunders, 1898. Price in cloth, $5.00. Sheep or half morocco, $6.00

Prof. Hirst, as he states in his preface, has devoted himself exclusively for the past twelve years to the practice and teaching of obstetrics. His position as an author and teacher warrant the expectation of a work of special merit.

In this book, Prof. Hirst has given the profession a thoroughly excellent *précis* of the subject of obstetrics.

He has considered the subject under the following main headings: I. Pregnancy; II. The Physiology and Management of Labor and the Puerperium; III. The Mechanism of Labor; IV. The Pathology of Labor; V. The Pathology of the Puerperium; VI. Obstetrical Operations; VII. The New-Born Infant.

It is questionable whether it would not have been better to consider the subject of Mechanism, before taking up that of the Management of Labor and the Puerperium.

From the point of view of the student, scarcely sufficient attention has been paid to the explanation of the mechanism of labor.

In the treatment of eclampsia, the author condemns strongly the employment of pilocarpin. He recommends highly the use of veratrum viride, and the hypodermic injection of large quantities of normal saline solution. In plethoric cases he considers bleeding gives very good results. He condemns the *accouchment forcé*, claiming that "the necessary operation for the delivery of the woman distracts one's attention from the treatment of the convulsions, and adds for the time being, a violent source of irritation to the already highly wrought nervous system."

The chapter on Puerperal Sepsis is most masterly, it is probably the best presentation of this subject, take it all round, in English to-day. He condemns the use of the vaginal douche before labor, as it cannot be depended upon. In those cases where it is necessary, the vagina must be scrubbed out as well as douched, as for a surgical operation. He recommends the routine examination of the vaginal discharge before labor.

The author's observations on the employment of antistreptococci serum are worthy of the gravest consideration.

Prof. Hirst has had an unusual experience in the performance of

Cæsarian section, and declares his preference for the Porro to the Sänger operation.

The section devoted to the consideration of the new-born infant is particularly good. The author considers Schultze's method, together with mouth to mouth insufflation, the best treatment in asphyxia neonatorum. He condemns the method of Sylvester, because the pectoral muscles are too weak to draw up the thorax.

The whole presentation of the subject is most practical, and the author's style clear and concise.

We highly commend the book to the senior student, and the physician, as being one of the very best text-books on obstetrics.

The publisher's work leaves but little to be desired. The illustrations, many of them original, are if anything too numerous, while the colored plates had much better have been omitted.

The index is very full and has evidently been very carefully prepared.

D. J. E.

Society Proceedings.

MONTREAL MEDICO-CHIRURGICAL SOCIETY.

Stated Meeting, November 21st, 1898.

J. G. ADAMI, M.D., PRESIDENT, IN THE CHAIR,

Drs. A. D. Stewart, D. A. Shirres and I. C. Sharp, of Montreal, were elected ordinary members. Drs. Schwartz, Peters, Smith, Brown, Patterson, Harvey, Lynch and Thomas, of Montreal General Hospital, were elected temporary members.

A Case for Diagnosis.

Dr. J. A. SPRINGLE exhibited a child with multiple firm tumours of the hand. In the absence of a histological examination he had been unable to make a diagnosis, but having received permission to remove one of the growths, would report the result at the next meeting. A full report of this case will appear later.

Dr. F. J. SHEPHERD had carefully examined the case and could come to no definite conclusion regarding its nature. From its course he thought that it must be infective, and the diagnosis lay between a fibrous, a tuberculous, or a sarcomatous condition. He did not think it was syphilitic. The question would have to be decided by a histological examination.

Tuberculosis of the Fallopian Tubes.

Dr. F. A. LOCKHART reported this case and showed the specimen. The report will appear next month.

Dr. LAPTHORN SMITH thought this was likely a case of pus-tubes in which the pus had become inspissated. The first constriction had been probably the one at the uterine end, and this had cut off the cavity of the tube from the uterus and later the second constriction had developed, giving rise to the separate tumours. He considered that the operator had done well to leave the ovaries, it showed well-timed conservatism, and even if pregnancy did not occur, the patient was freed from the nerve storms and other discomforts of a premature menopause.

