blood-clot. It was whilst under this difficulty that the thought of collecting blood from an artery, defibrinating it, and then introducing it into the portal system, occurred to him. He had considered it possible that some slight effect might be perceptible, but had not anticipated the strongly marked result which is producible. The amount of blood used has been from ten to eighteen fluid ounces. After the production of anæsthesia by chloroform, the blood was collected from the carotid artery, stirred in order to defibrinate it, strained, and then very slowly injected into a branch of the mesenteric vein. In one experiment, where half an hour had been employed in making the injection, the urine at the completion of the operation contained a notable amount of sugar, and half an hour later showed by analysis the presence of fifteen grains to the fluid ounce. In a second, the urine contained ten, and in a third fourteen grains to the fluid ounce when collected three quarters of an hour after the operation. The experiments were performed upon dogs, and in each case it had been ascertained that the urine was devoid of sugar before the operation.

Having noticed the effect which have been described from the injection of oxygenated blood into the portal system, it became necessary to ascertain positively that it was attributable to the oxygenated condition of the blood, and not to any other cause. To decide this point, an appeal to the counterpart experiment was made. Defibrinated venous instead of arterial blood was injected into a branch of the mesenteric vein, and upon each occasion where such an operation has been performed a negative result has been obtained.

With the evidence thus furnished, the conclusion may be warrantably drawn, that oxygenated blood in some manner influences the liver so as to lead to the production of glycosuria. It may be inferred that, contrary to the effect of venous blood, it promotes the transformation of amyloid substance into sugar. The suggestion naturally occurs, that what has been stated above affords an explanation of the glycosuria occurring after Bernard's puncture of the fourth ventricle and the various lesions of the sympathetic. Without any new agent being called in, sufficient is presented in the state of the blood to account for the production of sugar that occurs. By a vaso-motor paralysis affecting the vessels of the chylipoietic viscera, the blood will reach the portal system without having become dearterialised in its natural way, and in this state it has been shown by the experiments narrated to possess the property of acting within the liver in such a manner as to determine the production of glycosuria.

STATISTICS OF FEVER, ENDING IN RECOVERY AND IN DEATH, OCCURRING IN TWO THOUSAND CONSECUTIVE CASES OF LABOUR.*

By I. HARRINSON, F.R.C.S.,
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IN making my contribution to the now all-discussed subject of puerperal fever, I propose to do so from, as far as possible, a practical point of view; to follow the example so admirably set on the introduction of the subject at our last meeting. By practical, I mean as the cases occurred in practice, with all their, it may be, obscurity of origin, irregularity of progress, and, in some cases, unlooked for terminations. I shall not attempt to make things fit where there was no fitness; much less shall I draw a line, hard and fast, in their causes, symptoms, pathology or treatment. I shall give the cases as they were written at the time of their occurrence, with the remarks, if any, appended at the time, and draw inferences from them fairly apparent to you and to me. It cannot be expected that the earlier cases would be examined as closely and with the same light as the latter ones, but, as far as they go, they may be taken as truthful representations.

I need not insist in this place on the normal abnormality of a puerperal woman, specially on her system teeming with the effete products of an involuting uterus and the waste of parturient muscular effort, oppressed with the diverted stream of foetal nutrition, and charged with the detritus of an obsolete circulation, to which kidney implication may be added; nor will I insist on her susceptibilities, increased as they must ever be by the accidents of hæmorrhage, unhealthy surroundings, and mental conditions. It is a state *sui generis*, and only has a parallel, as Dr. Ord remarked to me a few days ago, in a system saturated with the material of gout. It is a state which renders her specially vulnerable to toxæmic impressions.

A good illustration of toxic susceptibility, on a grand scale, is afforded

at the present time by our new Pacific acquisition. There, the virgin blood of the Fijians, unacclimatised to zymotic influences, unprotected by previous attacks, and unprepared for any, is at once disordered by the invasion of the weakest of European foes—that of measles—which revels in its freedom from antagonism, and now is numbering its victims by the thousand.

