which adopted the use of masks seven months ago has had no case of haemolytic streptococcal infection (except one which was infected on admission) during the whole of this time, whereas previously such cases occurred with sufficient frequency to cause grave anxiety.

I conclude, therefore, that the spread of haemolytic streptococcal infection from the throats of carriers to women in labour is a factor in the etiology of puerperal fever which requires most careful investigation from every possible angle.

PROPHYLAXIS.

To those who are prepared to take prophylactic measures upon prima facie evidence, and before absolute proof is obtainable, I would make the following suggestions:

1. Masks of suitable material, completely covering the mouth and nose, should be worn by *all* persons present whenever the vulva is exposed during labour or the puerperium.
2. The occurrence of even a mild sore throat in the private home of an expectant woman should necessitate a careful bacteriological examination for haemolytic streptococci. If positive, search should be made for other domestic carriers. In any case, the patient's throat and vagina should be examined for haemolytic streptococci.
3. In institutions nurses and patients with even slight sore throats should be isolated from lying-in wards.
4. In institutions the first rise of temperature should necessitate an immediate vaginal or cervical test for haemolytic streptococci. If positive, the throats of contacts must be examined, and any carrier removed from the wards.
5. Doctors in private practice should have their throats examined at frequent intervals. If they have a case of puer-

peral sepsis, they should, for their own protection, have the throat of the patient and of all contacts examined.

6. It should be part of the duty of the Local Supervising Authority to have examinations of the throats of midwives who are concerned with cases of puerperal fever.

RECOMMENDATION.

There is enough evidence for the throat "carrier" theory to justify a full investigation. The time for isolated study has gone by, and some authoritative body should plan and control the research in selected areas. The full co-operation of the profession, and of the public health services would be essential, but, above all, the bacteriological work must be above suspicion.

The deductions made in this paper depend entirely upon the very expert bacteriological work which has been so ungrudgingly carried out in the department of bacteriology (Professor J. S. C. Douglas) by Dr. Edington in the University laboratories, and by Dr. Kirk in the laboratory attached to the Jessop Hospital for Women. The county bacteriological departments at Derby and Wakefield have also given most valuable assistance in the examination of contacts.

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SPONTANEOUS INOCULATION OF MELANOTIC SARCOMA FROM MOTHER TO FOETUS.

REPORT OF A CASE.

BY

F. PARKES WEBER, M.D., F.R.C.P., E. SCHWARZ, M.D., M.R.C.S.,

AND

R. HELLENSCHMIED, M.D.

(From the German Hospital, London.)

THE following is, we believe, the first reported case of transmission of a malignant neoplasm from mother to child by spontaneous inoculation.

The child, B. M., aged 8 months, a well-developed boy of normal size, was admitted to the German Hospital on November 19th, 1929. The patient's mother (D. M., aged 27 years) had died

Inferior surface of the liver showing melanotic tumours.

at the German Hospital on July 4th, 1929, of melanotic sarcoma with visceral and subcutaneous metastases. There was no post-mortem examination, but she was known to have been suffering from melanotic sarcoma for a long time previously. Eighteen months ago she had undergone operation at the London Hospital for melanotic sarcoma of the thigh. The child was delivered on April 9th by Caesarean section at full term at the London Hospital, and was thought to be quite normal at birth. Development seemed to be satisfactory till recently, when enlargement of the abdomen and a somewhat cachectic appearance of the skin was noticed.

On admission to the German Hospital the liver was found to be enlarged, reaching down to the umbilical level, and some large, hard, rounded nodules could be felt projecting on its anterior surface, suggesting malignant neoplasm. The spleen was likewise, but evenly, enlarged, its lower border (on palpation) being three fingerbreadths below the costal margin. Otherwise the child seemed fairly healthy and quite lively and happy at first, excepting that at times there was moderate fever. Nevertheless, after some time the child began to show increasing cachexia. The Wassermann and Meinel reactions were negative, and so was Pirquet's cuti-reaction for tuberculosis. The blood count (November 22nd) was: haemoglobin 76 per cent., erythrocytes 4,680,000 per c.mm. of blood, colour index 0.81, white cells 12,400 (polymorphonuclear neutrophils 32 per cent., lymphocytes 57 per cent., monocytes 10 per cent., eosinophils 1 per cent.); nothing abnormal was noted in the appearance of the red cells.