Dr. LOCKHART, in reply, said that the tube was lying quite free in the pelvis, and there was no sign that adhesions had ever taken place, so that there was no possibility of Dr. Smith's ingenious explanation being the correct one.

Carcinoma of the Ovary.

Dr. F. A. L. LOCKHART read the report of the case and Dr. D. P. ANDERSON demonstrated the pathological specimens from it. It will be published later.

Dr. LAPTHORN SMITH referred to a similar case of his own in which operation had meant three months more of life to the patient and a comparatively easy death. He thought that operation was justified in these cases even from that consideration alone. He asked if there had been any fluid found free in the peritoneum, as in nearly every case in which he had found fluid free with tumour, that tumour had proved to be malignant.

Dr. LOCKHART said that there was no fluid in the abdomen.

Intestinal Obstruction.

Dr. F. J. SHEPHERD reported this case. (See page 46.)

Dr. JAMES BELL said that the question of intestinal obstruction following abdominal operations was becoming an important one, owing to the frequency with which the abdomen is opened nowadays. He referred to a case recently under his care: A woman, aged 32, who had had a double ovariotomy performed on August 26th and had made a good recovery and kept quite well (with the exception of intestinal symptoms) until October 13th. At 2 a.m. of that day, symptoms of obstruction set in and persisted until the operation 83 hours later. On opening the abdomen he had found a foot and a half of the small intestine, at about one foot above the ileo-cæcal valve, coiled up and adherent. The intestine above was distended to the size of a coat-sleeve and quite black, while the adherent portion was not larger than the little finger. He had separated the constricted part and disentangled it, but, on returning the distended portion of intestine, his finger had perforated it, and a gush of fluid fæces took place. After washing up, the rent in the intestine was repaired and recovery took place without a bad symptom. The speaker thought the fact that these cases get permanently well was most extraordinary, on account of the damage done to a considerable portion of intestine. This made the third case that he had operated on under similar circumstances with recovery.

Dr. Bell furthermore advocated evacuation of the bowel in these cases. The portion above the obstruction was always found full of liquid fæces and kept up the distension, and drainage and irrigation were an important part of the operation. One often hesitated about opening the intestine on account of the impending gangrene, which made one dread being unable to suture it satisfactorily.

Dr. A. LAPTHORN SMITH had opened the abdomen five times for

obstruction. One of these was for a knuckle of small intestine caught by adhesions to a raw place left by detaching omentum, and on freeing these the vermicular movements had at once carried off the contents. The intestine was enormously distended above this point, but collapsed below it. In another case, on opening the abdomen, he had found the bowel attached to the stump of an ovary, and separating the adhesions was all that was necessary.

He suggested to Dr. Shepherd that some of the dangers of an operation of this kind could be reduced by having made, the same size as the operating table, a pan of hot water to be kept at a temperature of 110° F. There would then be less difficulty in keeping the intestines properly warmed while outside the abdominal cavity, and the patient would leave the table warm, instead of with a temperature lowered to 96° as was often the case.

Dr. Wesley Mills suggested that when hyperæmia in the region concerned in this case was not to be explained on obvious mechanical principles, the nervous system was to be interrogated. The splanchnic region was that so much used by nature to regulate the blood pressure and relieve the heart; it alone, as physiology had demonstrated, could contain all the blood of the body, and its importance in the vital economy,—even in the maintenance of life itself,—recent experiments had rendered clearer than ever. Considering the rapidity of the changes after the unfortunate conditions had been altered, it seemed highly probable that the restoration of a normal circulation in the intestine was to be explained through the action of the vaso-motor centre by means of the splanchnic and other nerves of the sympathetic system. The abnormal condition of the circulation as well as the restoration to the normal, was probably brought about reflexly, though the direct action of toxins, etc., on the nervous centres as a cause of the hyperæmia, was also to be considered.

Dr. Shepherd, in reply, said that Kocher looked upon the congestion as due to hyperdistension of the intestine, and claimed that he could produce the same condition in animals by distending the intestine with gas. With regard to the volvulus, he had never seen or read of a similar case, but had not looked the matter up.

Functional Heart Murmurs.

Dr. James Stewart read a paper on this subject by Dr. Maude Abbott. (See page 1.)

