early discovered to be merely fallacious. The importance and the methods of applying simple tests for the hearing should be known to every practitioner. I have already called attention to the fact that a person may be extremely deaf in one ear, and yet with the remaining good ear hear so well that no defect is noticed; but I have known physicians themselves to say of such a case that they did not believe there could be any deafness, simply because they had never tested the hearing of the two ears separately. It would seem superfluous to notice, but experience shows, that the failure to close one ear when testing the other, is a very common source of error. The first step in testing, then, should be the closure of the opposite ear with one finger.

Having done this, the open ear should be tested for the distance at which both the watch and the voice can be heard; not whether they can be heard at all, but whether they can be distinguished as far as normal. In regard to the watch, it should be remembered that the intensity of the ticking varies in different watches, and for comparative tests the same watch should always be used, and the normal distance established by tests on normal ears. It should also be borne in mind that the normal hearing distance for the ticking of a watch varies with age, being very much less in old persons than in children. With the voice the hearing distance should be established in the same way; but as it is generally impossible to get space enough to thoroughly test a clear, distinct tone, that is, as it is impossible, for want of space, to reach anything like the normal distance for that tone in ordinary houses, it is necessary to lower the intensity of the tone to make the normal hearing distance for that tone correspond, approximately at least, with the size of the room in which the test is made. Having done this, words or short sentences should be used, and the patient required to repeat them; for if this repetition is not insisted upon, many patients will assert positively that they understood, when they have merely heard the sound of the voice, but have been wholly unable to distinguish the words. It is also well to use a number of different words or sentences, as certain letters of the alphabet have much less distinctive sounds and are much less readily heard than others. If this caution of employing several sentences is used, however, it can scarcely occur that in each of them these difficult letters and sounds should predominate.

Having determined the distance at which both voice and watch can be actually heard, and knowing the distance at which they should be normally heard, the hearing for that case can be well expressed for practical purposes as a fraction, the numerator of which is the actual distance, and the denominator the normal distance. The necessity of testing for both watch and voice depends upon the fact that the hearing for the two is by no means always proportionally the same; while it may be very good for one, it may be very bad for the other. This is seen especially in the class of cases where the degree of hearing enables us to judge very fairly how the case is progressing, and whether any treatment for the restoration of the functions of the ear is absolutely necessary; these cases are those of acute tympanic inflammation, where, as the inflammation subsides, the hearing slowly returns, but much more rapidly, as a rule, for the voice than for the watch, so that it is found when the hearing for the watch is only perhaps $\frac{1}{8}$, that for the voice will be $\frac{1}{2}$.

2

In children the test for the watch is often uncertain, from fright, mischievousness, or lack of intelligence on the part of the patient, but that for the voice can generally be made effectual by a little ingenuity on the part of the physician in asking questions on subjects in which the child takes an interest, the answers to which will show that the question was well understood.

In all testing great care should be taken to exclude the imagination, which is not infrequently both active and ingenious in deaf persons. If such tests were in common use, it would often be found that the cessation of pain, discharge, and other well-marked symptoms did not necessarily imply a restoration of the ear to its normal condition; any deafness would be by this means discovered early, long before it had begun to inconvenience the patient, and the question of treatment could be determined before the serious and irremediable tissue changes had come on, at a time when treatment is, in a large number of cases, of real positive value.

CEDEMA OF THE LUNGS, WITH CASES.¹

BY HENRY F. BORDEN, M. D., BROCKTON, MASS.

To choose a subject to write upon, and read before a body of medical men, at the present day is not an easy task. I do not mean to imply that medical subjects have been exhausted, for the field for work and the amount of material to work upon seem as large as ever, and the chance for seeing the end is not a very brilliant one for those engaged in investigation. Circumstances often give one an opportunity to choose a subject for discussion which may be interesting to himself, if to no one else. My experience during the past year has interested me in œdema of the lungs, having lost a patient under very trying circumstances with this sudden and fatal malady.