I will take it for granted that puerperal fever is admitted to be a toxæmia, the septinous matter being introduced by contagion (contact) or by infection (any and every other mode of introduction). It is possible that this septinous material may be inoculated before labour; and it is pretty certain that infection, as of scarlatina or erysipelas, may be received at an indefinite time during pregnancy, and only develop itself after delivery.

I will not attempt to draw a line between the access, progress, terminations, lesions, etc., of the specific poisons, as scarlatina, erysipelas, and those induced by animal matter, fresh as after a *post mortem* examination, or more or less putrid, as from a dissecting-room, though I believe it is generally admitted that the fresher the subject greater the danger; or those arising from a bit of retained placenta, or a non-expelled clot; between differences in kind and differences in degree.

Undoubtedly there are differences, and each cause produces a different series of effects, which only require to be carefully classified to establish a differential diagnosis. This is the work yet to be done. It is possible, also, that a practitioner, having met with a number of similar cases dependent on the same cause or causes, as in one hospital or locality, might be led to believe that there was one special puerperal fever.

It is proved by the experiments of Dr. Richardson that septinous poisons may be diluted with water to a given extent, and yet remain active up to an extreme of dilution. This fact warns us how the smallest quantity of morbid matter may inoculate a puerperal woman. He proves, also, that the intensity of the poisonous matter is increased in the most marked degree, and in a manner that seems to defy computation, with the transmission of the poison through fresh series of animals. This fact explains why, when a practitioner has a succession of unfortunate cases, particularly if he redoubles his attentions to each, the last cases may be and are the worst.

It is reasonable to suppose that the risk both of contagion and of infection will be greater in some cases than in others, in the specific than in the non-specific; for example, in those arising from the inoculation of erysipelas than in that produced by the mere non-expulsion of a clot.

"It appears to us", says the *Lancet*, "that the history of puerperal fever proves that the disease can be communicated without contact in some cases, and by contact in all. It is not essential that the disease should be taken from one patient to the other; the original contamination of the fingers may give rise to the disease in a series of consecutive cases. I cannot decide how, when a septinous material is inoculated, the effects ensue; whether directly or whether by first inducing inflammation, from the product of which the virus is derived."

I may here state that to the more immediate effects of the introduction of septinous matter, either by contagion or infection, I limit the term septicæmia. Pyæmia is a secondary, or rather a tertiary, state, the product of phlebitis—one of the sequelæ of septicæmia. Blocking of the arteries—pulmonary, cerebral—by clots, local and migratory, must also be included among the dependencies of poisoned blood.

It is not easy to determine whether the dire effects of septinous poisons, seen in some instances, be dependent on the intensity of the poison, on the quantity introduced, or on the susceptibility of the individual to poison or to shock. Mr. Davis related, a few weeks ago, the case of a woman who died in half an hour from the sting of a hornet. It is well known what a very small amount of hæmorrhage from the uterus proves fatal in some cases. The greater protective power of four vaccine vesicles over one, demonstrates that quantity has some influence; some people catch everything; others take nothing. Practically, identity of cause does not produce uniformity of effect. Bear in mind Mr. Jeston's cases. A young woman nursed her father with erysipelas. She was seven months advanced in pregnancy. Two of her children had erysipelas, and, lastly, she was seized. Premature labour came on; puerperal fever supervened, and death occurred in seven days. The nurse who attended this woman was immediately afterwards seized with acute rheumatism and pericarditis, and died in seven days.

Among the things to be remembered in cases of puerperal fever are, that, though the origin may be erysipelas or scarlatina, it does not follow there should be the ordinary expression of either on the patient, nor does it follow that either the practitioner or the nurse introduced it. The patient may have been exposed unconsciously or recklessly to the poison of either, which, during her pregnancy, may have remained in a state of quiescence. A curious question arises, whether a woman in this state may not poison the fingers of the medical attendant at

* Read before the Pathological Society of Reading.

her confinement, so that he unwittingly may convey the contagion to another woman. I shall have to enumerate cases affording some ground for such a supposition.