Dr. W. F. Hamilton expressed his admiration for the character of the work displayed in the paper. So full of important details is it that in order to derive the full benefit therefrom one must peruse it thoughtfully and meditatively. To all interested in the study of cardiac cases such observations are especially helpful.

He regretted, however, that more attention had not been paid to the character of the cardiac sounds, for much aid in diagnosis may be thus obtained. The division of cases into anæmic and non-anæmic classs was of doubtful utility since the division was not made in many instances by the hæmoglobinometer, but by the general appearance of the patient.

Again, no reference was made to the posturing of patients as a means of diagnosis between functional and organic murmurs.

These remarks were not meant to detract in any wise from the value of the paper. Such features as these could only be secured by a careful supervision of all cases presenting cardiac murmurs with the end in view, viz., that of distinguishing the functional from the organic murmurs.

Dr. J. B. McConnell had listened with both pleasure and pride to the excellent paper from his former student, Dr. Abbott. The paper pointed to the great importance of having careful detailed reports of all cases in our hospitals. He was a little surprised that no reference occurred in it to cardio-pulmonary murmurs. Potain claimed that most of the functional murmurs were of this class and a large proportion of those hitherto regarded as cardio-hæmic and cardio-vascular. They are heard chiefly in the pulmonary area and over the conus arteriosis, as antero-posterior excursion during the cardiac systole is greatest here. The murmur is caused by compression of the lung against the chest-wall during the heart's systole or during the diastolic phase by aspiration of a portion of the lung lying in contact with the heart. The murmur may be heard at all periods of the cardiac cycle, presystolic, systolic, telesystolic, diastolic and telediastolic. They can as a rule only be diagnosed by exclusion, and they usually disappear on forced inspiration or expiration and sometimes in the horizontal position. That hæmic murmurs existed and depended on a changed relation between the vessel and its contents would seem improbable from the fact that such changes would be expected to show themselves by a murmur in the aorta where the pressure is very much greater than in the pulmonary artery. The absence of hypertrophy was a point in diagnosing a cardio-pulmonary murmur, but this, he thought, could only apply to the systolic variety which is usually explained by supposing a relative mitral insufficiency to have occurred, and they would occur the more readily if the heart was enlarged because the heart would more likely impinge upon lung then. In a case he had examined about a week previous, where from excessive bicycling there was hypertrophy but no valvular lesion, he was able to make out cardio-pulmonary murmurs. Their innocence

in many cases made it important to diagnose them from a life insurance point of view.

The PRESIDENT held it a matter over which the Society might well congratulate itself that the first communicaton addressed to it by a medical woman was of such high scientific value. Although Miss Abbott very modestly depreciated the value of the results obtained by her, undoubtedly those results must receive wide attention and be the basis of yet other researches in other hospital records and other hospital wards. It is painful to think how much material of the utmost value is stored away in our hospital reports—material which only needs to be carefully collected in order to yield results which must establish the name and reputation of whoever will give the necessary time. For of all methods establishing facts and theories in medicine, the statistical stands preëminent. Granted only that observations have been honestly recorded in the clinical notes, an observation, of relatively small value in itself as establishing any point, becomes most important when treated as a member of a group. For himself he became impatient with the blind folly of too many of the younger members of the profession in our larger hospital towns in thinking that they will more likely ensure a competence and a name by sitting still in their office waiting for practice to come to them instead of going forth to do some piece of work in the wards or registraries of the hospitals, work which conscientiously performed not only strengthens the individual and renders him a more capable member of the profession, but also must bring him into favourable notice and help him onwards. For the beginner in medical research nothing is simpler, few things more valuable, than to work through hospital records in order to establish or refute one single point. It was only necessary to read that most popular and valuable of all modern works upon medicine, of which, and of the author of which, Montrealers are so proud, to see how its value depends upon the constant adherence to the statistical method. He could assure the younger members of the Society that they would find every opportunity given to them at both of the larger English hospitals to carry on such work.

Coming to Dr. Abbott's own work, in his opinion the most important results achieved was the light thrown upon the causation of functional murmurs by the study of cases, such as those of pernicious anæmia, in which we know that the heart muscle itself is affected, as compared with others in which no such affection is reasonably to be anticipated. This alone, in a subject so obscure, is an advance of high value.