I have received the histories of three other cases, which occurred in the practice of neighboring physicians, two being out of town, and the third in the practice of Dr. A. E. Paine, of Brockton. Only two of these seem to have been pure cases of pulmonary œdema, the third, in all probability, being embolism of the pulmonary artery without manifestations of dropsy.

After reviewing the cases, and considering the causes which can produce the different forms of œdema, I shall endeavor to make a practical application to each case. Through the courtesy of Dr. F. C. Shattuck I have been able to collect a few notes on experimental research as to the cause of pulmonary œdema, published in Virchow's *Archiv* of 1878, and in German medical pamphlets by Welch, Mayer, and others. I shall first report the cases and then discuss the causes.

CASE I. Mrs. M. S., aged twenty-four years; occupation, housewife. Grandparents on father's side lived to an advanced age. Grandmother on mother's side died of organic disease of heart, at seventy odd years of age. Father and mother are both living, and enjoy good health. I have attended both at different periods for slight sickness, but for nothing of a severe type. Lost one brother by diphtheria, and one sister with pulmonary phthisis following typhoid fever. Has three brothers and one sister living, and in good health.

¹ Read before the Massachusetts Medical Society at its last annual meeting, and recommended by the society for publication in the JOURNAL.

The subject of this history was a picture of health. I had known her from childhood, and attended her for slight attacks of "nervous headache," which were of a congestive form, being accompanied by a rush of blood to the head and face, without dyspeptic complications, being easily relieved by the use of bromide of potash or belladonna. I never knew of her being otherwise sick. About the fifth month of her pregnancy I was called by her husband to see her. I found her feeling quite happy, but presenting a general oedematous condition of face, hands, and limbs, although not to an alarming extent; the breathing good; action and sounds of heart normal; digestion good; bowels correct; the urine passed as usual. On examining the urine I could detect nothing abnormal: no albumen, no casts, no sugar, nor even anything that denoted a disorganized or disordered condition of any organ. I examined the urine from time to time with the same result. She never exhibited any signs of uræmic poisoning, either by looks, spasms, or any other condition, to the time of death. She continued in about the same situation until the day of her death. I ordered moderate doses of potass. bitart. to be taken daily, with a marked increase in the amount of urine passed, although there were not at any time symptoms of diminution of this secretion, as far as her history showed, before I saw her. I recommended moderate exercise, fresh air, avoidance of over-exertion and excitement, together with good nourishment, all of which she had. The fl. ext. jaborandi was given a few times, but no increase in the urine passed was observed over that produced by the potash. On the afternoon of the day of her death (about three P. M.), I was summoned in haste to see her. On arriving, I found her much changed. There was increased oedema generally; marked lividity; pulse nearly one hundred beats per minute, and much weaker than usual; no rise in temperature; intellect clear; some dyspnoea, but less than previous to my arrival, according to the family's statement; extremities cold and oedematous; dullness over the lower two thirds of both backs, with subcrepitant râles, and supplementary loud breathing over the upper half of lungs in front, with normal percussion. I ordered stimulating applications to back, and gave tinct. digitalis and whisky, all that was at my command. The heart showed nothing but diminution in strength of action, and neither murmur nor irregular action could be detected. She received relief from this treatment, and felt quite comfortable until evening, the lividity and weak symptoms disappearing rapidly. At about forty-five minutes past eight P. M. she was taken in labor, — it being full term, — and upon arrival I found the "os" dilating rapidly, and the pains unusually strong and frequent. She was able to lie down, and expressed herself as feeling easier and better able to breathe than at any time during the day. Her labor continued regular until about forty-five minutes past nine P. M., or one hour, when the membranes, which had descended very low, ruptured, and immediately she exclaimed, "There is water escaping from me!" while her breathing became labored and heavy. I endeavored to give her some wine, but she could not swallow, and as her breathing was growing more difficult, and amounting to dyspnoea of the worst sort, I threw open a window and commenced artificial respiration, but to no purpose, as her face was blue as slate-stone, and a stream of pink frothy serum gushed from her mouth and nostrils in sufficient quantity to saturate all the clothing about

her and the bed. She was dead in less than two minutes from the time the waters escaped. The heart was the last to give out, continuing to beat slower and weaker until it stopped, even after respiration and life had ceased. The general oedema after death was enormous. During the interval between her first attack in the afternoon and her death, the pulse had beat as full and well as could be desired in almost any case. No autopsy.