It will, I think, be admitted that the nurse must, if previously unclean, have the opportunity of conveying contagion more frequently than the medical attendant. Let it be remembered that the examinations of the medical man may be few, that his fingers will be smeared with some unctuous sheathing, that the secretions of the woman are copious and protective, and that the process of labour rouses her to a pitch of excitement opposed to the imbibition of septiculous contamination. After delivery, the state is altered; the frequent ablutions, although with antiseptic fluids, give ample means for its introduction. For the future, the antecedents of the nurse must in every case of fresh engagement in her duties be rigidly supervised. It is evident that lacerations, ulcerations, etc., of the woman's structures may favour the inoculation, and that bruising may even furnish a supply of septiculous material.

The statistics will include cases of extreme irritation, originating in the undue retention of a small portion of placenta. Clots may produce the same effects. In ordinary cases, such occurrences should rarely happen. I have no instance of septicæmia in cases of abortion, in some of which the expelled ovum smells. I am aware of the lesser development theory, but I have much faith in the ability of Nature to accomplish her own processes without disaster.

What, then, is puerperal fever? I reply, fever occurring in a woman recently confined. I say simply fever; neither a fever, nor the fever. The causes are numerous, the effects various; all are referable, however, to one condition—that of poisoned blood. The first impression, it is true, may be on the nervous system, as from fright, etc.; but the consequences are disturbance of function and vitiation of product. We shall see this more clearly when we come to compare puerperal fever with the effects of cold. In asking what puerperal fever is, I exclude, of course, the state of reaction from the labour, the physiological condition, the tumult of the novel situation, the secretion of milk, and the troubles of lactation.

The fever in mammary abscess sometimes runs high in the earlier periods after labour, being generally, I believe, a local pyæmia, extending from a cracked nipple; in the later, a deposition from a more general affection. The same rule applies to pelvic complications.

It must next be asked, What are the symptoms of puerperal fever? I will answer this practical question in the words of an able article in the *Lancet* of January 30th, 1875.

"We are not going to be drawn into any fine definitions of puerperal fever. The state we have in view is that of a woman recently confined, who, after a shivering, or something that represents a shivering, has a quick pulse. Temperature 101—105 deg.; more or less pain in the uterine region, suppression of milk, distention of abdomen. Lochia deficient or absent; more or less wandering"; and, I would add, an expression of distress.

One state must be here noticed, which has been most unaccountably ignored by—as far as I have seen—all speakers in the recent discussion. I refer to "intestinal irritation", as delineated by Dr. Marshall Hall. In it we get all the symptoms above named, with one important exception: the expression is not altered; it is good.

I cannot now discuss that important point how loaded bowels block the chief avenue of that eliminative process, so imperatively needful in the puerperal woman, and in what a rapid ratio, if neglected, mischief would accumulate. On many grounds, I must here do homage to that traditional dose, on the third day, of castor-oil, or something of the kind. Intestinal irritation is by far the most frequent after-illness of puerperal women.

"The truest and safest view we can take of such symptoms is to regard them as indications of septicæmia, arising from the introduction into the blood of materials more or less putrid, or capable of giving rise to changes in the direction of putrescence and to low forms of inflammation."

Before we say anything about the treatment of these symptoms, I propose to show how frequently they have occurred, their causation (as far as ascertainable), and whether ending in recovery or in death, in two thousand consecutive cases of labour attended by myself.

After-illness, of the poisoned class, in the first 100 cases. Intestinal irritation, 14 cases; uterine phlebitis, 1; phlegmasia dolens, 3; irritation from morbid infection, 1; mania, 1; variola, 1; rheumatic fever, 1. This class includes only the graver manifestations of mischief.

Some of the cases of intestinal irritation were very severe; often, doubtlessly, confounded with, and not readily distinguished from, cases of puerperal fever. The morbid infection arose in this way. While making a *post mortem* examination, I was called to a labour. Intense irritation followed, and she narrowly escaped with her life. Only one case of uterine cellulitis is reported.

Death occurred in six cases. 1. Abdominal inflammation (epidemic); died in four days. 2. Phlebitis from scarlatina. I was attending several cases of malignant scarlatina, and had opened an abscess in a fatal case immediately before seeing the patient, and she required the catheter till her death. She died in six days. 3. Puerperal inflammation; premature labour; dead child; died in eight days. 4. Erysipelas, by contagion. Before and during the time of my attendance on this case, I was frequently visiting a lady residing close by, who had erysipelas and retention of urine, requiring the frequent use of the catheter. Died in six days. 5. Scarlatina, or the rash of septicæmia. Died in six days. This case will be named again presently. 6. Fright and exposure to cold, followed by fever. The nurse fell down drunk, and was reported dead. Died in eleven days.