Iniencephalic Monstrosity.

Drs. H. S. SHAW and D. J. EVANS then exhibited the body of an apparently full time female infant which was an almost perfect specimen of *Iniencephalus*.

The whole aspect of the specimen was that of a "Brownie."

The head was well-formed but owing to lack of development of the neural canal throughout nearly its whole length. the occipital and sacral regions were in direct continuity. Just at their junction below the hair of the scalp was a small meningocele. The face was well formed and directed upwards. Anteriorly the chin seemed to extend to the sternum there being no neck. The chest and abdomen were well-formed. The extremities seemed to be unusually long and were free from deformity.

Photographs had been taken of the specimen. Frozen sections of the monster would be made and studied and a later report made to the society.

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PRIVATE WARDS IN PUBLIC HOSPITALS A CAUSE OF HOSPITAL ABUSE.

Private wards in public hospitals are occupied by the more wealthy patients. The wealthy patients set the fashion. It is no longer difficult to induce poor patients to go to the hospital. The changed attitude of the poor toward the hospital is in part due to the improved character of the public wards, the good nursing and medical care, and also to the fact that the leaders in society and the wealthy so commonly go to the hospital in case of sickness and for surgical operation.

A lady or a gentleman who has been in a hospital as a patient and has learned to appreciate the advantages of hospital facilities for treatment, advises a poorer neighbour to go also. The poorer neighbour replies, "Oh, yes, I know; but the expense is greater than I can afford." The natural rejoinder is, "Well, then, go into the public ward. I saw the public wards, they are clean and bright, and you get there the same nursing and the same physician or surgeon as you would in a private ward, and no expense at all, or a matter of fifty cents a day."

Let any one go down to the court house and it will be obvious that the legal profession, as a class, make their living out of the very class of people that the medical profession treat gratis. But if lawyers had two adjoining offices, and in one gave advice for nothing and in the other charged fees, how many would try to get into the free office? The lawyers adopt a much wiser plan. Before a man can take out a suit in "Forma Pauperis," he must go before a judge and, under oath, state that he has not the means to pay.

Of course the circumstances of the two classes, the patients on the one hand and the clients on the other, are very different, and the

comparison cannot be carried too far. Yet, nevertheless, it is unquestionably true that the members of the medical profession are often imposed upon, and the imposition is assuming alarming proportions in the large cities of Canada and the United States, as well as in England and on the Continent. Something must be done and done wisely, to lessen this growing evil without letting the deserving suffer, and one way is to remove private wards altogether from public hospitals, and leave to private enterprise the care of those who can afford to pay for and should pay for private wards. When it becomes known that only the poor go to the public hospital, then pride alone will largely diminish the numbers applying for free treatment in public hospitals.

McGILL MEDICAL LIBRARY.

Donations to the Medical Library for quarter ending Nov. 30, 1898, presented by the authors.

George William Balfour, M.D., (St. And.), F.R.C.P. Ed., F.R.S. Ed.—Clinical Lectures on Diseases of the Heart and Aorta.

James B. Ball, M.D. (Lond.)—A Handbook of Diseases of the Nose and Pharynx, 3 Ed., 1897.

Gilbert A. Bannatyne, M.D. (Glas.), M.R.C.P. Ed.—Rheumatoid Arthritis; Its Pathology, Morbid Anatomy, and Treatment, 2 ed., 1898.

Charlton Bastian, M.D.—Some Problems in Connection with Aphasia and Other Speech Defects, 1897.

Nathan Smith Davis, A.M., M.D., LL.D.—Lectures on the Principles and Practice of Medicine, 2 Ed., 1886.

Nathan Smith Davis, jr,, A.M., M.D.—Diseases of the Lungs, Heart and Kidneys, 1892.

L. Emmett Holt, A.M., M.D.—Care and Feeding of Children, 1897.

Roberts P. Hudson, M.D.—Michigan a Summer and Health Resort State, 1898.

Horace Jayne, M.D., Ph.D.—Mammalian Anatomy, a Preparation for Human and Comparative Anatomy. Part I., The Skeleton of the Cat, 1898.