The liver and the bosses on it probably increased somewhat in size, but certainly only very slowly. About the middle of January, 1930, the child, who was gradually losing in weight and becoming more cachectic and obviously ill, developed considerable fever and left-sided purulent otorrhoea. The blood count on January 13th was: haemoglobin 60 per cent., erythrocytes 4,380,000 per c.mm., colour index 0.7, white cells 16,550 (myelocytes 4 per cent., metamyelocytes 10 per cent., polymorphonuclear neutrophils 36 per cent., lymphocytes 45 per cent., monocytes 4 per cent.). The pyrexia continued, and the child died on January 24th.

We may say here that the child's urine throughout, whenever examined, was clear and of a golden brown colour, sometimes deeper than at other times. It was free from albumin, sugar, indican, acetone, diacetic acid, and excess of urobilin or urobilinogen. The colour deepened when the urine was kept, but never became blackish. Only on one occasion (January 1st) was a supposed positive reaction for melanogen obtained—that is to say, on using the potassium nitroprussiate (Legal's) test for acetone we obtained a deep green coloration on addition of the glacial acetic acid, but this test is of doubtful value.

Necropsy (January 25th).—There were several minute subcutaneous melanotic nodules scattered over the front and back of the thorax of the emaciated child. The liver was enlarged (weighing 455 grams after being kept for some days in alcohol), and several melanotic rounded tumours (up to the size of a pigeon's egg) projected under the capsule, notably those that had been palpated during life in front of the liver. The spleen was moderately enlarged (weighing 100 grams), but was otherwise not

Lower portion of the upper surface of the right lobe of the liver, showing the melanotic tumours felt during life.

abnormal in appearance. There were a few minute subpleural melanotic patches in the lungs, and some small melanotic nodules were attached to the periosteum at the back of the anterior wall of the thorax. Many of the abdominal lymphatic glands showed melanotic infiltration; one or two of these glands were adherent to the kidneys and pancreas. Otherwise macroscopically the viscera appeared normal. We were not permitted to examine the head and central nervous system.

Microscopical Examination.—The melanotic tumour cells were typical in all the pieces of growth. The small nodule in the liver which we examined was found to be surrounded by a kind of spurious capsule, due to compression of the neighbouring tissue, and suggesting that the tumour growth had not been very rapid, but there were also melanotic tumour cells outside this spurious "capsule," and many of the Kupffer "Sternzellen" in the neighbourhood contained melanin granules. A piece of lung showed minute broncho-pneumonic areas, as well as a minute subpleural melanotic growth. A tiny subcutaneous nodule appeared to be encapsuled by the distended wall of a capillary blood vessel. Pieces of pancreas and kidney showed nothing abnormal, but a small lymphatic gland adjacent to the pancreas was infiltrated with the melanotic growth. A lymphatic gland removed from the right inguinal region was free from melanotic growth, and so was the piece of spleen examined. The slight splenomegaly was apparently due to increase of the splenic pulp. The Malpighian follicles of the spleen were not large, and contained very little "germ-centre."

Remarks.