These are copied from statistics of 1,000 cases read before this Society sixteen years ago.

After-illness in second 1,000 cases. 1. On the tenth day, the patient had a rigor, followed by fever; and, on the fourteenth day, a bit of placenta came away, with immediate relief. 2. The patient had diarrhoea three weeks before delivery. For three days before labour, the pulse was more than 100. Fever followed. No probable cause. After a few days, the nurse was seized with erysipelas. This was case No. 70 in 1857. 3. No. 75. Slight phlegmasia dolens. 4. No. 76. Pulse above 100, without any discoverable cause. These three cases were the only ones of sequence and seeming consequence, and will be mentioned again. 5. Forceps. At the end of three weeks, had rigors, followed by slight pleurisy and phlegmasia dolens. 6. Severe intestinal irritation. The outcome of *ante partum* neglect. 7. Rigor; fever; herpes oris. In thirty days, a piece of placenta came away. 8. Swelling of calf of left leg during the third week. 9. Pain in calves of both legs during the third week. 10. Craniotomy; rigors; herpes oris; intestinal irritation. 11. Forceps; mental distress; rigors; intense fever for a little while.

I must give a little detail of this last case.

A lady, aged 28; first child; powerless labour. The forceps was used. She was a stranger, and begged I would not ask her name, nor make any inquiries about her. On the evening of the fourth day, she had a rigor, followed by high fever. On the fifth day, every symptom was so intensely aggravated, that the case seemed hopeless. As a last resource, late at night, I placed a box containing iodine under her nose, and requested her to breathe it during the whole night. She followed the direction with desperate obedience; and, next morning, I had the satisfaction to find every symptom marvellously ameliorated. She rapidly recovered. The note appended (1867) is, "to the iodine, I think, is to be given the credit of being the chief means of cure". I think so still. The effects of iodine in these and kindred cases are not yet all told.

Deaths in second 1,000 cases. 1. Mrs. S., aged 29, in labour with her third child, went on well to the end of the third week, when she was exposed to cold, followed by rigor and so-called acute rheumatism. At the end of a fortnight, she was quite free from pain, and up and about; when, one morning, she was found dead in her bed. This case was clearly arterial, from poisoned blood. 2. Mrs. C., aged 35, was confined with her first child. All was well for three weeks. During the fourth week, she had diarrhoea. On the day month from her delivery, after exposure to cold, she had rigors, fever, pleuropneumonia, and died in five days. This case was clearly venous: a pyæmia not to be distinguished from that arising from, e.g., a case of otitis. 3. Mrs. A., aged 30, was confined with her fourth child. She had diarrhoea during her whole pregnancy; slight fever; phlegmasia dolens. She died from diarrhoea—probably through ulceration of the bowels—at the end of two months. 4. Mrs. S., aged 28; first child. Craniotomy; hydrocephalus; rigors; fever. She had pleasant delirium, and died on the tenth day. This case was unfortunate in all its surroundings, nursing, etc. 5. Mrs. T., aged 22; third child; had phlegmasia of one leg, and died in five weeks. In this case, there was no rigor, little fever, some nausea, occasional vomiting, but no prominent symptom, except increasing weakness. The tenderness and swelling of one leg told, however, of phlebitis and pyæmia. Another woman died after confinement, in the same street, the week before.

What do these cases show? A variety of causes and a variety of effects. Where the history is clear, they show that the main means of propagation was by contact, and not by infection. I have no unequivocal case of infection. I have no cases of sequence and consequence, except after-illnesses, marked No. 70, 75, and 76. No. 70 had fever without any ascertainable cause. She had diarrhoea for three weeks, and a pulse above 100 for three days before delivery. Presently the nurse was seized with erysipelas. No. 75 had slight phlegmasia; and No. 76 a pulse above 100, without any discoverable mischief. My suspicion is—it can only be a suspicion—that No. 70 at, and for some

time before, delivery, was charged with the material of erysipelas; that though there was no external expression of the disease, she by contagion, and probably by infection, gave it to the nurse, and that she poisoned my finger at the time of her labour, and that I communicated it—in a slight degree, it is true—to Nos. 75 and 76. The catheter was a ready method of communicating mischief in two cases.

