lobe, with bronchial breathing, bronchophony, and patches of crepitations irregularly scattered throughout the lung. The evening temperature was 100° F., morning temperature 99° F. or normal; the pulse rate 80, and respiration rate 21. The patient had severe cough (no sputum), cold night sweats, and the clear bright eyes of tuberculous toxæmia. She remained seriously ill for a month, but in two months she was convalescent, and in three months the affected lung showed no abnormality.

Cases 2 and 3 are in marked contrast. In Case 2 there was acute tuberculosis of the bronchial glands only, and every evidence of acute toxæmia. In Case 3 the toxæmia was obviously less, although the lung was extensively involved. This would seem to suggest that in Case 2 there was a definite acute tuberculous infection, whereas in Case 3 the condition was probably caused by an enlarged and adherent tuberculous gland suddenly opening into a bronchial tube, distributing its contents, largely consisting of caseous material with a minimum of tubercle bacilli, into the corresponding lung. This would account for the much more favourable prognosis in the so-called "acute pneumonic phthisis," as compared with the more deadly "broncho-pneumonic" type, in which there is diffuse and profuse infection, probably from acutely inflamed glands, either by an extensive retrogressive spread, or by the passing of large numbers of virulent tubercle bacilli into the blood stream.

Case 4.—A male, aged 37, was under observation for two years suffering from periodic attacks of bronchial asthma, with violent and persistent cough and great lassitude. Repeated examinations revealed crepitations over large areas of the lungs, sometimes in one situation, sometimes in another, and occasionally almost entirely absent. No definite clinical signs of enlarged bronchial glands or of tuberculous invasion of the lung parenchyma were present. There was an old history of pleurisy; the sputum was negative. The patient was suddenly taken acutely ill, and a large consolidation was found at the apex of the right upper lobe, extending to the third rib, and tubercle bacilli were found in large numbers in the sputum. In view of the fact that the lung parenchyma showed no previous evidence of tuberculous invasion, old or new, it is hardly likely that a large consolidation, extending to the third rib, could have occurred in such a brief period, except by a sudden mass infection from such a source as an infected gland.

Case 5.—A male, aged 47, gave a history of persistent cough and lassitude for one year prior to his illness; repeated examinations of the lungs were negative. It is of interest to note that the "reflex areas of dullness" were absent. He was suddenly taken ill with influenza-like symptoms, but a physical examination showed, at the apex of the left upper lobe, delayed movement, impaired resonance down to the third rib, broncho-vesicular breathing, and fine crepitations over the whole affected area. The evening temperature was 100° F., morning temperature 99° F. or normal, and the pulse rate 90 to 100. Although absent before, the reflex areas of dullness were now most obviously present, and the sputum was positive. In three months he was sent to a sanatorium, and the x-ray report was as follows: "There is definite infiltration about the left middle zone and at the left apex. Probably, too, the right upper lobe can be looked upon as slightly abnormal. But on the whole the chest seems to be but moderately slightly involved with tuberculosis."

The usual explanation of this sort of case is that an old lesion has been suddenly lighted up by an attack of influenza. In the first place, there was no old lesion, and, secondly, there was no influenza. The symptoms could, and there is every reason to believe that in this case they did, arise from the shock of a sudden invasion of the lung parenchyma. This would quite obviously give rise to influenza-like symptoms. On the other hand, a sudden invasion from a bronchial gland would account more satisfactorily for all the details of the case, including the preceding persistent dry cough and the large area so quickly affected. The patient's splendid recovery with the minimum of physical signs remaining also seems to support this view. The final sanatorium report was: "His physical signs—never very well marked since admission—are less than ever."

Since writing the above I have again had this patient under treatment. On April 26th, just over one year since his first illness, he complained of cough, did not feel well, and had a temperature. Examination was completely negative. Four days later there was a sudden invasion of the whole left lower lobe, very similar to that of Case 3. This relapse gives further support to the original diagnosis.

