

this number to the number of spinsters at the same age in the Census year. Marriages under 25 years have steadily declined; marriages between 25 and 35 have perhaps held their own; marriages at other ages have varied in no very definite manner: on the whole, however, there are fewer marriages in the later than in the earlier period. The difference, however, is not very great, and if Körösi's figures of fertility may be taken as sound, as will be seen is the case, the change of age at marriage would only give rise to a decrease of 3 per cent. in the birth-rate, whereas the decline of fertility, as we shall see, is more than 27 per cent. The later age at marriage, therefore, is not a factor of any importance in decreasing the number of births.

Change in Fertility.

The manner in which the fertility of the married women between the ages of 15 and 45 has varied since 1860 will now be discussed. These changes can be very fairly estimated, though the number of births registered in the neighbourhood of the Census of 1861 is probably in defect of the actual number of births which occurred. In spite of this defect, however, I take the figures of the Registrar-General in the years in the neighbourhood of 1861 as the standard. The method of Sir Arthur Newsholme and Dr. T. H. C. Stevenson for calculating the fertility is applied. In this method the fertility rates at each group of ages among married women, as given by Körösi, are applied to the numbers of married women in these groups of ages during the epoch in question. The ratio of these rates to one another (after much work in this subject in different regions of Great Britain) represent, I think, very closely the relative fertilities at different ages. The birth-rates as investigated are calculated from the average number of legitimate births in the three years with the Census year as the mid-year from 1861 to 1911. It is found that if the fertility of 1861 be taken as the datum line and denoted by a hundred, that the fertility around 1871 is represented by 103; that around 1881 by 102, and that thenceforth the decline is continual until in 1911 it has fallen to 73. Now I have held and still hold that a large part of this fall in fertility is due to race physiology. The idea of race physiology is not in favour at the present moment, largely, I think, because of the neglect by physiologists of the larger aspects of life. In view of the great number of races and empires which have risen and fallen in history, the question as to whether there is a race physiology seems at least worthy of consideration. It has been advanced from the time of Aristotle, who says it is a law of nature that great men come in groups. The effect of deliberate restriction of the birth-rate must be admitted, however, to be of increasing importance, but I feel inclined also to consider this an aspect of race physiology; the refusal of responsibility is a mark of the decline in the vitality of the individual. The tendency to interfere with the processes of nature seems to recur at intervals in the history of society. The same neglect of the maintenance of the family is one of the charges of the Roman satirists of the empire, a change which was coincident with the decay of the empire, and to which it must be held to have had some relation.

The Physiological Aspect.

We now go from the statistical to the physiological aspect of the matter. On this I do not feel that I possess the kind of knowledge to enable me to dogmatise, but within the range of my own observation and from conversation with many medical friends I have seen and heard sufficient to render some things probable. When a young married couple deliberately for a number of years avoid having children, it is often found that if later the desire to have children arise, the capacity to procreate no longer exists. This position has been met by saying that the first pregnancy should never be postponed. With that statement I am in complete agreement, but at present the first pregnancy is in far too many cases among the middle classes postponed. Further, if family life is to exist with true social intercourse there should not

be too much difference between the ages of the children. I can quite well understand parents limiting their family to three or four children, but in that case the interval between the children would be preferably 18 months. The ideal that a woman should bear children at long intervals during 15 or 20 years is to me socially wrong. An only child is an admitted anomaly. Colonel Martin Flack, moreover, who has large experience in selecting the flying men for the Air Force, concurs in this; he tells me that the only child is hardly ever of use for flying. Children born at intervals of four or five years, however, are practically "only" children and in place of one anomaly several are produced. That, however, this intermittent kind of fertility exists is a pure assumption. It is highly improbable that parents who considered the production of such multiple anomalies desirable would receive any encouragement from nature.

NIGHT SWEATS:

THEIR SIGNIFICANCE AND PREVENTION.

BY MARCUS PATERSON, M.D. DURH.,
M.R.C.S. ENG.,

MEDICAL SUPERINTENDENT, COLINDALE (M.A.B.) HOSPITAL,
HENDON, LONDON, N.W. 9.

I HAVE used the term "night sweats" at the head of this article, as they are the ordinary words employed to designate the symptom about which I am writing. They ought not to be called night, but slumber sweats, as the condition which brings them about occurs not only at night, but also during a morning nap or an afternoon siesta. As a medical student I was brought up to understand that slumber sweats in adults were diagnostic of pulmonary tuberculosis, and from my experience since then I think that the great majority of our profession still adhere to this view. It is not surprising that such a creed prevails to-day, when the medical dictionaries thus define this symptom:—Dorland: "Night sweats, a symptom of phthisis"; Lippincott: "Night sweats, as in phthisis and hectic fever," hectic fever being exemplified in the same dictionary as "peculiar to consumption." Lauder Brunton apparently held similar views, as he endeavoured to explain the origin of the sweats as being due to the exhaustion of the respiratory centre in the medulla, and the consequential accumulation of carbonic acid in the blood, which condition he contended stimulated in turn the sweat centre. As evidence for this view he pointed out that the "remedies for night sweats are respiratory stimulants."

