Clerical work may be classified as follows:

1. Correspondence with A.D.M.S.
2. Correspondence with field ambulance and battalion headquarters. This includes (*a*) sick report for every sick man sent to a field ambulance; (*b*) tallies for all wounded; (*c*) indents for stores.
3. The Regimental Aid Post Diary, recording the diagnosis, treatment, and disposal of sick men dealt with by the medical officer; and the Sanitary Diary, which should be handed in daily to the orderly room for inspection and initials of the commanding officer.
4. Other records include (*a*) nominal rolls of all officers and other ranks in the unit who have had dysentery or enterica, and of all cooks and men employed in the handling of the food supply. These are kept in order to trace "carriers." (*b*) Record of inoculations. (*c*) Notes of all self-inflicted and accidental wounds, which should be made at the time the medical officer sees the case.

The foregoing sketch should, we think, not only give the young surgeon a clear conception of his importance in the chain of medical responsibility in war, but demonstrate to other readers that the unit medical officer is the keystone of the arch on which was built up the finest service which ever served with an army in the field.

ON AUTO-HAEMAGGLUTINATION:

A CONTRIBUTION TO THE PHYSIOLOGY AND PATHOLOGY OF THE BLOOD.

BY

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PART II.—CLINICAL.

WE have already seen in Part I that shed human red blood cells and those of the sheep, guinea-pig, and other animals when standing *in vitro* under suitable conditions pass through certain degenerative changes during which they form non-specific agglutinogens, and thus become agglutinable by their own native serum and by foreign serums with which they do not react when freshly shed.

We shall now proceed to consider certain tendencies on the part of the red cells to become more agglutinable during life as the result of disease. This change can be recognized in freshly shed blood by the same auto-agglutination reaction as that previously considered in Part I. (BRITISH MEDICAL JOURNAL, December 18th, 1920.)

The Wassermann Test in Relation to Auto-agglutination.

In order to test this point a routine examination was made of 500 samples of blood from 500 consecutive patients attending Dr. Mackarell's Pathological Department at the Leicester Royal Infirmary for the Wassermann test. The result from the haemagglutinative point of view is given in the following table:

No. of Cases, 500	Positive 231	+ auto-agglutination	30 = 13 %
		Trace reversible auto-agglutination ...	51 = 22 %
		− auto-agglutination	150 = 65 %
	Negative 269	+ auto-agglutination	16 = 7 %
		Trace reversible auto-agglutination ...	43 = 18.5%
		− auto-agglutination	210 = 91 %

Thus we see that in 500 samples of blood taken from 500 different individuals, of which 231 were found to be positive and 269 negative to the Wassermann test, 13 per cent. of the positive cases showed well-marked auto-agglutination, as against 6 per cent. of the negative cases. A trace of auto-agglutination was present in 22 per cent. of the positive, as against 18.5 per cent. of the negative cases. On the other side 91 per cent. of the negative cases gave no evidence of auto-agglutination, as against 65 per cent. in the positive cases.

The clotted blood was used in each case. A drop of the clear serum was placed on a slide. A fine-pointed pipette was then thrust into the centre of the clot and, by gentle suction movements, a drop of the concentrated red cell suspension was withdrawn and added to the drop of serum. These were mixed together with a glass rod and allowed to stand for a few minutes, then gently agitated and examined with a hand lens. A well marked non-reversible agglutination of the red cells occurring within a quarter of an hour was recorded as a *plus* reaction. A slight degree of agglutination, which disappeared on agitation and reappeared after standing and renewed agitation, was recorded as a *reversible* trace of auto-agglutination. An entire absence of clumping was recorded as a *minus* reaction.

These results were controlled in some of the *plus* reaction samples by washing the red cells in normal saline before mixing them with the serum. The results were the same as in the unwashed cells. A review of these findings shows that although the proportion of cases which gave a marked auto-agglutination reaction was nearly double in the positive as compared with the negative cases, and the numbers in the "trace" heading and in the *minus* heading were in like proportion, there is no constant association between a syphilitic's blood serum and auto-agglutination of the red cells by that serum. On the other hand, a careful investigation into the history and other accessory factors in both negative and positive cases soon suggested that auto-agglutination of red cells by native serum occurred for the most part in those blood samples in which either the positive reaction was high (indicating a marked reaction), or in which some ulcerating secondary focus could be demonstrated which afforded a basis for infection by pathogenic organisms.