No attempt is made to discuss the controversial question of path of infection, but merely to state clinical facts, as observed over the long period of fifteen years.

CONCLUSIONS.

1. Hilus tuberculosis can be demonstrated clinically in a relatively large percentage of children.

2. In spite of much clinical evidence suggesting pulmonary tuberculosis in children actual invasion of the lung parenchyma is relatively rare.

3. The natural tendency in all cases is towards a cure, although in many cases physical signs will remain which, in adult life, will be regarded as old healed lesions.

4. Pressure from enlarged bronchial glands can be demonstrated clinically; it has the effect either of restricting the free flow of air into a lung or of interfering with the free circulation of blood in the lungs, and giving rise to localized areas of oedema, rarely to general pulmonary oedema.

5. Rupture of adherent caseous glands into a bronchial tube is a not uncommon cause of pulmonary tuberculosis.

The diminished death rate from pulmonary tuberculosis is probably due mainly to the lessened risk of infection from "open cases" in the homes, and to the more prompt recognition of early cases. It is unlikely that a further appreciable decrease will occur unless a serious attempt is made to deal with the large number of potential cases *before*, not *after*, the lung parenchyma has been invaded by the dreaded tubercle bacilli.

TONSILS AND ADENOIDS: THEIR MEDICAL AND SURGICAL ASPECTS.*

BY

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INTRODUCTION.

THE subject of "tonsils and adenoids," which has been chosen for discussion to-day, might well be described as a "best-seller," for there is no medical topic which the laity of these islands discuss with more frequency, more verve, and more ignorance than this, not excepting appendicitis itself; while professional opinion, always with us in a small trickle, sometimes attains the level of a flood, as witnessed by the spate of letters in the *British Medical Journal* early this year. The subject may be said to have reached its apotheosis with the publication of Irwin Moore's monumental work last year.

For the purposes of this discussion I shall confine my remarks to those conditions produced by chronic sepsis and hyperplasia of these lymphoid masses—that is, to the clinical and pathological conditions generally meant when a patient is said to be suffering from "adenoids and tonsils." Acute conditions and such rarities as calculi of the tonsils will not come within my purview. The palatine tonsils and the third or pharyngeal tonsil, commonly referred to as adenoids, form part of Waldeyer's ring—that group of lymphadenoid tissue in the nasopharynx and pharynx which surrounds the entrance to the respiratory and alimentary passages.

The function of the tonsils is still *sub judice*; various theories have from time to time been put forward, but it would seem most reasonable to regard them as organs for the defence of the respiratory and digestive tracts during the early years of childhood. As StClair Thomson succinctly puts it:

"The ages in which they are most in evidence, their situation, their structure, the recognized emigration of lymphocytes through their epithelium, their frequent enlargement with an infectious process, the frequency with which, when their resistance is overcome, they appear to be starting-points for infection, their lasting hypertrophy after prolonged or repeated infections, experimental observations which show that they serve as ports of entry for infection and their normal involution at puberty, all appear to confirm this view."

* Read in opening a discussion in the Section of Diseases of Children at the Annual Meeting of the British Medical Association, Manchester, 1929.

When a tonsil becomes chronically hypertrophied or diseased it loses its defensive powers and comes to resemble a choked filter. This is borne out by the greater frequency with which children possessing these hypertrophies contract infectious diseases and the severity of the ear and throat complications in such cases.

It will now be convenient to discuss the question of adenoids and tonsils separately.

ADENOIDS.

When Luschka's tonsil, as the third or pharyngeal tonsil is sometimes called, becomes hypertrophied and gives rise to definite symptoms, adenoids or adenoid vegetations are said to be present.

