THE CONNECTING LINK BETWEEN THE HIGH-TENSION PULSE AND ALBUMINURIA.

Read in the Section of Medicine at the Annual Meeting of the British Medical Association, held in Leeds, August, 1889.

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I have previously mentioned the great interest which the writings of the late Dr. Mahomed on the pre-albuminuric stage of Bright's disease have had for me, in connection with uric acid, as a cause of high pulse tension, and I have pointed out that many of the symptoms of this stage which he mentions appear to me to be the work of uric acid.[1]

Dr. Mahomed considered that the symptoms of the early stage of Bright's disease were due to a poison in the blood, and I have suggested and believe strongly that those very symptoms are just the ones which an excess of uric acid in the blood produces; and I have thus come to believe that the high tension which, as I have said, I can produce or remove at pleasure, may be the cause of the kidney disease rather than the other way.

And, thinking over this matter, and looking for a possible connection between high pulse tension and albuminuria and nephritis, it has seemed to me that in the writings of Professor Semmola, of Naples, there may be contained just the connecting link required to show that the high pulse tension of uric acid may be the absolute cause of the nephritis, and that thus it may be possible to explain in the most simple manner the frequent association of chronic nephritis with gout.[2]

I have for several years carefully read and reread all the writings of Professor Semmola which have been published in French, and to which I have had access; and I have been more and more convinced, not only of the great care bestowed on all his work, the truthfulnes with which it has been reported, and the caution with which inferences have been made, but also of its very great importance as an aid to the elucidation of many obscure points in the pathology of Bright's disease. And it has been a matter of considerable surprise to me that so little notice has been taken in this country of the researches of this great Professor, who is evidently held in high esteem in his own country and in France.

There is a short notice of some of his conclusions in the British Medical Journal, vol. ii, 1886, p. 885; but in textbooks, when Professor Semmola's name is mentioned, it is dismissed in a few words with the remark that his experiments are insufficient,[3] and without, so far as I know, any attempt to discuss the questions which he raises.

Since the above was written, Dr. Saundby's very able *Lectures on Bright's Disease*[4] have appeared, and as he, unlike the writers of some of the textbooks to which I have referred, devotes considerable space to the discussion of Professor Semmola's arguments, I now add a few remarks on one or two of the chief points he adduces against them.

As it is impossible to go minutely into the matter without more than doubling the length of this paper, I must summarise, and say that the chief points urged by Dr. Saundby are: (1) the fact noticed by Tizzoni that albumen from the urine of a case of morbus Brightii did not cause albuminuria when injected into the circulation of animals; and (2) the assertion of Snyers that egg-albuminuria—that is, the albuminuria caused by the injection of egg-albumen into the veins or skin—leaves no ill effects on the kidneys.

It may be noted in passing that this assertion of Dr. Snyers is in direct antagonism to the results of many carefully made and fully recorded experiments by Professor Semmola. Then, on turning to Snyers,[5] it becomes evident that he bases his argument to a large extent on the experimental criticisms of Stokvis; and on turning to the writings of this latter authority,[6] it becomes clear that his facts are not nearly so fatal to Professor Semmola's arguments as might be supposed; for, first of all, Stokvis found that the injection of egg-albumen did sometimes lead to persistent albuminuria; and, with regard to these cases, he says (page 302): "Il nous semble probable que nous avons determiné dans un cas une hyperemie des reins sans le savoir ni le vouloir et que c'etait finalement de l'albumine du serum qui passait dans les urines," which is almost precisely Semmola's conclusion from the same facts. Again, Stokvis draws conclusions against the presence of diffusible albuminoids in the blood in Bright's disease on the strength of one single experiment, in which he injected 60 cubic centimètres of serum (from a case of Bright's disease), containing 7.7 per cent. of albumen, into a dog weighing about 14 pounds.

It seems to me obvious that, as the diffusible albuminoids in the blood serum of advanced Bright's disease bear only a very small proportion to the non-diffusible, the dose of diffusible albumen in this case was probably much too small, and that Semmola would not have expected a positive result from such an experiment, unless the serum was from an acute case; but this point is not mentioned. The same remark applies to Tizzoni's experiment with the albumen from the urine of Bright's disease, for here, especially in advanced stages, the diffusible albumens bear an even smaller proportion to the non-diffusible than in the blood serum, and Semmola himself is careful to advise the use of the albumens of the blood serum of acute cases, and not those of the urine.

Further, though I speak in this paper only of the indirect effects of plus tension in causing albuminuria through deficient skin activity, I am quite aware that high arterial tension has a direct effect on the kidney circulation, and possibly also on its nutrition, and I intend to go into this matter in a future paper; but it does not appear to me that any alteration of kidney circulation or even of its structure will account for an excess of diffusible albuminoids in the blood serum, and in the matter of this excess of diffusible albumen and its excretion in the saliva, sweat, and bile Dr. Saundby acknowledges that Professor Semmola has the support of Vulpian and others; and some of the facts given even by Stokvis are not unfavourable to Semmola's assertion.