As we shall see later, it is this presence of an infective focus and the defensive reaction made by the organism to the bacterial toxins formed there which accounts for the auto-agglutination in those blood samples which gave a negative Wassermann test.

This examination of these 500 samples of blood from different individuals, all coming under medical observation for some form of ill health, showed that a well-marked auto-agglutination was present in about 9 per cent. of all the cases, and a slight degree or trace of the same reaction in nearly 19 per cent.

The marked auto-agglutination present in some of the syphilitic samples must therefore be regarded as an indication of a reaction by the body against invasion by the spirochaete of syphilis, in common with the reaction made to other forms of infection in negative as well as in positive cases.

A number of control observations carried out on the blood of presumably healthy individuals showed for the most part an absence of the auto-agglutination reaction in the freshly shed blood. In a few cases in which the reaction was present in slight degree and in the "reversible" form careful inquiry revealed a previously unrecognized focus of infection, such as the presence of oral, nasal, intestinal, or genito-urinary sepsis.

An examination of the blood of a large number of different samples of the population on these lines would, I feel sure, reveal facts as to the prevalence of infection and other forms of ill health which remain at present unrecognized.

Pneumonia.

A series of cases of acute lobar pneumonia of pneumococcal origin were next examined. A few drops of blood drawn from the finger into a small Wassermann tube were either defibrinated by whipping or, generally, allowed to clot. A drop of the serum so obtained was then placed on a slide and mixed with the red cells obtained from the clot by the method described in the case of the Wassermann test samples. In all the cases of acute pneumonia so far examined (generally about the fifth day of the disease) a well-marked auto-agglutination of the red cells by the native serum was obtained. This often amounted to + + degrees, and was non-reversible in character.

Although I cannot at present state at what period of the disease the reaction first becomes recognizable, repeated examinations of the blood of pneumonia patients at recurring intervals after the attack have shown that the blood condition does not return to the normal after the crisis has taken place and the temperature has become normal. The auto-agglutination reaction persists for two or three months in most cases. It gradually diminishes in intensity and passes in reverse order from the + + through the "reversible" and "trace" stages to the "absent" stage.*

* Since this was written I have had an opportunity of testing the blood in two of the pneumonia cases after an interval of nine months, both patients being now in good health. In both the blood has lost the auto-agglutinable character present during and after the illness, though the power of specific agglutination of red cells from an individual of another blood group remains unimpaired. In another case of a patient with a positive Wassermann and a suppurating focus of secondary infection in the leg, three + + + degrees of auto-agglutination were present in January, and when examined in October auto-agglutination was absent, the blood had returned to normal, and the suppurating focus was healed.

This delay in the return of the red cells to a normal condition of stability, as shown by the absence of auto-agglutinability, may probably be associated with the duration of life of the individual red cells.

In young children the return to a normal blood condition after pneumonia is more rapid than in the adult, possibly because the renewal of the cellular elements in the blood is more active in early life.

The question also arises whether the rise and fall of this auto-agglutinative cycle in the red cells in certain diseases affords any indication of the duration of the immunity conferred by attacks of those diseases. The facts do not seem to be sufficiently definite to warrant any conclusion on this head at present.

The reaction has also been obtained from the freshly drawn blood in cases of pneumococcal empyema. Also in a patient with typhoid fever on the fifteenth day of the disease. A month later, when the temperature was normal, the reaction was still present, but in the reversible form, and a month later still, when the patient was convalescent, it had disappeared.

It has been obtained in cases of cancer of the uterus, in which a secondary infection of the ulcerating surface with offensive discharge was present, and also in a number of cases of infective endocarditis. It was demonstrated in a marked degree in one fatal case of cancrum oris, also in a case of septic abscess of the neck. The most marked reaction occurred in a man suffering from purpura, the end stage of mediastinal growth with liver metastases and secondary infection.