CASE II. Mrs. A., aged about twenty-seven years; occupation, housewife. The physician in charge had examined her urine with negative results. She showed but little general oedema, and that only about two weeks previous to her death. She had not arrived at full term of gestation, and was consequently not in labor at the time of her death. There is heart disease in the family history, but I have been unable to find that she was the subject of any such trouble. Her general appearance was that of one in usually good health. The fatal termination was as follows: She awoke her husband at about three A. M., and told him she could not breathe; he arose and assisted her to the adjoining room, where she sat down, and died almost instantly. Medical aid was summoned, but too late to render assistance. The physician was informed that she died by being "filled up with frothy water," and he thus described her appearance as he found her: "Extreme cyanosis, frothy serum in large quantities about the mouth and nostrils, and an enlarged, distorted look of the face." There can be but little doubt that this was a pure case of non-inflammatory oedema of the lungs.

CASE III. is one where a doubt exists whether oedema was a cause of death. A clot of the pulmonary artery is, to my mind, the cause that entered most prominently into the case as a chief factor in its production. I shall omit the history of this, and proceed to detail Case IV.

CASE IV. O. G. T., aged fifty-eight years; occupation, dealer in horses, — hence he was employed out-of-doors the greater part of his life. He was one of our best-known citizens, and was acquainted extensively with all parts and people in Brockton and vicinity. He came of a healthy stock, and was himself as robust and strong a man as one will often meet. He was never ill, to my knowledge, but once, and that about three years ago, when he was seized with an attack, at first feared to have been apoplexy, but which proved to be a disturbance in the cerebral circulation, due to a reflex influence from a badly disordered digestion. He completely recovered from this attack, as far as can be ascertained, and has been as robust as ever. Dr. A. E. Paine, who was summoned at the time of his death, assures me that no cardiac disease ever existed, or was ever suspected, and furnished me with the history of his condition at the time of death. He was a man of excitable temper, and on the day of his decease had some business trouble which worked him up to a high pitch of irritability; and from the nature of this trouble, I should suppose it must have continued to torment him during the few hours that he lived. He continued out-of-doors as usual until after six P. M. on the same day, and complained only of having caught a slight cold. He retired as usual between nine and ten that evening, and, after he got into bed, requested his wife to bring him some little remedy, which it was his custom to use for colds. She found him getting up on her return, and asked him why he was doing so, when he had just time to tell

her he could not breathe, and fell back into a chair dead, and at the same instant a stream of frothy serum flowed from the mouth and nose in a quantity amounting to more than three pints. His face was cyanotic and puffy. This serum continued to flow from the same avenues all night. No autopsy could be obtained in any one of these cases, and certainly I do not think it was at all needed to establish a diagnosis. Before considering the causes in these cases, I will give a résumé of the results attending the previously mentioned experiments, to determine what *will* and *will not* cause œdema of the lungs. The experiments were mostly made on rabbits and dogs. Welch remarks that none of the explanations of the causes of acute general œdema of the lungs up to the present time have been satisfactory.

I. That increased action of the heart alone is not sufficient is proved by cases of compensated mitral stenosis, in which the pressure in the pulmonary artery must be greater than in a heart offering no impediment to the onward flow of the blood; and that the resistance of the pulmonary capillaries is full as great as that of any others.