Lastly, Dr. Saundby quotes from his own experience cases of albuminuria lasting for many years without kidney mischief becoming obvious, and he argues from them that the kidney was not injured by the passage of albumen; but against this I think that some of the cases in Dr. G. Johnson's most interesting paper[7] may fairly be quoted as evidence in favour of a different conclusion.

I now propose to go at some length into the points in Professor Semmola's researches which seem to me to bear on the relation of albuminuria, and possibly of nephritis, to pulse tension.

Now, Professor Semmola has shown[8] by experiments, which, to my mind, are as sufficient and conclusive as any I have ever read of, that in animals the injection of a certain quantity of a foreign albumen into the skin causes not only albuminuria, which lasts so long as the injections are continued, but that, if the injections are kept up for a certain number of days, you get in the urine not only the foreign albumen originally injected, but also the serum albumen from the blood; and that, if the injections are now suspended, the serum albumen will continue to pass in the urine for some considerable time. He has also found that the passage of the foreign albumen causes irritation in the kidney, especially in the glomeruli; and he infers that the passage of the serum albumen is the result of this irritation, and it is proportional both in time and quantity to the extent of the irritation. He further shows that, if this kidney irritation by the passage of a foreign albumen is kept up for some time, definite structural change takes place with permanent injury to the kidney, and chronic discharge of serum albumen—in fact, nephritis.

He also shows, in reply to those who would be inclined to argue that there is no albuminuria till there is a kidney lesion, however slight, that where there is a foreign albumen in the blood (heteroalbuminæmia), it is excreted not by the kidney only, but by the liver in the bile, by the salivary glands in the saliva, and by the sweat glands in the perspiration; and that the passage of this foreign body causes irritation, and eventually parenchymatous change in the excretory organs concerned; and, further, that when albuminuria is due to conditions local to the kidneys, as conges-

[1] Journal, February 9th, 1889.
[2] See M.D. Thesis, *Formation and Excretion of Uric Acid.* John Bale and Son, 1888, p. 29-30.
[3] Sir W. Roberts's *Urinary and Renal Diseases.* Ed. iv, p. 204; and Professor Grainger Stewart *On Albuminuria.* Edinburgh, 1888, p. 55.
[4] London: Hamilton Adams. 1889.
[5] *Pathologie des Nephrites Chroniques.* Bruxelles. 1886.
[6] *J. de Méd. de Bruxelles.* 1867.
[7] Journal, vol. ii, 1889, p. 419.
[8] *Archives de Physiologie,* 3 l seri s, iv, and elsewhere.

tion, and not to morbus Brightii, then there is no albumen in the bile, saliva, and sweat—this fact serving, as he remarks, to distinguish the one kind of albuminuria from the other.

We have here, therefore, the presence of a foreign albumen in the blood, causing eventually the passage of serum albumen in the urine, and in time setting up a nephritis, which, carried beyond a certain point, progresses independently of its first cause: a nephritis due to hetero-albuminæmia. But, in proving that a nephritis may result from the continued passage of a foreign albumen through the kidney, the important researches of Professor Semmola are by no means at an end, for he goes on to show that the hetero-albuminæmia, which is the cause of the albuminuria and of the nephritis, may be brought about in other ways besides the mechanical introduction of a foreign albumen under the skin.

Thus he shows that in the serum of normal blood there is only about 4 per cent of albuminoids that will dialyse, and that when from any cause this small percentage of dialysable albuminoids is exceeded, albumen at once appears in the urine and other excretions; and, according to him, one of the most potent causes of an excess of dialysable albumen in the blood is a deficient activity of skin function. Thus he says there is a constant relation between (1) activity of skin function; (2) amount of dialysable albuminoids in the blood serum; (3) appearance of albumen in the urine; and he mentions many facts, both clinical and experimental, in support of these statements.

We have, then, according to Professor Semmola, the following sequence of causation: (1) deficient activity of skin function; (2) hetero-albuminæmia, that is, excess of diffusable albuminoids in the blood, these having failed to reach or fallen away from the condition of elaboration necessary for assimilation and the nutrition of the tissues; (3) albuminuria which, if allowed to continue for a certain length of time, sets up changes in the structure of the kidney.

There are, according to Professor Semmola, other organs besides the skin whose deficient action may cause hetero-albuminæmia, as the liver; but the action of the skin is that with which I am specially concerned in this paper.