Donald MacAlister, M.D., and Henry W. Cattell, M.D.—Text Book of Special Pathological Anatomy, by Ernst Zeigler, Translators and Editors, 1897.

Wesley Mills, M.A., M.D., D.V.S., F.R.S.C.—The Nature and Development of Animal Intelligence, 1898.

Edward Nettleship, F.R.C.S.—Diseases of the Eye. A Manual for Students, 6 Ed., 1897.

William Osler, M.D.—The Principles and Practice of Medicine, 3 Ed., 1898.

G. N. Stewart, M.A., D.Sc. Ed., D.P.H. Camb.—A Manual of Physiology with Practical Exercises, 3 Ed., 1898.

Augustus D. Waller, M.D., F.R.S.—Lectures on Physiology, 1897.

David Walsh, M.D. (Edin.)—Premature Burial: Fact or Fiction? 1897. Excretory Irritation, and the Action of Certain Internal Remedies on the Skin, 1897.

William I. Walsham, M.D.—Nasal Irritation.

The Library is also indebted to the following contributors:

Professor Adami—Medical Chronicle, Vols. 1, 2, 6, N.S. Lyon Medical, Vols. 84, 85, 86. Gazette des Hopitaux, 1892-1895-1896, 3 Vols..

Professor Armstrong—Boston Medical and Surgical Journal for 1898.

Prof. Blackader—International Medical Magazine, 1897.

British Balneological and Climatological Society, Journal of, Vol. 1, 1897.

Professor Cameron—American Journal of Obstetrics, 1897. The Extra Pharmacopœia, by W. Martindale, 8 Ed., 1895.

Chicago Medical Record, V. 14, 1898.

Editor, Landon B. Edwards, M.D. Virginia Medical Journal, 1898.

Professor Johnston—Fortschritte der Medicine, Vols. 6 and 7.

King's College Hospital Reports, Vol. 4, 1898.

Liverpool Royal Infirmary Reports, 1898.

Professor MacAlister—History of Epidemics in Britain, by Charles Creighton, M.A., M.D., 2 Vols., 1894. A Geographical History of Mammals, by R. Lydekker, B.A., F.R.S. etc., 1896. Manual of Practical Morbid Anatomy, by H. D. Rolleston, M.A., M.D., and A. A. Kanthack, M.D., M.R.C.P., 1894. Vertebrate Skeleton, by Sidney H. Reynolds, M.A., 1897.

Catalogue of the Osteological Portion of Specimens Contained in the Anatomical Museum of the University of Cambridge, 1862. The Electrical Researches of the Honourable Henry Cavendish, F.R.S., written between 1771 and 1781. Ed. by J. Clerk Maxwell, F.R.S., 1879.

Methodist Episcopal Hospital Reports, Vol. 1, 1898.

Middlesex Hospital Reports, 1897.

McGill's Graduate Society—The Doctor's Window, 1898.

The Histopathology of the Diseases of the Skin, by Dr. P. G. Unna, tr. by Norman Walker, M.D., 1896. Masters of Medicine—Life of Sir Benjamin Collins Brodie, by Timothy Holmes, 1898.

Life of John Hunter, by Stephen Paget, 1897.

Life of William Stokes, by Sir William Stokes, 1898.

Proceedings of the New York Pathological Society, 1897.

Professor Shepherd—Medical Record, 3 Vols. Proceedings of the 10th Annual Meeting of the Association of American Anatomicals, 1898.

Transactions of the Canadian Institute.

Transactions of the American Surgical Association, 1898.

Transactions of the Association of American Physicians, 1898.

Transactions of the American Association of Obstetricians and Gynæcologists, 1898.

Transactions of the New York State Medical Association, 1897.

Transactions of the American Pediatric Society, 2 Vols., 1894-1897.

Twenty-fourth Annual Report State Board of Health, Michigan, 1897.

Transactions of the Medical Society of Ohio, 1898.

Transactions of the Medical Society, State of New York, 1898.

Transactions of the Chicago Pathological Society, 2 Vols., 1896-9.