II. The rarity with which pulmonary œdema follows direct irritation of the lungs from the inhalation of irritating substances, and the like, renders it very probable that it has the same cause (when it is met with) as the œdema which ends in death from all sorts of causes (according to Welch), *paralysis of the left heart*.

III. Collateral hyperæmia. Two essentially different conditions are generally included under this term.

(1) Acute general œdema occurring in the course of diseases which involve an impediment to the circulation in a portion of the lung; and (2) local œdema about inflammatory foci and new formations, *which* œdema bears a constant proportion to the size and extent of such foci in growth. By cutting off three fourths of the circulation of the pulmonary artery, Lichtheim was able to show that difficult circulation in a portion of the lungs is not sufficient to cause general œdema. General œdema is not a constant result of embolism of the pulmonary artery, and must have associated cause or causes to produce it.

In the case of local œdema, the most important factor is inflammatory changes in the walls of the vessels, facilitating serous transudation.

In passive œdema two causes have been assigned: (1.) Accumulation of blood in the pulmonary veins from valvular disease of the heart; not liable to occur, for the reason that as long as the obstruction is compensated by increased work on the part of the right ventricle, œdema does not occur (compensated mitral stenosis).

(2.) General cardiac weakness. Syncope never leads to œdema of the lungs. By irritation of the vagus, paralysis of the heart can be induced, but œdema does not follow. In rabbits, œdema takes place only when one carotid or one subclavian is the sole passage of the arterial blood from the heart. In dogs, the general circulation must be still more impeded before œdema occurs, or the pressure in the pulmonary artery is markedly increased.

Occlusion of all the pulmonary veins of one lung is not sufficient to produce œdema, *but* hæmorrhagic infarction. In fact, almost all the pulmonary veins must be tied, in order to produce œdema or perceptibly increase the pressure in the pulmonary artery.

Suppose, however, that the left heart is paralyzed while the right continues to work and drive in more blood than the left ventricle can dispose of, congestion of the lungs results, and is followed by œdema.

Mayer finds: That if the great arteries leading to the brain were occluded, in non-curarized animals severe convulsions followed, and in a great majority of the cases extensive pulmonary œdema. In curarized animals treated in the same way œdema did not occur. The œdema was so marked as not to require a post-mortem to establish it; the tracheal canal was filled with reddish frothy fluid in from one to two minutes after occlusion of the arteries, and immediate artificial respiration was fruitless. Arterial tension is greatly increased by anæmia of the brain, and the forcible respiration and muscular spasms, causing constrictions, force the blood upwards to the right ventricle.

In short, causes which hinder a free discharge of blood from the left ventricle lead to œdema of the lungs. In curarized animals, muscular irritability is abolished.

So much for results obtained by experimentation on healthy animals with regard to œdema of the lungs. The only mention made in books of midwifery, that I have seen, is by Cazeaux; in speaking of the prognosis in œdema in pregnancy without renal complications, he says, "Sometimes after delivery the disappearance of the infiltrated liquid has been followed by a serous congestion of the nervous centres or the respiratory organs, often terminating in death." He thus recognizes the fact of a sudden accumulation as well as disappearance of serum in limited localities, without attempting to explain the cause of it. It is evident to all that when an inflammation exists in the lungs, either as pneumonia, an inflammatory process around a mass of tubercular deposit, or any sort of neoplasm, there must be more or less œdema, just as one sees it in cellulitis when a slough of the subcutaneous tissue is the cause. It is therefore unnecessary to dwell upon circumscribed inflammatory œdema, or that due to mechanical obstruction of a portion of the lung circulation, from either disease or growth. In Bright's disease, or destructive nephritis, or uræmia during pregnancy, I think one explanation of the cause of œdema is the poisoning of the nervous centre of the action of the left heart, and consequent paralysis and œdema, instead of the local irritative effects on the mucous membrane of the lung area; although I can conceive it possible for the walls of the vessels to be weakened by a sort of continued inflammatory process by this action of urea in the blood, and transudation made more easy. One physician of eminence suggested that an eczematous condition of the bronchial surface might exist, without febrile manifestations, which I think possible, and can conceive how such a condition might affect the integrity of the capillary walls. I have not seen anything to lead me to suspect that as a cause in the cases given.