The essential factor in all these cases seems to have been microbic infection in some form or other.

The following record illustrates the sequence of events in a patient with a chancre and suppurating buboes, the result of a secondary infection:

When examined at the beginning of the illness the blood of this patient gave a positive Wassermann reaction and + + auto-agglutination in the freshly drawn clotted blood. The blood serum gave + + + degrees specific agglutination with human (C. J. B.) red cells, and the red cells were agglutinated + + with the same human (C. J. B.) blood serum.

Five months later, when the buboes were soundly healed and the blood, after antisyphilitic treatment, gave a negative Wassermann test, auto-agglutination of the patient's red cells was only present in the reversible form, and a trace only of agglutination was produced by C. J. B. blood serum, while the specific agglutination of C. J. B. red cells by the patient's blood serum remained + + as before.

In simple anaemia from haemorrhage no increased auto-agglutinability of the red cells has been observed. This fact is of interest in connexion with the results obtained by Warburg and Morawitz (quoted by Bancroft, *The Respiratory Function of the Blood*). These observers found that in phenylhydrazine and in haemorrhagic anaemia the metabolism of the blood was increased as compared with the blood of normal animals. They attributed the increased oxygen exchange to the large number of young erythrocytes present in the blood under these conditions.

We have already seen in Part I that an increase in the agglutinability of the red cells is an indication of age rather than youth in these cells; hence we should not expect to find it in cases of anaemia due to haemorrhage, in which the relative proportion of young to old red cells would be high. On the other hand, where the anaemia is the result of red cell destruction from septic poisoning, and where bacterial toxins are circulating in the blood, in such cases increased agglutinability of red cells, as shown by the occurrence of the auto-agglutination reaction in the shed blood, is also found. It may happen that in patients profoundly ill from anaemia of septic origin the auto-agglutination reaction may be absent in the blood shortly before death.

Differences in the Agglutination Reaction in the Blood from Different Parts of the Body.

A sample of blood was tested from a ligatured portion of a varicose vein removed by operation. The blood was allowed to remain in the "living test tube" of the vein for some hours, and was still fluid when removed, but coagulated in five minutes after withdrawal. The red cells with the native serum gave a slight auto-agglutinative reaction. This was absent in the blood from the general circulation of the same patient.

Samples of blood taken immediately after operation from veins leading from thyroid adenomata and fibroids of the uterus have also been tested for the agglutinative reaction in comparison with samples from the general circulation of the same patients. In many of the cases examined blood taken from a vein after circulating through the tissues of the growth showed a definite auto-agglutinative reaction. This was absent, or present only in the reversible form, in blood drawn from the finger of the same patient.

When a cross test is made between the washed red cells from the general circulation and serum from the blood which has circulated in the tumour and vice versa, it is found that the increased auto-agglutination is due to an increase in the agglutinin content of the serum, and not to an increased agglutinability of the red cells in the tumour blood. This is shown in the following scheme:

Auto-agglutinative Reaction.

Systemic blood – auto-agglutinative.	Tumour blood + auto-agglutinative.
Systemic washed red cells with Tumour blood serum + agglutinative.	Tumour washed red cells with Systemic blood serum – agglutinative.

Thus the red cells of the blood which has circulated in the tumour are not agglutinated by blood serum from the general circulation, but they are agglutinated by serum from the tumour blood. In the same way red cells from the blood from the general circulation are agglutinated by tumour blood serum but not by systemic blood serum.

Blood serum from the general circulation and blood serum from the tumour were also tested with the same sample of sheep's washed red cells. The systemic blood serum failed to agglutinate these while the tumour blood serum gave a definite agglutination.

It is clear from this experiment (which has been repeated in several cases) that blood which remains for some time in stationary contact with tissue cells, and especially tumour cells, undergoes some change, which can be recognized in the shed blood as an increase in the haemagglutinative capacity of the blood serum. This change takes place more rapidly and more completely in blood which has been in intimate contact with the cells of a new growth. The increase in agglutinative capacity is due to an increase in the amount or activity of the non-specific auto-agglutinin A and not to increased activity of the specific agglutinins B, C, D, etc., previously present in the blood.