The precise cause of this hypertrophy is at present unknown, but it may be stated that in most cases it is due to a lowered vital resistance and an exciting or local cause, such as catarrhal attacks or infectious fevers. William Meyer of Copenhagen, in 1868, was the first to recognize this very common affection of childhood. So widespread is it that no climate or race seems exempt, the negro being perhaps the least affected; but there is no doubt that adenoids are more frequent in damp temperate climates like our own; indeed, Osler has stated that there are more mouth-breathers to the acre in England than in any other country. They are most common between the ages of 3 and 14 years, but may be present at birth, and not infrequently produce symptoms during the first year of life, especially among the poorer classes. This is a fact that I should like to emphasize, and to which I first called attention in a paper in the *Lancet* in 1922. The main symptoms produced in the first year of life are nasal obstruction and discharge. The mother complains that the child has to fight for breath, and takes the breast or bottle with difficulty, as the nasal obstruction prevents the infant from breathing and suckling at the same time. If this obstruction is not removed spoon feeding has to be resorted to, otherwise death from inanition takes place. Such infants are liable to convulsions and vomiting. At the Royal Manchester Children's Hospital I operate on between 30 and 40 of these cases every year, with excellent results, many of the infants being only a few weeks old. I have, indeed, operated as early as the third week. In these cases no anaesthetic is required, and this practically eliminates all risk from the operation. Infants stand the operation remarkably well, and are often enjoying a feed within an hour afterwards.

Symptoms.

Symptoms of adenoids in older children vary within wide limits, depending upon the degree of hypertrophy or sepsis present and the capacity and form of the nasopharynx. They may be classified into the following groups.

1. *Nasal Obstruction, often accompanied by a Mucopurulent or Purulent Discharge.*—I have already referred to these symptoms in the infant. In older children, from 3 years onwards, the classical picture is often met with—open mouth, rabbit teeth, receding chin, pinched alae nasi, high arched palate, noisy respiration, snoring at night, vacant expression, the so-called adenoid facies, etc. It must always be remembered that adenoids can be present causing such marked secondary symptoms—for example, aural—that operation is urgently called for, and yet all the above classical symptoms of nasal obstruction may be absent.

2. *Secondary Affections.*—The most common and most important secondary affection is aural, with consequent deafness, suppuration, and possible sequelae. It is no exaggeration to say that 90 per cent. of cases of deafness in children are due to adenoids. So-called "glandular fever"—recurring febrile attacks with swelling and tenderness in the glands of the neck—is due to local infection of the adenoid tissue of Waldeyer's ring, most commonly the pharyngeal tonsil. Other secondary affections are: chronic cervical adenitis (affecting the glands of the posterior triangle of the neck), defective pulmonary expansion, chronic bronchitis, and gastro-intestinal catarrh.

3. *Reflex Neuroses.*—A long list of these could be compiled, but I will give you only those of which I am sure from personal experience: enuresis, night terrors, and

twitchings are frequently benefited by the removal of adenoids. In asthma adenoids should always be removed if present, but they form merely a contributory factor in the disease. The same may be said about chorea, laryngismus stridulus, stammering, and convulsions.

4. *Constitutional Changes.*—These may be physical or mental. The physical include high arched palate, projecting teeth, retracted chest, and pigeon-breast; while among the mental changes are those producing the aprosexia of Guye.

Diagnosis.

The diagnosis of adenoids is usually quite easy. In older children posterior rhinoscopy will reveal the growth in many cases. In younger children, and in those cases in which posterior rhinoscopy is impossible, it may be necessary to palpate the nasopharynx with the index finger; but this is a very disagreeable procedure for the patient, and should only be resorted to when really necessary.

If there are evidences of nasal obstruction in a child, and the examination of the nose and pharynx does not explain it, the presence of adenoids can be assumed as a rule. The other conditions which may simulate adenoids are a nasospatial polypus and a nasopharyngeal fibroma in its early stage.

In infants, when adenoids are strongly suspected, after excluding syphilis, it is my practice to introduce a small curette into the nasopharynx, without general anaesthesia. If adenoids are present they are removed. If the nasopharynx is free no harm is done. It must be borne in mind that in the Mongol type of idiot nasal obstruction is frequently present with but a trifling amount of adenoids. As these patients are bad subjects for an operation, I usually leave them alone, unless a very strong case is presented.