The conditions for the production of acute general non-inflammatory œdema of the lungs seem to me to be twofold, namely: (1.) A cause or causes which first lead to accumulation of blood whose local pressure becomes greater than normal, varying in intensity according to the agents in its production. (2.) A certain loss of natural vitality and power to resist serous transudation in the walls of the vessels, brought about by a change in their nutrition due to some abnormal condition, either recognizable or not. The causes of accumulation, as shown by experiment, may be summed up in

a few words. Paralysis of the left heart; obstruction to the direct flow of blood from the left heart; obstruction to the pulmonary veins; anæmia of the brain by sudden occlusion of the great vessels leading thereto, seem to be the chief causes of congestion of the lungs. A deficient supply to the lungs, as would be the case in embolism of the pulmonary artery, is not regarded as a sufficient cause alone. Of all these causes of congestion, only one or perhaps two were sufficient to carry the process to œdema. These were paralysis of the left heart and sudden occlusion of the large vessels leading to the brain. We must remember that these experiments were carried on in small rabbits, whose tissues were undoubtedly in the best possible condition, and not in full-grown men, whose heart power is so much greater, and whose tissues, at least in those people thus attacked, must be more or less deficient in resisting power.

Whether the minute arteries undergo fatty changes or their walls become thinned, I cannot say, but one would hardly expect to see œdema occur in perfectly healthy well-nourished subjects. In case I am right, and deficient nutrition is one of the causes which favor transudation, then I think it may occur under circumstances which will favor congestion, brought about as follows:—

Supposing an irritation of that portion of the cardiac branch of the vagus, which is said to govern the vaso-motor apparatus regulating the minute vessels of the pulmonary circulation, should result in paralysis of the same, then complete dilatation of these vessels must follow, and as their calibre is increased, the force of the pulmonary circulation is distributed over a larger space, and the current of blood is proportionately slower; and with increased action of the right heart there would occur a crowding of the capillaries, and consequently increased blood pressure with serous transudation in cases where the subject is fit for such a change. Supposing the nervous influence of that portion of the vagus governing the right heart should produce an increased action, and that of the left heart a decreased action, then it is evident that sooner or later an overplus of blood and increased pressure in the lungs must be brought about, and where long-standing disease or unknown deficiency in nutrition of the arterial walls exists, serous transudation or œdema must follow to a greater or less extent, in my opinion. That these sudden rushes of blood to and from different portions of the body are due to nervous agency acting through the vaso-motor system, no one will dispute; the blush on the face, either of shame or of excitement, illustrates this. So in these cases of œdema, I can see no other explanation but that nervous action is the prime mover in the production of those sudden appearances and disappearances of serous collections. In Virchow's *Archiv* of 1878, the results of three autopsies on male subjects who died of general œdema of the lungs are reported. There was not a sign of heart disease, arterial occlusion, or anything abnormal to be considered as a cause, but the lungs were in a state of œdema, and that was all which could be said. General œdema is not caused by a pulmonary artery embolism alone; but death from this is undoubtedly accompanied by some œdematous effusion, not alone sufficient to produce death.

In Case I. there may have been a clot, but I doubt it. The symptoms of a clot are as follows: Death may suddenly occur without any marked symptoms.