As a matter of fact, there is no proof that the respiratory centre has anything to do with these sweats, and all the evidence is against this statement. Not only do they occur in early cases of pulmonary tuberculosis, when there is no suspicion of dyspnoea and no excess of "carbonic acid in the blood," but they occur at a period of the disease when dyspnoea is not present, whilst the patient is in bed, and they disappear when dyspnoea is observable. Moreover, they occur in other diseases, such as rickets and Malta fever.

Night Sweats in Malta Fever.

Staff-Surgeon C. C. Godding, R.N., writing in 1891¹ on an analysis of 42 cases of Malta fever, refers to the profuse night sweats as one of the symptoms of the disease. He also makes the interesting statement that Malta fever was known at that time as "sweating fever." The text-books do not, as far as I am aware, associate this symptom of Malta fever with slumber. Dyspnoea is not one of the usual symptoms of Malta fever; slumber sweats do constitute a usual symptom. Slumber sweats are not usually associated with the intense dyspnoea of advanced emphysema and heart disease, neither are they prevalent during the acute dyspnoea of a spon-

¹ Brit. Med. Jour., 1891, i., 1065.

taneous pneumothorax or of an acute pleural effusion. The fact is that in an uncomplicated case of advanced pulmonary tuberculosis, dyspnoea, whilst a patient is in bed, is very uncommon, except just prior to the terminal event, and even then the orthopnoea of pneumonia is practically never observed. And as it will be stated later, the sweats do not occur for three weeks before death—i.e., when dyspnoea is present. Slumber sweats should not therefore suggest a respiratory disease, but should be associated with microbic action—i.e., toxæmia.

Night Sweats in Pulmonary Tuberculosis.

The sweats in Malta fever occur with the onset of the acute attack, and as the patient recovers they cease. In pulmonary tuberculosis they may occur quite early in the disease, even when no fever has been detected. As far as I am aware, little attention is paid to the sweats of early tuberculosis, for the reason that they practically always pass away without treatment. Indeed, many patients have sweats during the early days of their ailment without at the time mentioning the fact to their medical attendant. These sweats are, however, usually at their worst at a late period of the malady, when the patient is more or less febrile and confined to bed. It is well to emphasise here that they can occur when there is no fever and no dyspnoea. They do not occur when a patient is dying; they cease spontaneously without treatment about three weeks before death. By slumber sweats, sweats are meant that wet a patient's night attire, not slight sweats around the head and neck. The facts are therefore that these sweats:—1. Occur during the early stages of pulmonary tuberculosis, and pass almost unnoticed. 2. That they are very severe, and have been difficult to treat at a late period of the disease. 3. That they are not present for about three weeks before death.

Slumber Sweats the Result of Auto-inoculation.

For these reasons I have come to the conclusion that these sweats are indicative of activity in pulmonary tuberculosis—i.e., auto-inoculations are taking place. In the early case, with presumably good resistance, the immunity is increased and a condition of balance is reached. In the advanced case the auto-inoculations are too severe and swamp all immunising responses. In the case of the dying man his resistance has disappeared and no immunising is possible. Auto-inoculations, as is well known, take place without any accompanying slumber sweats, it appears therefore to be probable that they only occur when a very great stimulus has been given to the production of antibodies, owing to the very large amount of bacterial products that have entered the blood, and it is quite possible that the inciting cause is the actual presence of bacilli in the blood.

To put the idea in the fewest possible words, a slumber sweat is indicative of a severe fight between bacterial products and antibodies. The facts that they occur in the early period of the disease and are of a very transient nature because of the higher immunity of the individual, and that in the later stages they are of longer duration because of the lowered immunity and the consequential breaking of the resistance of the body, very strongly supports the view which I have enunciated. This is corroborated by the fact that they cease in the last three weeks of the patient's life.

As has been stated elsewhere,² we have no simple test for discerning whether a patient has or has not active disease. To make this quite clear let me cite an example. A patient presents himself for examination for life insurance. On examination he is found to have dullness and a few crepitations at one apex and tubercle bacilli in his sputum, and to be free from fever. Is this an active case which therefore requires treatment, and the consequential enormous expenditure of time and money, or is it an arrested case which does not require treatment? To reiterate, we have at present no simple test for ascertaining

this extraordinarily important fact in an early case of pulmonary tuberculosis without fever. If therefore this theory prove correct, it will give a simple method of detecting active tuberculosis in the first stage of the disease—i.e., slumber sweats plus tubercle bacilli in sputum equals active tuberculosis. On making careful inquiry into the history of slumber sweats, it will be found that they are of a very much more frequent occurrence than is generally supposed. These sweats therefore, in a recent case of tuberculosis should indicate not only the necessity of immediate treatment (of the disease, not of the symptom), but of the need for absolute rest.