The main facts of the case may be shortly stated as follows. A woman known to be suffering from melanotic sarcoma was delivered, three months before her death, of a child who appeared at first to be healthy. When 8 months old the child was admitted to hospital with an enlarged liver on which bosses could be distinguished by palpation, suggesting a malignant neoplasm. With increasing cachexia the child died at 10½ months of age, minute nodules having developed beneath the skin shortly before death. The post-mortem examination showed that the bosses which had been felt in the liver were melanotic tumours. The size of the growths suggested that the primary infection was in the liver, but there were many infiltrated lymphatic glands in the abdomen and minute metastases in the lungs and in the subcutaneous tissue. Palpation during life and examination after death pointed to the growth of the neoplasm in the liver having been rather circumscribed and slow, as if there was considerable resistance on the part of the child's tissues. The tumour cells from which the growths in the child's liver had developed had evidently been carried to the hepatic capillaries (via the blood stream in the umbilical vein) from the placenta, which is known to have been melanomatous.

The splenomegaly noted by us in the child may be compared to that which has been occasionally known to be associated with hydatid disease of the liver. The spleen of infants and children becomes readily enlarged from any toxic or distending cause. Thus, the spleen was enlarged in a boy with hydatid disease of the liver shown by A. F. Voelcker at the Medical Society of London in November, 1907. A certain amount of splenomegaly was still present in February, 1909, after successful treatment by drainage of the hydatid cyst, when Dr. Voelcker again showed the case.¹ In our case the following note kindly given us by Mr. Eardley Holland, who performed the Caesarean section on the mother at the London Hospital, makes it certain that melanotic tumour cells must have passed from the mother through the placenta (owing to actual melanotic metastases in the placenta) into the foetal blood stream. The blood in the umbilical vein having chiefly to circulate through the liver, some of the melanotic cells were caught up in the hepatic capillaries, and these cells were the parents of the multiple melanotic tumours (the "primary" tumours, as far as the child was concerned), some of which could be palpated in the enlarged liver when the child was eight months old.

This is the note by Mr. Eardley Holland, who is going to publish separately an account of the case from the obstetrical and gynaecological point of view.

"I saw the mother in consultation with her doctor on April 5th, 1929. She was an ill woman, in about the thirty-eighth week of pregnancy, with multiple melanotic nodules scattered liberally beneath the skin of the abdomen and thorax. The uterus was above the usual size at term, and a large soft mass occupied

the lower segment. On April 9th I delivered a live child by Caesarean section. The lower uterine segment was occupied by a huge black placenta, which proved to be infiltrated with masses of melanotic growth. The mother made a smooth recovery, and the child left the hospital thriving and without evidence of disease."

There are many remarkable published examples and different varieties of inherited constitutional predisposition to malignant neoplasms.² Especially notorious is the constitutional predisposition due to familial multiple polyposis of the large intestine.³ We would suggest that J. S. Manson's examples of the "hereditary transmission of sarcoma" (mother and two sons affected with lymphosarcoma)⁴ were examples of inherited constitutional predisposition to lymphosarcoma, and analogous to rare cases of familial tendency to lymphatic leukaemia,⁵ which, like myeloid leukaemia, is probably also of neoplastic origin. Our present case, as above stated, has nothing to do, as far as we can judge, with any inherited constitutional predisposition to malignant neoplastic growth.

It is, as mentioned above, apparently the first case of the kind reported in human pathology. We have not even heard of any analogous case in animals—that is to say, a case of congenital malignant tumour due to intrauterine inoculation of the foetus by the passage of malignant tumour cells from the placenta to the child by way of the umbilical vein. Nature's experiment in the present case could only be imitated in experimental animal pathology by inoculating the blood stream of the umbilical vein at the moment of birth with a dilute emulsion of tumour cells obtained from a sarcoma or carcinoma that had previously developed spontaneously or had been artificially produced in any part of the mother's body.

Professor J. A. Murray has, however, kindly referred us to a paper by Dr. Makoto Takahashi,⁶ who also quotes similar experimental work carried out by other investigators on rats or mice. Takahashi produced growths in the lungs by the intravenous injection of carcinomatous or sarcomatous material. He found that a large proportion of the cells introduced into the blood stream disappeared after undergoing vacuolation, surrounded by leucocytes. Although the lung capillaries formed an efficient filter some tumour cells were able to pass through the lungs and give rise to growths in various parts of the body. The cells which passed through the lungs and were disseminated in that way exhibited a predilection for a limited number of sites.