The poisons of scarlatina and erysipelas did not produce those diseases as such, but virulent septicæmia. I have no case of the specific rash of scarlatina, doubtfully excepting one—that of a lady who, during pregnancy, had been visiting cases of scarlatina, and who had a rash like scarlatina on the third day after delivery, and who died on the sixth day. I was not then acquainted with the efflorescence seen in some cases of septicæmia, and I now hesitate to which class to refer it.

The cases show that septicæmia sometimes follows forceps deliveries. This is no more than we might fairly expect on surgical grounds. The contingency applies to all obstetric operations. It is satisfactory, however, to find only one death from fever after instrumental interference: that of craniotomy for hydrocephalus.

It will be observed that there is a marked difference in the duration of the cases fatal by septicæmia and pyæmia. In the former, a few days; in the latter, all died in five weeks from the time of delivery.

After thus briefly telling what the cases show, the next important lesson is, What do they teach? They teach this: that, in some instances, mischief, even unto death, arose from preventable causes, viz., intestinal irritation, in default of previous attention, from the hand tainted with the virus of a *post mortem* examination, from the fingers poisoned with the matter of a scarlatinous abscess, or reeking with the venom of malignant erysipelas. These lethal causes, and all such like, may and must be avoided, if we are for the future to practise our profession in peace, quietness, and safety. Endemic influences must be counteracted as well as may be, operative measures avoided when practicable; and the whole physiology of labour and the pathology of its sequences must be carefully observed and ministered to with patience, with a definite aim, and with clean fingers.

I have used all the preventive means I knew—habilitmentary, detergent, and antiseptic. When scarlatina has broken out in a family where the lady was pregnant, I have recommended her to go away to be confined, as we all know with what pertinacity the poison of scarlatina sticks to a house or to a room. I have avoided, as far as possible, attending cases containing the elements of septinous contamination, and I have never been obliged to cease from practice, even for a limited period.

Before just running over the principles of treatment, it may be useful to compare puerperal fever, the unity of its cause (some septinous material) and the non-uniformity of its results, with some other common agent, and the diversity of its effects. Let us take one with which we are just now familiar: that of the cold of last winter.

We have nothing to do now with the annihilation of function produced by extreme cold, as of a flock of sheep being found dead in the morning after premature shearing, or of a rabbit denuded of its fur. A coat of varnish has nothing to do with the event; they are frozen to death. What I refer to are those lesser perturbations of the system, spoiled functions, with all their noxious entail, not consequent on a cold, but dependent on the cold.

What were they? Affections of every region of the body, from the head to the feet; convulsions, coma; inflammation of tonsils, pharynx, larynx, trachea, bronchi (rarely of smaller tubes), lungs, pleura, stomach, rheumatic fever (so-called), and neuralgias everywhere; as usual, extremes of exposure and extremes of susceptibility must be taken into account.

Observing these every day occurrences, how can the effects of poison introduced into, or generated in, a puerperal woman, be expected to be uniform?

I should like to know how members treated all these various affections during last winter. If a remedy were sought for each organ affected, your therapeutic resources must have been pretty well exercised. For my part, I treated them all alike, on general principles.

So in puerperal fever, turpentine, internally and externally; breathing an iodised atmosphere; opium and quinine, with, of course, proper food, air, and stimulants, etc., do best what can be done, on the same general principles; modified, of course, by the cause.

Quinine, according to Dr. Richardson, counteracts the different poisons in their power of liberating oxygen, his theory of their bane.

Salicine is allied to quinine, and is given as its substitute. Salicine is obtained (analytically) from the willow tribe, the meadow sweet, and winter green.

Salicylic acid is found to be the most effectual disinfectant. It can be made, also, and commercially it is so made—synthetically—by the action of carbonic acid on carbolic acid.