I have failed to find any very helpful reading in the literature on night sweats, as practically all references relate to their treatment by drugs. How useless the various forms of treatment have been may be inferred from the eulogies of the very numerous drugs which have been recommended, not one of which is a specific like, for instance, salvarsan. I have already³ recorded how common and severe were the night sweats when I first went to Brompton Hospital, and how with the advent of open windows they became much less frequent and severe. I can still remember how Sir James Fowler, before the days of open windows, used to smile at my efforts to control, by the use of many of the drugs mentioned in the pharmacopœia, the sweats of his patients, until one day he told me that I was only following the example of all my predecessors, with similar results.

Mechanical Factor of Production of the Sweats.

No medical man without practical experience of slumber sweats can appreciate what a terrible thing it is to suffer from them. I had treated hundreds of cases, but did not realise this myself until some few years ago the *Micrococcus melitensis* took possession of my body; I then endured agonies from such sweats, so that I did everything I could to refrain from slumber—of course, without avail—and I would wake even after a brief sleep absolutely wet through. Under such circumstances, it can be readily understood, my brain was not functioning at its best; but I did observe that the reason one sweated was because one lay still with one part of the body in contact for a long period with the mattress, and it was this that acted as a compress or poultice. This idea brought back to my memory the many tropical nights, spent in the service of the P. & O. Company, when I had slept in comfort on a grass mat over the mattress, and under the sheet. Fortunately, I had brought one home, and from the moment one was arranged on my bed the sweats disappeared. When a person is awake and there is a feeling of heat and sweating around any part of the body in contact with his bed, he moves on to another part of his body automatically, and so he does not break out into a profuse sweat, because he is frequently moving. When he falls asleep one part of the body remains in contact with the bed, because he is immobile, and so the slumber sweat is produced. We have now at Colindale (where we have many severe cases of slumber sweats) been using similar mats for many months, and the results fully confirm my own personal experience. The only case in which after several nights' use I have known a mat fail to cure the sweating was relieved by taking away the mattress and placing the patient on a piece of canvas stretched between the head and foot of the bed. Whatever therefore be the factor, to be referred to in a moment, in the body that makes a slumber sweat possible, the actual production is the mechanical contact of the body with the mattress. To understand this, it is only necessary to sleep on a mat during a hot night, when all the heat and discomfort caused by a mattress will disappear.

Movement of Air to Counteract Insufficient Loss of Heat.

To consider the factor in the body that causes these sweats, the primary stimulus to the "sweat centre" is obviously insufficient "loss of heat," and this has been

² Auto-inoculation in Pulmonary Tuberculosis, p. 215.

³ The Shibboleths of Tuberculosis, p. 134.

shown by Leonard Hill to be rectified by movement of air. Hence the opening of windows diminish slumber sweats and the mats permit of the insulation of the body from the mattress which conserves the heat. The sweats may therefore be due to the toxins in the blood after an auto-inoculation stimulating the sympathetic, which is, as shown by Langley to be so, closely associated with the sweat glands, and so causing the sweats. The sequence of events being as follows: (1) patient lying still in bed because of toxæmia and feeling "rotten," and consequential deficient aeration of body; (2) heat loss therefore small; (3) smaller loss of toxin, because sweat evaporation is less than usual; (4) accumulation of toxin in body; and (5) stimulation of sympathetic and causation of excessive sweats. This theory supports the view already enunciated, as to why sweats do not occur in all cases where bacteria and their products are present in the blood-stream. The sweats only occur when there is a great amount of toxin in the blood. Obviously the more the toxin the quieter the patient.

Many imported mats have patterns worked on them; these kinds should be avoided because they leave a pattern on the skin and so cause discomfort. It should not be overlooked that these mats are chiefly used when a patient is in an advanced state of tuberculosis, and is very thin, and his skin very sensitive and liable to bed sores, so that the grass or straw must be fine. Probably a panama straw mat would be the ideal article for anyone who objects to the "hardness" of a Japanese mat. Patients some-

times disapprove of the mats at first because they feel hard; if such a criticism is raised, the mattress is often at fault. It should therefore be seen to, before ordering a mat, that the patient has a comfortable mattress. Once a patient is at ease on a mat it gives him relief almost at once. Mats will not prevent sweats if the patient is over-clothed either by personal or bed clothes, and before its use these matters should be gone into in detail. Neither will mats stop the sweats caused by the contact of the neck and head with the pillow; but these are not what is meant by slumber sweats. Messrs. Mayer and Phelps, of New Cavendish-street, stock suitable mats.

Summary.