The fact that blood serum after prolonged contact with tissue cells undergoes a change whereby the agglutinin content of the blood (when shed) is increased, would seem to be a matter of some physiological importance. It may be asked, If contact with the tissue cells has this effect on the blood, why does it not happen that the whole volume of blood in the circulation becomes increasingly charged with haemagglutinin? It must be remembered, however, that under normal conditions the blood is not in stationary contact with the tissues, but that any given volume of blood is in fairly rapid movement.

Whether anti-haemagglutinins are present in the living circulating blood, or whether the renewal changes which take place when the circulating blood is exposed to the oxygen of the air in its passage through the lung capillaries, or whether other influences exerted on the blood by organs like the liver and spleen prevent this overloading with haemagglutinin—these are matters for further inquiry. The fact, however, remains that blood which has remained in prolonged contact with the tissues does show this change, while blood from the general circulation, which has passed through the lungs, liver, spleen, and other organs, does not show it.

We have seen that the effect of stasis and contact with tissue cells is to increase the haemagglutinin in the blood serum rather than the haemagglutinogen in the red cells. There is reason to think, however, that, if the stasis and contact with the tissue cells is prolonged for some hours, then the red cells also become more agglutinable, because red cells exposed to such conditions are, even when washed, more readily agglutinated by foreign blood serums with which, when freshly drawn from the general circulation, they give no reaction.

It would seem that a lapse of time is necessary both *in vivo* and *in vitro* in order that increased agglutinability of the red cells may take place, whereas an increase in the haemagglutinin content of the blood serum can occur

rapidly as the result of shorter contact with tissue cells. This increase in agglutinin content affects the non-specific auto-agglutinin A, and not the specific agglutinins naturally present in the blood serum. This suggests that the liquid portion of the circulating blood obtains from the body cells (or from some of them) its charge of non-specific agglutinin capable of reacting with the agglutinogen A formed by the red cells, and it would seem that this reaction may represent an attempt at some adjustment between the red cells and their fluid environment, the blood plasma. Hence, both on its own account and as an indication of other collateral blood changes which occur in disease, it deserves further and closer study.

Lymph and other Transudates in Relation to the Haemagglutination Reaction.

I have already shown (see BRITISH MEDICAL JOURNAL, June 14th, 1919: "The physical state of the blood serum in relation to its agglutinin and antibody content") that certain transudates, especially tissue lymph, contain less specific haemagglutinins than the blood serum of the same patient. This is also true of the non-specific agglutinin A. An examination of the lymph flowing from a Southey's tube introduced into the subcutaneous tissue of the leg in a case of cardiac dropsy showed that this fluid gave no auto-agglutinative reaction with washed native cells, while the blood serum from the same patient gave a marked reaction with these cells.

We have already seen in Part I that blood serum is deprived of both its non-specific and its specific haemagglutinins by passage through a porcelain earth filter. It is interesting to find that blood which has filtered through the capillary wall and blood which has filtered through an earth filter candle both provide a good medium in which red cells can develop agglutinogen. There is, however, this curious difference, that whereas blood plasma which has percolated through the capillary wall loses its specific and its non-specific haemagglutinin (as it does also when filtered through an earth filter), blood plasma which has remained in contact with tissue or tumour cells—provided that it has not left the blood vessels—gains in non-specific haemagglutinin content. Can these facts be harmonized?

In the first case the transuded blood plasma or lymph has actually passed through the capillary wall and then lies in the intercellular spaces, in direct contact with the tissue cells. We do not know the relative share taken by the endothelial cells and the tissue cells in the reduction of the agglutinin content of the lymph. On the other hand, while blood obtained from a vein leading from a tumour has not passed through the capillary wall, it has remained in prolonged physiological contact with the tumour cells, separated only from them by the endothelial cells of the capillary wall. Some interchange takes place between the blood and the tumour cells, the result being that the blood serum gains in non-specific agglutinin, and, later still, if the stasis is prolonged, the red cells increase in agglutinability.