In regard to the last-mentioned "metastatic predilection" we might add that the author did not refer to the wonderful examples of the predilection in human pathology of metastases from particular tumours for particular sites—for instance, the predilection of metastases from the Hutchison type of suprarrenal medullary "sarcoma" (neuroblastoma) in children for the orbits and skull,⁷ and the generalized infiltrating metastatic osteogenic carcinomatosis of the skeleton occasionally resulting from a primary carcinoma of the prostate gland.⁸ Such examples might be compared with the secondary pyaemic foci limited to the bony skeleton, which formerly occasionally occurred in septic cases.

In regard to the way in which in our case the maternal melanotic tumour inoculated the foetal circulation by "growing through" the placenta, an analogy may be found in what happens in cases of true congenital tuberculosis, but, of course, tubercle bacilli are much less likely to be held up in the blood capillaries of the foetal liver than are large tumour cells. Some years after Koch's discovery of the tubercle bacillus it was maintained that the bacillus could not pass through the placenta and infect the foetus *in utero*, and it was held that only a constitutional predisposition towards tuberculosis could be inherited, and even that was denied by many authorities. However, it was soon proved that in very rare cases the foetus was actually infected *in utero* owing to the tuberculous process in the mother infecting the placenta, and producing one or more tuberculous lesions in the placenta. By means of such placental lesions the tubercle bacilli were enabled to "grow through" the placenta, with a resulting tuberculous inoculation of the foetal circulation and true congenital tuberculosis. It was alternatively but less convincingly suggested by A. Sitzenfréy, in 1909,⁹ that tuberculous foci, localized in the decidua vera, might penetrate the amnion, infect the liquor amnii, and thus give rise to intrauterine infection of the foetus—indeed, F. W. Andrewes¹⁰ already

in 1903 had suggested that pre-natal tuberculosis might in some cases possibly be caused by the foetus swallowing liquor amnii containing tubercle bacilli derived from a disintegrating lesion of the placenta. How rare cases of true congenital tuberculosis must be may be judged by the fact that in 1916 one of us was able to summarize all the hitherto proved and probable cases of true congenital tuberculosis in a short article published in the *British Journal of Children's Diseases*.¹¹

In our present paper it would perhaps have been better to use the term "malignant melanoma" instead of melanotic sarcoma, as it might possibly be suggested that the primary growth in the mother (the site of which is not certain) was a melanotic carcinoma. The tumours in the child's liver were obviously of multiple metastatic type, and it is the absence of any primary tumour in the child that makes the case unique, the real primary growth having been, of course, in the dead mother. The inoculation of the child may have taken place not long before, or even at, birth.

The liver, now in the museum of the Royal College of Surgeons of England, was demonstrated by us on February 28th, at the Section for the Study of Disease in Children, of the Royal Society of Medicine.

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APHONIA, DYSPHONIA, AND THE "SINGER'S ATTITUDE."

BY

SIR JAMES DUNDAS-GRANT, K.B.E., F.R.C.S., M.D.

At the last meeting of the Section of Laryngology of the Royal Society of Medicine I referred to my application to the treatment of functional aphonia of Mr. Negus's observation that in certain animals, particularly the climbing ones, the efforts made with the fore limbs were accompanied by an approximation of the vocal cords. Mr. Negus kindly placed at my disposal an apparatus containing a strong spiral spring, which resisted the pressure made by the hands on the two ends. The result was most satisfactory, as the involuntary approximation of the cords added a valuable additional factor in the restoration of voice. I found that in the absence of the spring-pressure instrument a similar result was obtained by making the patient press together my hands or those of a nurse standing behind her (or him). This procedure helped to restore the voice with less frequent resort to the distressing faradic stimulation which is sometimes inevitable.