When the clot partially fills the pulmonary artery, a systolic murmur may be heard over the left second intercostal space, or at the base of the heart; but this is not a certain symptom, as other causes may produce it,—dyspnoea, cyanosis, irregular action of the heart, expectoration of bloody sputa, cerebral disturbance, etc. In Case I. the serum collected rapidly in the afternoon, until the posterior two thirds of the lungs were filled, and in about two hours subsided, the heart's action continuing regular, but weaker, during this onset. No pain, no cerebral trouble, whatever. I cannot believe that a clot in the pulmonary artery could form and cause this amount of œdema, or be a factor in its cause, and the system become tolerant of its presence in so short a time as to calmly return to so comfortable a condition as subsequently existed. In the evening the whole process was repeated, and the waters returned and in two minutes' time filled the tubes to overflowing. It seemed to me too regular and gradual for the sudden production of a pulmonary clot to have taken place. Paralysis of the left heart could not have existed, for the pulse must have stopped immediately. No evidence of sudden occlusion of the cerebral vessels showed itself. We must fall back on a nervous origin, I think. I have been tempted to regard the sudden rupture of the membranes and escape of the amniotic fluid as the shock that excited to action the agents in the production of the œdema. As the other cases had no physicians to watch the failing pulse and vital powers, I shall not attempt to name the exact cause that originated these fatal conditions, but leave them as members of the same family. It is self-evident that any disease that affects the tone of the system, impedes the circulation in the lungs, or taxes the vital powers, must render the accumulation of serum more speedy and fatal. The lungs being in continual motion in the acts of inspiration and expiration, in my opinion, renders the chances of œdema of these organs more liable than in any other parts.

The treatment of œdema in its active state depends on the amount of lung substance involved. When it is limited in extent, counter irritation and tonics to the sympathetic and circulatory systems are the agents most generally indicated. If the trouble is extensive and general, as in the cases above cited, practically nothing is of any avail, although artificial respiration should be tried as the only means to seize upon. Prophylactic treatment is to be mostly depended upon, and if the theories advanced be correct, it is evident that good nourishment and tonics, such as iron, bark, and such others as the case in hand shall indicate, are the articles to be used in order to maintain the proper tone and integrity of the system and tissues. Quietude of mind and body to save the strength, mild diuretics and diet to maintain regularity in the bodily functions, fresh air, and a moderate degree of exercise, avoiding fatigue, and the strict observance of the general laws of hygiene are all desirable. If I were to favor the use of any particular drug, I think iodide of potash would be as good a general medicine, providing the stomach bore it well, as I know of. If these means should fail, I do not see any way but to trust to nature and accept whatever comes as inevitable.

I have written this paper for two reasons: first, because of the interest which I have taken in the subject, from being thrown in contact with it under peculiar and trying circumstances, and secondly, because I believe there have been many deaths from this disease

which have been dismissed as congestion of the lungs, or under the still more vague term, heart disease, and nothing more thought of it, as long as the friends and family were satisfied.

I do not claim to have given a solution of the causes or conditions of its occurrence, but if I have brought it sufficiently before the profession, and rendered it deserving of more attention and study, it may be the means of making physicians more cautious in cases where œdema exists, especially in pregnant women, even without renal trouble. I think that this condition has been regarded too lightly, and the care of such patients should be made a more careful study than has been the custom in time past.

RECENT PROGRESS IN OBSTETRICS.

BY W. L. RICHARDSON, M. D.

INTRA-UTERINE VACCINATION.

FOLLOWING out the ideas suggested by Professor Bollinger (Munich) in his monograph published in Volkmann's series, Dr. A. E. Burckhardt¹ gives the result of some experiments made in the obstetric wards in the hospital at Basel. During the years 1877 and 1878 he revaccinated twenty-eight pregnant women. Owing to circumstances beyond his control, only eight of the children of these women were available for future experiment. Four children were then vaccinated, whose mothers had not been vaccinated during the pregnancy, and in every case perfect pustules were produced. With this same lymph, whose efficacy had thus been proven, he vaccinated the eight children of the mothers who had been vaccinated during pregnancy. The results were as follows: The children of four women, whose revaccination during pregnancy had been perfectly successful, were found to be insusceptible of the vaccine lymph. The children of two of the women, whose revaccination during pregnancy had been only partially successful, were also found to be proof against vaccination. Of the two children whose mothers had been unsuccessfully revaccinated during pregnancy, one was vaccinated successfully, and the other failed.