1. Night sweats should be called slumber sweats, as they do not occur except when a person sleeps.
2. In early stages of pulmonary tuberculosis, and in the absence of any other infection, they are often the one indication of *active* tuberculosis.
3. Slumber sweats cease some weeks before death, because the patient's resistance has been overcome.
4. Slumber sweats can in nearly all cases be prevented without drugs by sleeping on a grass mat over the mattress.
5. Sleeping without a mattress on canvas will stop sweats.
6. The sweats are due to the presence of bacterial products (toxins) in the blood (in large quantities); they can therefore occur in any bacterial disease, and are not in consequence in any way diagnostic of pulmonary tuberculosis.

THE CALCIUM AND INORGANIC PHOSPHORUS CONTENT OF THE MATERNAL BLOOD DURING PREGNANCY AND LACTATION.

BY O. L. V. DE WESSELOW, M.B. OXF.,
F.R.C.P. LOND.,
CHEMICAL PATHOLOGIST TO ST. THOMAS'S HOSPITAL.
(A Report to the Medical Research Council.)

THOUGH a considerable number of observations are available on the calcium and phosphorus balance in pregnancy, comparatively little attention has been devoted to the content of the maternal blood in these elements. The demand for calcium is indicated by the fact that, according to Söldner,¹ the foetus contains at birth 20.4 g. of this element, while the intake of an ordinary diet amounts to 0.74 g. per diem²; in addition calcium is, of course, needed for the construction of the new-formed maternal tissues. Similar considerations apply to the phosphorus metabolism during pregnancy. The same conditions are also operative during lactation, when the parasitism of the foetus is replaced by that of the sucking infant. It

phosphate was determined by Lehman's modification of the Bell-Doisy method.⁴ Oxalated plasma, spun out as soon as possible after the blood had been drawn, was used for this determination in preference to serum, since Howland and Kramer have shown that the phosphate content of serum standing in contact with the clot tends to rise, owing to the large amount of phosphate present in red blood corpuscles. For the amounts of oxalate used, and for the modification of the original method employed, the criticism of Denis and von Meysenburg,⁵ who find that excess of oxalate seriously affects the ultimate readings, does not apparently hold. In the majority of cases the solids of the serum were also determined.

Since it was impossible to obtain patients in a fasting condition, all blood samples were taken between 11 A.M. and noon, to obviate as far as possible the effect of food upon the phosphate readings. As controls healthy female out-patients between the ages of 20 and 40 were used. The results are given in the table.

Normals.—The figures for the normals call for no comment. The inorganic phosphorus content shows a close agreement with the figures given by McKellips, de Young, and Bloor,⁶ who, using the nephelometric method, found an average value in the plasma of

TABLE SHOWING CALCIUM AND PHOSPHORUS CONTENT OF MATERNAL BLOOD.

| Description of case. | No. of cases. | Inorganic phosphorus as mgs. P per 100 c.cm. | | Calcium mgs. per 100 c.cm. | | Total solids per cent. | |
|---------------------------|---------------|--|-----------|----------------------------|-----------|------------------------|-----------|
| | | Average. | Extremes. | Average. | Extremes. | Average. | Extremes. |
| Normal controls | 17 | 2.99 | 2.15-3.98 | 9.9 | 9.3-10.5 | 9.48 | 9.07-9.91 |
| Pregnancy— | | | | | | | |
| 2½-5 months | 10 | 3.12 | 2.26-3.71 | 9.5 | 9.1-10.1 | 9.41 | 8.43-9.87 |
| 6th and 7th month | 9 | 2.76 | 2.22-3.43 | 9.1 | 8.7-9.8 | 9.03 | 8.48-9.96 |
| 8th and 9th month | 13 | 2.72 | 2.20-3.15 | 9.4 | 8.9-10.2 | 8.74 | 8.46-9.36 |
| Lactation— | | | | | | | |
| 3rd-11th day | 9 | 3.72 | 2.95-4.25 | 9.8 | 9.3-10.2 | 8.85 | 8.73-8.96 |
| 7th week to 14th month .. | 13 | 3.56 | 3.06-4.97 | 9.8 | 8.6-10.4 | 9.33 | 8.97-9.84 |

is therefore of some interest to determine how far the demand for these elements affects the calcium and inorganic phosphorus content of the maternal blood.

Methods.—The calcium was estimated in serum by the direct precipitation method of Kramer and Tisdall.³ All determinations were made in duplicate, and showed a satisfactory agreement. The inorganic

fasting adults of 3.1 mg. inorganic phosphorus per 100 c.cm., with an extreme range of 2.2 to 4.3 mg. The calcium figures fall within the usually accepted limits. Very few estimations of the solids of serum are apparently available, but Schmidt⁷ found 9.2 per cent. in human serum, and Hammarsten⁸ states that the solids vary from 6.7 to 9.7 per cent.