The stasis and the prolonged contact between blood and tumour cells brought about by the clamping of the blood vessels during the operation for the removal of the tumour accentuates the interaction which goes on under normal conditions between the circulating blood and the tissues. Under normal conditions we do not discover any haem-agglutinative indication of this interchange in blood drawn from the general circulation, possibly because this systemic blood has passed through the lungs, liver, spleen, and other organs.

An examination of the haemagglutinin content of the venous blood flowing from each organ of the body might provide valuable information as to the qualitative and quantitative nature of the interchange between the blood and the tissues in so far as the auto-haemagglutization reaction is concerned. The fact, however, would now seem to be established that blood which leaks through a capillary wall loses in haemagglutinin content, while blood which does not pass out of the vessels, but which remains in sufficiently prolonged physiological contact with tissue and tumour cells to allow of the interchange taking place, gains in haemagglutinin content.

The Age Period at which Haemagglutinins appear in the Blood Serum and Haemagglutinogens in the Red Cells.

In 1918 I was able to trace the development of haem-agglutinin in the blood serum of a full-term infant (removed from the womb by Caesarean section) at recurring intervals up to 12 months of age. The blood serum from the umbilical cord (that is, on the foetal side of the placenta) did not agglutinate my own washed red cells. The mother's blood serum and the liquor amnii strongly agglutinated these cells. The infant's red cells were not agglutinated by my blood serum, but were agglutinated by the blood serum of another individual belonging to another blood group. Thus the red cells of this infant at birth already possessed some capacity to form specific agglutinogens. They were not agglutinated by the mother's blood serum, neither did the infant's blood serum agglutinate the mother's red cells. The infant was breast-fed by the mother. At the age of 6 months the baby's blood serum gave a trace of agglutinization with my red cells, a + agglutination with sheep's red cells, and a trace with the mother's red cells. Thus the infant's blood serum at 6 months had developed some agglutinins of a specific kind. At the age of 12 months the baby's blood serum strongly agglutinated my red cells, also the mother's red cells.

In animals, as in the human infant, the blood serum at birth is deficient in specific agglutinins. An examination of the blood serum of a number of lambs up to the age of 3 months showed a marked absence of agglutination when tested with human and guinea-pig's washed red cells. Thus of fourteen lambs only two of the older ones gave any agglutination with guinea-pig's red cells, although, as we have previously seen (Part I), the blood serum of all full-grown sheep agglutinates these cells. The washed red cells of all the fourteen lambs, on the other hand, gave some degree of agglutination with human (C. J. B.) blood serum. Thus, as in the case of the human infant, the red corpuscles of the blood of the lamb elaborate agglutinogen before the blood serum contains agglutinin.

The age at which the red cells of different animals and birds develop non-specific auto-agglutinogen and the blood serum forms non-specific auto-agglutinin is a problem which requires further investigation. The presence of the auto-agglutination reaction in disease in animals in which the conditions can be controlled will probably shed light on the same problem in the human subject.

W. M. Happ (*Journ. Exper. Med.*, March, 1920) has made an exhaustive inquiry into the iso-agglutination reaction in the blood of a large number of infants, from which he concludes that the blood group to which different infants belong is not established till 12 months, and is not finally determined till two years of age. He also finds that the blood grouping is established in the red cells before the serum—that is to say, the red corpuscles acquire agglutinophilic receptors before the serum acquires agglutinin. Happ also concludes, as I have also found, that iso-agglutinins are present in the mother's milk in harmony with the blood group to which the mother belongs.

Do Haemagglutinogens and Haemagglutinins Exist in the Living Circulating Blood?

One important problem still remains for discussion. Up to the present we have only described the occurrence of the haemagglutination reaction in shed blood—that is, in blood plasma which has passed through the coagulation phase and has been converted into blood serum. The following observations show, however, that the haemagglutination reaction may occur with blood plasma before it has been changed by coagulation.