This alliance between quinine, salicine, salicylic acid, and carbolic acid, strikes me as at least curious, and possibly may have a more intimate relation with the success of these substances in the treatment of puerperal fever and such like diseases, than we are at present aware of.

INTRATHORACIC TUMOURS.*

By J. W. F. SMITH-SHAND, M.D.,

Physician to the Aberdeen Royal Infirmary, etc.

I.—PRIMARY CANCER OF THE LUNGS. (Concluded from page 845 of last volume.)

CASE II.—In the next case I shall bring before you, I have no positive proof to adduce in support of my diagnosis of primary cancer of the lung, although I think I have strong presumptive evidence in its favour. George McKenzie, aged 28, a coachman, was admitted into the Royal Infirmary on July 1st, 1872. The patient stated that he enjoyed good health till last winter, when he was exposed to cold and wet, but he was not confined to the house in consequence. He noticed after this, however, that his breathing was short after any exertion, and that he had a short dry cough. He never had any pain in the chest, and he could lie on either side in bed. About a fortnight ago, his cough became much more troublesome, and he had a white frothy expectoration. There was no hæmoptysis.

Condition on Admission.—The patient had a puffy bloated appearance, with lividity of lips and cheeks, and lay in bed with his head and shoulders raised. There was slight fulness of the jugular veins. There was deficient movement of the left chest, with dullness on percussion in the lower half, and absence of breath-sound and fremitus. A subtypanic note was heard in the left infraclavicular region. There was puerile breathing on the right side. The heart-sounds were best heard to the right of the sternum. Pulse irregular, 125 per minute. Bowels costive. The urine was scanty, of specific gravity 1030; contained no albumen. Temperature 103 deg.—July 6th. There was dullness over the whole left chest, with absence of breath-sounds, except on deep inspiration, when distant feeble breathing was heard. The left hand and arm were œdematous.—July 8th. The nurse stated that he had been confused and delirious the previous night. The œdema of the arm had increased, and there was marked swelling of the left side of the face and neck. On inspection, the left side of the chest was smaller than the right, and the infraclavicular region was flattened. The heart-sounds were heard more to the right of the sternum. There was no change in the other physical signs. He expressed himself as feeling easier. The left pupil was slightly more dilated than the right. His voice was rather husky. He said that of late his voice had altered in character, and had not been so clear as formerly. About 3 P.M., he fell into a heavy sleep, from which he could with difficulty be roused. The stupor gradually became more profound, and he died at 6 P.M. The last two days, his urine was copious, as he passed three pints in twenty-four hours.

Strong efforts were made to obtain a *post mortem* examination, but they failed. All that I could venture to do was to insert a trocar into the chest in the axilla. Considerable resistance was offered to the passage of the trocar, as if it were passing through some dense firm substance, and when the trocar was withdrawn, nothing followed, except two or three drops of bloody serum.

REMARKS.—The existence during life of well marked inward pressure signs on the left innominate vein in this case, and the result of the exploratory examination after death, conclusively prove that the dullness on percussion was due to a solid tumour, and not to fluid. It is a clinical law laid down by Walshe, that evidences of pressure on internal parts, as the aorta, arteries, veins, nerves, trachea, bronchi, œsophagus, or thoracic duct, are solely due to tumour, whatever its nature may be, and that neither hydrothorax, pneumothorax, or empyema can produce them. There can be little doubt also that the obstruction to the venous circulation was the cause of death, by inducing stupor and cerebral congestion. The only point to decide is, whether the tumour was aneurismal or cancerous. My reasons for fixing on the latter were the absence of pain and impulse, and the evidence of slight displacement of the heart to the right side.

In this case, the left chest was flattened and smaller than the right, while in Mackie it was enlarged, as judged by the eye. Dr. Walshe, in his last (fourth) edition of *Diseases of the Lungs*, states that, when there is simple cancerous infiltration associated with limited tuberculous formation, the affected side is either retracted or depressed, but that, when the cancer of the lung is mainly tuberculous, the diseased side is generally

* Read before the Aberdeen, Banff, and Kincardine Branch.