Treatment.

If adenoid vegetations are present in sufficient bulk, or are sufficiently septic, to cause any of the cardinal symptoms referred to, in my opinion only one form of treatment at present exists—that is, surgical removal. All palliative treatment is useless, and some forms, such as nose drill, worse than useless.

The best instrument to employ, and one that is used by the majority of surgeons, is StClair Thomson's curette, by means of which the adenoid mass is removed in one entire piece. Any lateral prolongations which may not have been included in the sweep of the curette are planed down with a simple Gottstein's instrument. The La Force adenotome is much used in America and on the Continent, and has a warm advocate in this country in Dr. Pearce Sturm. I have given this instrument a fair trial, but in my hands it has not proved satisfactory. A point to keep in mind is that the cutting blade of the curette should have a sharp edge.

As regards haemorrhage after the removal of adenoids: immediate bleeding is usually very brisk, but as a rule ceases within a few minutes; freely bathing the face and forehead in cold water greatly assists in diminishing this. If the bleeding persists search should be made, under good illumination, for a tag of lymphoid tissue which may have been left behind. This is the most common cause of immediate excessive haemorrhage. Should the bleeding still persist after the removal of a tag, or should no tag be present, a swab of suitable size should be squeezed out of liquid hydrogen peroxide and introduced into the nasopharynx by StClair Thomson's forceps and pressure made for a few minutes. This, in my experience, together with the administration of haemoplastin, is invariably successful, except in the case of a genuine bleeder. Since Dr. Lowndes Yates published his interesting investigations in regard to the relationship of blood-clotting time and the acidity of blood and urine, it has been my practice to give all my patients a course of sodium bicarbonate before operation.

True secondary haemorrhage is rare. I have had two cases in my own practice. One was a hospital patient who had been operated upon and returned home. Her own doctor telephoned to me on the fifth day to say that he had been summoned in the middle of the night for nose-bleeding, and had plugged the nasopharynx and nose. She was readmitted to hospital, but no further bleeding occurred. The other was a very marasmic infant of under 2 months,

who was so badly obstructed that nourishment was taken with the greatest difficulty. This infant was under the care of my colleague Dr. Heywood. On the fourth day after operation attacks of vomiting with blood in the vomit occurred, and death soon supervened. I did not see the patient myself, but death was attributed to secondary haemorrhage from the nasopharynx.

One other fatality from post-operative haemorrhage I have had, making two in all, and I have operated upon well over 12,000 cases. In this case death occurred about eleven hours after operation. I was not notified of the boy's condition until about forty minutes before death took place, and when I reached him he was moribund.

The results following removal of adenoids in suitable cases, especially in cases of nasal obstruction or aural symptoms, can only be described as extremely good. I think that there is no other surgical operation in which results are more promptly beneficial and of such far-reaching effect upon the health of the patient.

Do adenoids recur? The answer is Yes, in a small percentage of cases. Irwin Moore says that statistics show that, if thoroughly removed, there is a tendency to recur in about 10 per cent. of cases. Many cases of so-called recurrence are really cases of inefficient removal, but after these have been allowed for there still remains about 10 per cent. in which recurrences will occur in spite of the most efficient removal. Not only have I seen such recurrences in my own practice, but also some in the practice of well-known colleagues in different parts of the country.

TONSILS.

The indications for tonsillectomy may be set forth as follows:

1. Any interference, night or day, with respiration or with speech.
2. Chronic enlargement of the cervical glands. This may be due to chronic sepsis of the tonsils or to tubercle. The incidence of tubercle in tonsils which have the clinical appearance merely of chronic hypertrophy and inflammation has been studied at various times by different observers with results varying from 0.3 to 9 per cent. Scarff and Whitby give this incidence as 3 per cent.