Transactions of the British Dermatological Society of Great Britain and Ireland, 4 Vols., 1895-98.

Text Books and other new works:

Centralblatt für Innere Medicin, 1897; Journal of Physiology, 1897; American Text Book of Physiology, 1897; Revue des Sciences Médicales, 7 Vols., 1873-98; Journal of the American Medical Association, 1898; New York Medical Journal, 1898; Medical News, 1898; Centralblatt für Physiologie, 1897; Zeitschrift für Physiologische Chemie, 1897.

Deutsche Zeitschrift für Chirurgie, Bds. 46, 47, 1897-8.

Chemisches Centralblatt, 1897.

System of Medicine, by Loomis, Vol. 6, 1898.

Retrospect of Medicine, 1898.

Organic Chemistry, by Ira Remsen, 1897.

Histology, by E. Kleins, M.D., and T. S. Edkins, M.D., 1898.

Zeitschrift für Hygiene und Infectionskrankheiten, vols. 21, 26, 27, 1896-97.

Journal de la Physiologie, 1860.

Archir für Klinische Medicin, 1887.

Index Medicus, 1897-8.

Archiv für Physiologie, Vols. 69, 71, 1898.
Zeitschrift für Kl. Med., 1898.
Journal of Physiology, 1897.
System of Practical Medicine, Vol, 4, Ed. by A. E. Loomis, 1898.
Pharmacy, Whitla.
Principles of Bacteriology, by A. C. Abbott, M.D., 1897.
The Art of Massage, by J. H. Kellogg, M.D., 1895.
British Pharmacopœia, 1898.
Materia Medica and Therapeutics, by J. M. Bruce, M.D.
Botany, by Charles E. Bessy, Ph.D., 1892.
Manual of Bacteriology, by Robert Muir, M.A., M.D., and James Ritchie, M.A. M.D., 1897.

The library of the late Allen Ruttan, Esq., M.D., has been presented to the Medical Faculty.

GESTA MEDICORUM.

"QUICQUID AGUNT MEDICI NOSTRI FARRAGO LIBELLI."

Dr. F. Gilday has decided to commence practice in Montreal.

Dr. R. H. Craig has been appointed Laryngologist to the Montreal Dispensary.

There is a proposal on foot to establish an 'Academy of Medicine' in Toronto.

Sir William Jenner, the distinguised pathologist and physician in ordinary to the Queen and the Prince of Wales, died Dec. 12th.

Koch, at Nürnberg, has recently shown a case in which there was abdominal actinomycosis starting from the vermiform appendix.

Dr. David Patrick has been appointed Clinical Assistant in Children's Diseases and the Dermatological Clinic at the Montreal General Hospital.

It has been decided to establish a fund for the benefit of St. Luke's Hospital, Ottawa, in memory of the late Dr. H. P. Wright, to be called the "Dr. H. P. Wright Memorial Fund." The idea seems to be meeting with favour as $3000 were subscribed in one day.

Dr. T. G. Roddick, M.P., Professor of Surgery in McGill University was elected an Associate Fellow of the College of Physicians of Philadelphia, at the last meeting. This body was founded in 1781, and is one of the largest and most important of the American Medical Associations.

The proprietors of the "Canadian Practitioner" and the "Medical Review" have agreed to amalgamate in 1899, the name of the new journal to be "The Canadian Practitioner and Medical Review." The

editors hope that by combining forces they will be able to publish a better journal than either has been in the past.

We learn that it has been decided to postpone the meeting of the third Pan-American Medical Congress which was to have met in Caracas, Venezuela, in 1899. This has been advisable owing to the late revolution in that country and to the fact that there has been a grave epidemic of small-pox. The Congress will therefore meet in 1900.

Rummo, of Palermo, at the Academy, reported a particular form of "dystrophic skin disease," under the name of geroderma genito-dystrophicum.

In this disease there is early senility, and a condition which has some relation to myxœdema, akromegaly and gigantism.

The skin of the face is waxy-yellow, the forehead low and furrowed, the nasolabial folds very deep, the ears thick, the feet and hands enlarged, the abdomen and breast relaxed, the hair bristling, and the voice nasal. He had been able to collect 15 cases of this disease in Sicily.