In the treatment of singers suffering from functional difficulty in voice production with paresis of the internal tensors or transverse arytenoid muscles (elliptical glottis or posterior triangular gap) I have found this "arm-adduction" pressure most helpful. The following is a recent case in point.

Miss Y., aged 23, a contralto of considerable ability who had been studying singing for over a year, found that for some weeks her voice was husky and the "attack" uncertain. The laryngoscope showed imperfect approximation of the cords, the vibrating parts of the cords having an elliptical opening between them and the cartilaginous portion a triangular one. The "arm-adduction" against resistance was followed by an approximation of the cords

to almost the normal degree, and with it a great increase in the certainty of the "attack." The confidence in herself, which is of such importance to a singer, was again established, and she was at once able to apply the appropriate general principles inculcated by her teacher with every success.

The "attitude" adopted by many singers in which the hands are held together as in prayer, or in holding a little book of the words, is in most cases probably a fashionable affectation, but it may become an extra help in producing approximation of the cords if the hands are not merely in contact but compressed together. I can conceive this application of the attitude described as being of great use to a singer. It should, however, be accompanied by the lower lateral costal method of breathing, which is, for singing purposes, probably the best.

Among other ancillary helps towards combating the perverse gaping of the glottis in functional aphonia I find induction of the "inspiratory phonation" of the vowel sound "ee" followed by the expiratory "aw" effective in spite of, or, perhaps all the more, on account of, the grotesque effect. Another adjunct is the application of "Bárány's noise-box" to the two ears while asking the patient such a question as he may be anxious to answer. If the noise is suddenly turned off while he is giving his answer he may be surprised at the discovery that he is talking in a loud or even shouting voice.

I am grateful to Mr. Negus for his masterly investigation, and am pleased to have given it immediate clinical application.

A NOTE ON DISSEMINATED SCLEROSIS.*

BY

FOSTER COATES, M.D., D.P.H.,
PHYSICIAN, ROYAL VICTORIA HOSPITAL, BELFAST.

DISSEMINATED sclerosis is one of the most common organic diseases of the nervous system met with in Northern Ireland, and apparently occurs more frequently here than in other parts of the world. In the Royal Victoria Hospital, Belfast, 728 cases of disease of the nervous system were treated in the wards during the past three years, this total including neuroses, psychoses, epilepsy, chorea, and neuritis; 106, or 14.5 per cent., of these were cases of disseminated sclerosis. I have analysed 100 cases, 40 of which were under my own observation. From this analysis I have obtained statistics regarding the sex, age, and occupational incidence, also mode of onset and relative frequency of certain symptoms. Doubtful cases were rejected, and the possibility of syphilis was invariably excluded.

ETIOLOGY.

Although the disease is stated to be equally common in males and females, I found that males were more frequently affected, the proportion being 3 to 2 (males 61, females 39). The onset is most common between the ages of 20 and 40; the average age at onset was 29, the youngest patient being 11 years and the oldest 59. Five children under 15 were affected, and three adults over 50. As regards occupation, two-thirds of the patients were town workers and one-third country workers; 10 per cent. were farmers. The hospital records show that in 1927, out of a total of 5,450 in-patients treated, 540, or 10 per cent., came from the country, and 157, or 3 per cent., were farmers.

This would seem to indicate that disseminated sclerosis was three times more prevalent in farmers and in people living in the country than among those dwelling in towns. To check this I analysed another series of medical cases (heart and kidney), and found, somewhat to my surprise, that the proportion of country cases was only 10 per cent. and farmers 2 per cent. From these findings there would seem to be no doubt that disseminated sclerosis is more common among those living in country districts. The occupational statistics which I have given do not seemingly support the contention that this disease is more common in wood workers.

Heredity seems to have no bearing on the incidence of infection. Even in families with a definite neuropathic history disseminated sclerosis does not necessarily show

* Condensed from a Presidential Address delivered to the Ulster Branch of the British Medical Association.