The question sometimes arises: Should the tonsils be enucleated first and the glands dissected out afterwards, or vice versa? In all cases in which it is possible it seems to me that the tonsils should be dealt with first and the original focus thus removed; but whenever the glands show signs of softening they should be dealt with first, thus making sure of a good scar in the neck; for, as you well know, it sometimes happens that directly after enucleation of septic tonsils, the neck glands break down or suppurate.

3. Recurrent sore throats due to frequent attacks of tonsillar inflammation or peritonsillar abscess.
4. Systemic infections attributable to a focus in the tonsil, such as rheumatism, irritable hearts, chronic ill health, and occasionally a rare toxæmia, such as that of the labyrinth.
5. Chronic lacunar tonsillitis, or "cheesy" collections in the supratonsillar fossa or between the tonsil and the pillars. These give rise to foul breath, a bad taste in the mouth, and pharyngeal irritation. This latter condition sometimes produces persistent fits of paroxysmal coughing.
6. In diphtheria "carriers" when the infection is persistent. These cases have not been common in my experience, but I have enucleated tonsils on at least three occasions when positive swabs had been returned for some months after the original infection, in each case with satisfactory results.

Diagnosis of Septic Tonsils.

And now as regards the diagnosis of chronic sepsis in the tonsils. The first point to mark is that the size of a tonsil is no guide to its septicity: indeed, small tonsils are often the worst offenders. It is often difficult to decide whether a given tonsil is really septic, and not infrequently the clinical signs are meagre.

The tonsil should be squeezed by pressing against the anterior faucial pillar with the end of a tongue depressor, so as to force out the contents of the crypts. Pus is sometimes made evident by this means. The anterior

pillar of the fauces should be pulled forward with a blunt hook and the supratonsillar fossa examined by the same means. Cheesy secretions may thus be discovered. The tonsillar glands in the neck at the angle of the jaw should always be searched for, and frequently will be found. Chevalier Jackson and Pavy-Smith have called attention to the presence of a narrow vertical red line of congestion, occupying, and limited to, the anterior surface of the entire length of the anterior faucial pillar. They have found this to be an important and distinguishing feature of a focal streptococcal infection. My own experience confirms this view.

Recently much work has been done on the tonsil puncture method of investigation, but of this I have little experience. The method promises to be useful in certain cases as a help in diagnosis, but my personal preference is always for a diagnosis on clinical grounds if possible. In those cases in which positive clinical signs of tonsillar sepsis may be doubtful and yet a definite condition due to focal infection, such as rheumatism, irritable heart, etc., is present, it is my practice to operate, after trying to eliminate all other possible foci, such as dental, nasal, etc.

Before leaving this part of the subject there is just one other point to consider. Many, indeed I think most, throat surgeons advocate the removal of tonsils as a routine measure in all cases in which an operation for the removal of adenoids is to be performed. This I do not quite agree with. It is true that in the great majority of such cases the tonsils are definitely diseased, or at least suspicious; but there remain a certain number in which, while the adenoids are causing definite symptoms, none can be attributable to the tonsils, and these bodies themselves present a perfectly innocent appearance—for example, cases of aural disease due to adenoids in which the tonsils are small, healthy-looking, and causing no symptoms. In such cases I remove the adenoids alone, for if the tonsils are not diseased we must presume, in the light of present knowledge, that they subserve a useful function, at any rate in childhood.

Tonsillectomy.

We now come to the operation of tonsillectomy, or the total enucleation of the tonsil in its capsule. Two methods are in use: (1) by means of the guillotine; (2) by dissection.