First, in regard to the specific agglutinins and agglutinogens. Two samples of blood from a human individual whose blood serum was known to agglutinate with my own red cells were taken. Sample (*a*) was received direct into a paraffin coated tube, standing in ice-cold water; this was rapidly centrifuged and the plasma immediately tested with my own (C. J. B.) washed red cells: ++ degrees of agglutination were recorded. Sample (*b*) was allowed to clot, and the serum from this sample gave a like amount (+ + agglutination) with the same (C. J. B.) red cells.

This comparative test between plasma and serum was repeated with sheep's washed cells, previously ascertained not to agglutinate with this particular blood serum. No agglutination occurred with plasma or serum.

This experiment shows that plasma and serum both contain the same kinds and amount of specific haemagglutinins, and that the change brought about in the blood

by coagulation does not apparently affect the specific haemagglutinin content.

In order to ascertain whether the same is true in the case of the non-specific auto-agglutinin—agglutinin A—the blood from a patient known to be strongly auto-agglutinative, as the result of infection, was drawn into a cold paraffin tube, rapidly centrifuged, and the plasma tested with the native red cells. Auto-agglutination occurred with the same rapidity and nearly, if not quite, to the same degree with the plasma as when the blood serum of the same patient was added to the same native red cells.

Although the occurrence of specific and non-specific haemagglutinin in the blood serum is thus evidently not the result of coagulation changes in the blood plasma, since they are present in the plasma before coagulation takes place, the question still remains whether haem-agglutinins exist as such in the living uninjured blood while it is still circulating in the body.

It is difficult to believe that non-specific auto-agglutinin is present in the free state in the circulating blood (even in disease), for if so, unless some mechanism exists by which the agglutinating effect of this substance on the native red cells is prevented, clumping of the red corpuscles would occur during life. We must remember, however, that the blood is a fluid of which the physical and chemical equilibrium is easily upset, and that the mere withdrawal of the blood from the blood vessels may injure it sufficiently to bring about a regrouping of its chemical and physical aggregates, and other changes which may be recognized as an alteration in the haemagglutinative reactions in the blood when shed.

The subject is one of fundamental importance, because that which is true of haemagglutinogens and haemagglutinins may probably be also true of other bodies in the blood stream which play important parts in the immunity reaction, and in the defence of the body against infection.

I am hopeful that a future investigation into the behaviour of haemagglutinins in the living blood may throw further light on immunity problems.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

A CASE OF IDIOSYNCRASY TO NOVARSENOBILLON.

THE following case of idiosyncrasy to novarsenobillon seems worthy of record.

The patient, a youth of 18, of moderately good physique, but somewhat anaemic, was admitted into hospital for treatment of syphilis on May 12th, 1920. Four small ulcers were present on the glans penis and skin of the prepuce; there were condylomata around the anus; inguinal and cervical adenitis were present, as well as ulceration of the tonsils. The organs appeared healthy, but the urine contained a trace of albumin. *Spirochaeta pallida* was recovered from the sore by the dark-ground illumination method, and the Wassermann test was positive. On May 14th 0.45 gram of novarsenobillon dissolved in 10 c.cm. freshly distilled water was given intravenously, and intramuscularly 1 grain of mercury in the form of a cream. These doses were repeated on May 22nd, when the patient looked quite well, the sores were healing, and the albuminuria had disappeared. He continued to appear well until the early morning of May 25th, when, after a report that the patient could not be roused, the orderly medical officer found him in a state resembling that following an epileptic fit. When I saw him at 10 a.m. he had just vomited and was unconscious. He was lying on his side with the legs drawn up; the pupils were widely dilated, but reacted to light; knee-jerks present, but exaggerated; no ankle-clonus; Kernig's sign and retraction of the neck absent. There was general hyperaesthesia of the lower extremities; also incontinence of urine; temperature 101° F., pulse 88; respirations normal. This condition altered little during the next forty-eight hours; there were no convulsions, and the patient could swallow milk placed in his mouth in small quantities. The bowels remained confined. Adrenalin solution mxv, three times a day, was given at first hypodermically and later by the mouth. Calomel also was administered. Lumbar puncture, on the 28th, showed clear fluid under high pressure, containing no globulin; the cell count was under 10, and the fluid was negative to the original Wassermann test. No growth was obtained from cultures made either from the cerebro-spinal fluid or from the blood. The calomel having acted, the motions were passed involuntarily. On May 27th the patient's condition was somewhat improved; the pupils were not so widely dilated, but he was still unconscious. There was no paralysis, but about midday he had a slight convulsion rather like an epileptic fit. The adrenalin was stopped and 160 c.cm. of blood was withdrawn by venipuncture. On May 28th the patient was much improved; the eyes were opened and the pupils were normal. He seemed to take notice, but did not speak until the next day, when the temperature was normal, the incontinence ceased, and he took his food well. On the night of May 30th he was restless and delirious. Subsequently continued progress was made; headache, paralysis, and other residual complications were absent. The blood pressure was not ascertained.