These experiments of Dr. Burckhardt, although few in number, agree with the results obtained by Rickett, who inoculated about seven hundred ewes during the last few weeks of gestation. Their lambs were inoculated, when from five to six weeks old, with sheep-pox lymph, with no result, although at the same time thirty-six lambs, whose mothers had not been inoculated, were all successfully operated on and had true pustules.

ANTISEPTIC OBSTETRICS.

Professor Stadtfeldt,² of Copenhagen, calls the attention of the profession to the great value of antiseptics in the treatment of obstetric cases. Since 1870 he has employed this method of treatment, and with the most gratifying results. During the last five years, out of 5098 lying-in patients, only one in 116 has died of puerperal fever. This result is more favorable than that obtained in any other lying-in institution. He advises that the vagina be carefully

washed out before the delivery, and that the moment the presenting part of the child shows itself at the vulva a carbolic vapor spray be thrown on the exposed parts. This should be continued until any lacerations occurring during the delivery are united by sutures. The vagina should then be carefully washed out with carbolized water, as also the vulva and adjacent parts. In all cases in which the hand or instruments have been introduced into the vagina, or where a portion of the membranes have been left behind, a three per cent. carbolized intra-uterine injection should be used after the delivery. He has never seen the slightest evil result follow an intra-uterine injection, although he has employed them in hundreds of cases.

Dr. Schucking advises that immediately after the delivery the vagina be wiped out with a tampon of cotton which has been dipped in a solution of carbolic acid (five per cent.). A sound is then carried up to the fundus uteri, having previously been surrounded by a piece of gauze soaked in the same solution. The uterus and vagina are then thoroughly disinfected by means of an irrigator, which is connected with the sound. For permanent irrigation he uses a solution containing ten per cent. of sulphite of soda and five per cent. of glycerine. Every twelve hours the sound and gauze are removed and a fresh instrument is inserted, while at the same time the carbolized injection, followed by the sulphite of soda wash, is repeated.

LACERATIONS OF THE PERINÆUM.

Dr. T. A. Reamy³ calls the attention of the profession to the necessity of immediately sewing up a perinæum which has been torn during a delivery. The parts should be sponged with warm water, and if the flowing at all impedes the operation a sponge can easily be inserted into the upper part of the vagina. There is no necessity for an anæsthetic, as the pain amounts to nothing; nor is there any for trimming the parts. The sutures should be placed very closely together, at least four to the inch. By placing them so closely, there is not the necessity of drawing the edges so tightly together. All clots and blood must be removed from between the parts before they are brought into apposition. The urine should be drawn for two or three days. It is not at all necessary to confine the patient's legs together, nor to insist upon her lying in any one position; but she should not be allowed to separate them widely.

PORRO'S OPERATION.

Dr. R. J. Kinkead, in a recent paper read⁴ before the Dublin Obstetrical Society, gives an interesting and critical description of this operation as compared with Cæsarean section, laparo-elytotomy, and craniotomy. Thus far thirty cases have been reported, with the result of fourteen mothers being saved and sixteen deaths, or a mortality of 53.3 per cent. The dangers which attend Cæsarean section are (1) peritonitis; (2) metritis; (3) hæmorrhage; (4) septicæmia; (5) shock; (6) intestinal obstruction, arising from a coil of the intestine being caught in the uterine wound. By Porro's method, the second, third, and sixth of these are abolished; the fourth is greatly reduced, and, with the proper use of antiseptic, should be entirely abolished. The danger from peritonitis is also greatly reduced. Several modifications

¹ Deutsches Archiv für klinische Medicin, xxiv.

² Centralblatt für Gynäkologie, 1880, vii.

³ Obstetric Gazette, January, 1880.

⁴ Dublin Journal of Medical Science, May, 1880.