The method of dissection was popularized in this country by Waugh in 1909, and for a few years he had many ardent followers, but at the present time I would say that the majority of laryngologists prefer the guillotine, at any rate for routine cases. Personally, I am quite convinced that the guillotine method, in expert hands, is the safest, being, as a rule, a much shorter operation, and it is equally efficient. Moreover, more cases can be dealt with in a given time. As performed by many of its advocates the dissection operation requires a degree of anaesthesia so profound that it cannot be free from anxiety, and in some cases in which the operator also believes in ligaturing every bleeding point a very considerable period of time may be taken. This at once places the operation as a major instead of a minor one as regards risk. I am also quite sure that the ultimate results of the dissection method are no better than those of the guillotine. In efficient hands complete enucleation can be accomplished by either method and the result will be the same. In inefficient hands both methods may result in failure. Again, both the efficient guillotinist and the efficient dissector will occasionally have an imperfect result, as I have observed, not only in cases of my own, but in those of my colleagues in this and other towns.

Briefly, the case for the guillotine may be stated as an efficient removal of tonsils by a simple, short operation, entailing less risk, less discomfort, and a shorter convalescence than does the dissection method. The type of guillotine used is a matter of personal choice. Heath's is my fancy, and just recently I have tried the Popper-Heath modification with every satisfaction, the serrated portion of the inner ring margin of the head effectively anchoring the tonsil. Others prefer the scissor-handled O'Malley or Ballinger, and of late the crushing instrument of La Force has produced warm advocates.

The main contention of La Force is that the operation is rendered nearly bloodless in the majority of cases, but

there is, of course, no guarantee that both late primary and secondary haemorrhages may not follow its use. Macnab states that after a considerable experience of this instrument both primary and secondary haemorrhages were more frequent than with the single-bladed instrument, and others have also discarded its use after a fair trial. My own view is that it is not nearly so precise or efficient an instrument to manipulate, and that in the vast majority of my own cases I find little difficulty in confining haemorrhage to a minimum. In those cases in which haemorrhage is really troublesome it is very questionable whether a crushing instrument would be of help.

The dissection operation is now reserved by most laryngologists for special cases in which enucleation by the guillotine may seem to present difficulties, but I venture to say that the more one uses the guillotine, and the more expert one becomes, the less does one dissect.

Haemorrhage.

Bleeding is brisk for thirty to sixty seconds, but usually ceases spontaneously if the patient's neck and face are freely bathed in cold water. If this does not suffice, pressure should be applied to the tonsil bed for a minute or two by means of a swab on a holder, and if in three or four minutes bleeding is still free a swab squeezed out of hydrogen peroxide should be applied and pressure maintained for a further few minutes. In the vast majority of cases this will suffice, but in a few (about 1/2 per cent.) further pressure with a swab squeezed out of adrenaline will be needed, at the same time haemoplastin being given subcutaneously.

Beyond this I have had to go on only four occasions. Twice I have ligated a refractory artery of unusual size, situated in both cases at the upper pole of the tonsil bed, and twice I have left pressure forceps on the bleeding point for two or three hours after operation. Some laryngologists advocate the ligation of all bleeding points at every operation. This is a tedious business and considerably lengthens the time of the operation; moreover, it is not an infallible method of stopping haemorrhage, as ligatures have been known to slip, with subsequent severe haemorrhage after the surgeon has left. Many ingenious instruments have been invented for suturing together the pillars of the fauces in cases of severe haemorrhage. It is not a method which appeals to me, and I have never come across a case requiring such drastic treatment.

The surgeon may be called upon to deal with haemorrhage, more or less severe, a few hours after operation. In those cases the tonsil bed is filled with a large clot under which bleeding is taking place. After clearing away the clot, pressure, with the help of hydrogen peroxide or adrenaline, and the hypodermic administration of haemoplastin, has, in my experience, always been effectual in stopping it.

Fatalities.

I have operated on well over 12,000 cases of adenoids and tonsils with two fatalities. These cases, due to post-operative haemorrhage after the removal of adenoids, I have already referred to.