Fifteen other men were injected intravenously, on the morning of May 22nd, with novarsenobillon in doses varying from 0.45 gram to 0.75 gram; the conditions were precisely similar, yet none of these fifteen showed the slightest trace of reaction. The case recorded was the first reaction which caused me anxiety in over 5,000 injections of salvarsan and its compounds.

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CAESAREAN SECTION FOR ECLAMPSIA: MOTHER AND CHILD SAVED.

IN November, 1920, there was admitted to St. Mary's Hostel, Croydon, a young primipara, who expected to be delivered early in January of the new year. She had had a fit and did not feel well. I saw her about half an hour afterwards; she had a violent convulsion as I examined her, and became unconscious and continued convulsed until the time of operation about an hour and a half later. The urine was loaded with albumin, there was no sign of labour, the foetal heart could be heard. I feared that both mother and child were doomed unless Caesarean section was at once performed. I sought out my colleague, Dr. E. M. Cowell, who quickly and brilliantly operated. I anaesthetized with open ether, and after that the mother had no other convulsion; she made a quick recovery, although some albumin continued in the urine. The child, though small, did well.

If done in time, Caesarean section is a dramatic and successful way of dealing with eclampsia.

Croydon. G. GILBERT GENGE, M.D.Lond.

Reports of Societies.

TOXAEMIAS OF PREGNANCY.

AT a meeting, held on December 2nd, of the Section of Obstetrics and Gynaecology of the Royal Society of Medicine, the President, Professor HENRY BRIGGS, being in the chair, Mr. CLIFFORD WHITE read a paper on sodium bicarbonate tolerance in the toxaemias of pregnancy. He reviewed Sellards's work on bicarbonate tolerance as a proof of the presence of acidosis, and pointed out the simplicity of this compared with other means of demonstrating the condition.

The patient had 1 drachm of sodium bicarbonate by mouth every three hours, the urine being tested by litmus before each dose. The number of drachms administered before the urine became alkaline was noted; if more than 2 drachms (8 grams) of bicarbonate were required, the existence of some degree of acidosis was indicated. The greater the quantity required, the greater the degree of acidosis. The test was simple, harmless, and apparently reliable.

In a series of patients tested in this way Mr. White had found that cases of pregnancy toxaemia that were clinically severe had a larger tolerance than the mild cases. In a severe case of eclampsia 20 drachms might be required before the urine became alkaline. A series of normal puerperal cases were tested as controls.

Dr. LAPTHORN SMITH was sure that the value of sodium bicarbonate in the treatment of the toxaemias of pregnancy was underestimated. The urine of every pregnant woman should be examined at intervals, and if any albumin were found sodium bicarbonate should be prescribed, and the patient put temporarily on a vegetarian diet; she should also be instructed to drink at least six glasses of water a day. This simple plan would lead to a great diminution in the death rate from eclampsia.

Etiology of Eclampsia and the Pre-eclamptic State.

Dr. JAMES YOUNG and Dr. D. A. MILLER read a paper in confirmation of the explanation of eclamptic toxaemia