In the records of the Royal Manchester Children's Hospital for the last sixteen years—that is, the period during which I have been on the staff—I can only find a further three deaths following this operation. One patient died from the effects of post-operative haemorrhage, one from tuberculous meningitis. Neither of these cases did I personally see. The third died from sepsis after being operated upon by a medical officer at the out-patient department. A fourth case in which death might possibly be attributed to the operation was that of a mentally defective child at the Children's Hospital. This child, seemingly perfectly fit in every way, collapsed suddenly and died from heart failure three days after operation. A history of diphtheria some two or three months previously was elicited. So that in a period of sixteen years, 1913-28, there was a total of five, or at most six, fatalities following the operation for tonsils and adenoids at the Royal Manchester Children's Hospital. During this period over 27,000 such operations were performed.

As to the ultimate results of the operation I have no figures of my own to give.

In general terms it may be said that if the tonsils are efficiently enucleated the results, as regards the indications for operation I have enumerated, with the exception of chronic enlargement of cervical glands and systemic infections, are excellent, the condition being cured in nearly 100 per cent. of cases. In those cases in which a cure is not obtained it will be found that tonsillar tissue has been left behind.

MATERNAL MORTALITY.*

BY

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DURING the year 1928 there were 676 maternal deaths in Scotland, representing a rate of 7 per 1,000 births, as compared with an average of 6.4 for the preceding ten years. Of these 676 deaths, 234, or 2.4 per 1,000 births, were ascribed to puerperal sepsis.

A preliminary basis for the discussion on maternal mortality may conveniently be found in the conclusions arrived at from the special investigation into a total of 252 maternal deaths occurring in Aberdeen (1928) during the ten years 1918-27—namely:

1. The analysis of the records of maternal deaths in Aberdeen over a period of ten years, as obtained by means of a special system of inquiry, has not revealed any definite relationship between environmental conditions and puerperal mortality. Thus, in turn, puerperal mortality in relation to cleanliness of house, size of house, crowdedness of house, and to congested areas, has been examined, and no significant association of any of these conditions with puerperal mortality has been ascertained.

2. Similarly, the influence on maternal mortality of more personal factors, such as age of the mother, illegitimacy, occupation of the mother, habits of the mother, health of the mother before and during pregnancy, stillbirths and syphilis, and sequence of pregnancies, has been examined, with results that confirm generally existing views as to the influence of these various factors on maternal mortality.

3. The comparative maternal death rates from all causes and for the separate causes of death in the practices of doctor, midwife, district, and institution have been analysed, the factor of selection being eliminated by transferring the case back to the original doctor or midwife in charge of the case, and it has been found that the death rate per 1,000 maternity cases is 2.8 in the practice of midwives, 6.9 in the practice of doctors, and 14.9 in in-patient institutional practice.

4. The evidence indicates that, in so far as midwife cases are concerned, the application of midwifery forceps has no prejudicial effect on the mother.

5. Statistical evidence has been obtained of the reduction in maternal mortality which accrues from the provision of ante-natal services.

6. There is reason to believe that the high incidence of puerperal sepsis in maternity institutions in Aberdeen is due to contagion.

7. The view is advanced that the higher incidence of puerperal sepsis in the practice of doctors as contrasted with that of midwives is also due to contagion, being dependent in considerable part on a streptococcal carrier condition in doctors.

8. It has been shown conclusively that the *Streptococcus haemolyticus* plays by far the most important part in the causation of puerperal sepsis.

9. It has again been demonstrated that the same type of haemolytic streptococcus can on occasion produce puerperal fever, scarlet fever, erysipelas, and pyogenic infections.

10. The evidence goes to show that an expansion and improvement of ante-natal services will result in an important reduction in maternal mortality.

11. The evidence supports the view that the development of a new midwifery organization in which midwives conduct all normal deliveries, and in which doctors provide the ante-natal services and deal with obstetrical complications, will result in a significant reduction in puerperal mortality.

12. It has been shown that if the risk of contagion in maternity hospitals is to be restricted, then the number of patients and their spacing in the wards must be rigorously controlled.

* The opening paper of a discussion in the Section of Public Health at the Annual Meeting of the British Medical Association, Manchester, 1929.