It is interesting to remember how constant a feature of Bright's disease is deficient skin activity, whatever may be thought of its position in the chain of causation; and Professor Semmola[9] makes a very interesting suggestion when he says that many skin diseases are really attempts on the part of the skin to perform excessive work, and that when the so-called skin disease (psoriasis, eczema, etc.) is "cured" by mechanical means, the skin is foiled in its attempt to do extra work, and an albuminuria or nephritis at once results; and he says that he has seen upwards of fifty cases illustrating this sequence of events, and my impression is that this is no new observation. I have not seen any thorough or effective criticism of Professor Semmola's work, and it would be useless for anyone to attempt to criticise without repeating his experimental work, which, however, it has obviously taken a large part of a lifetime to elaborate.

My object, however, is merely to point out that, taking Professor Semmola's own estimate of the importance of skin function in the causation of hetero-albuminæmia, albuminuria, and nephritis, it is not difficult to see that high pulse tension and albuminuria may be related to each other as cause and effect, and that thus the observations of the late Dr. Mahomed, previously referred to, as well as several other points of which I have still to speak, may receive a simple explanation.

I have several times spoken of the cold extremities and contracted skin vessels which are such common accompaniments of high arterial tension, as during an attack of headache, when it may, as I have said, be impossible to keep the feet warm by anything short of sitting with them in hot water[10]; and this shows, I think, that the cold extremities are not merely the result of external conditions, for if any source of external cold is removed they ought to keep warm; but they do not.

It seems to me, therefore, that the cold extremities and contracted skin vessels are not the cause of the high pulse tension, but its result, the contracted arterioles diminishing the circulation of blood in the skin and extremities. And we can now, I think, see how high pulse tension may be the cause of deficient activity of the skin, and thus, as pointed out by Professor Semmola, of hetero-albuminæmia, albuminuria, and nephritis; and the sequence of chronic nephritis on years of high pulse tension, cold hands and feet, headache, and other troubles of the pre-albuminuric stage, as described by the late Dr. Mahomed, receives an explanation which seems to me to meet all the facts of the case, and also to throw new and important light on the causation of several forms of albuminuria hitherto regarded as functional, and perhaps unimportant.

Not to mention the albuminuria of scarlet fever, in which the skin functions are so obviously interfered with, but in which there may also be other factors, as a poison in the blood, one of the kinds of albuminuria having an apparently simple causation is that which follows a cold bath in an apparently healthy individual.

It may be said that an albuminuria which follows a cold bath is due to failure of the heart and consequent stasis in the kidney, but it is surely an extraordinary thing that the heart should fail at the time of the morning cold bath just after a night's rest; then, beyond the cold and blue extremities, there are no obvious signs of heart failure, but I should have no difficulty in showing that these may be present (as in a uric acid storm) when the heart is acting strongly and well. On the other hand, we often see severe morbus cordis with all the signs of general congestion and disturbance of circulation, and yet the albuminuria from this cause is slight or inconstant. And it seems to me, in a word, that many of the explanations of these so-called functional albuminurias are insufficient and inconsistent with some or more of the facts; while Professor Semmola's work on the importance of skin function in the causation or prevention of hetero-albuminæmia will afford us a simple and consistent explanation of many or all of them. Thus Professor Semmola takes a case of Bright's disease from its very beginning, shows that it took origin in the climate of a cold damp valley, which interfered with the proper performance of the skin functions. He bleeds the patient, and shows that the albuminuria is accompanied by an excess of diffusible albuminoids in the blood serum. He then removes the patient to a drier and warmer place, uses every possible means to increase the activity of the skin function, and further gives milk as a diet, because he considers that its albumiuoids require less elaboration than those from other sources, to render them fit for assimilation and the nutrition of the tissues. It seems to me that there is an interesting point here, for as the mammary gland is essentially a greatly enlarged and highly developed skin gland,[11] one might expect that the albuminoids elaborated in it would not require any further skin action to render them assimilable. And, lastly, when by these means the patient has been brought back to comparative health, and the albuminuria has ceased, he again draws a little blood, and shows that the amount of diffusible albuminoids in the serum has returned to normal.

It seems to me, then, that interference with skin function and hetero-albuminæmia is a much better and simpler explanation of the causation of an albuminuria by a cold bath than those above mentioned. And if interference with skin function by external cold will have this effect, it hardly needs any words of mine to point out that contraction of the arterioles, depriving the skin of its normal blood-supply is likely to have just the same effect. And such a contraction of the arterioles is, I believe, the work of uric acid when it causes on the one hand high pulse tension, and, on the other, cold extremities and contracted skin vessels.[12]

Further, it is obvious that external cold and damp will have more effect on the skin of a person who is subject to this contraction of the arterioles than on one in whom they are generally freely dilatable. And we have here again, it will be observed, a simple explanation of the frequent association of Bright's disease with uric acid.

I have recently been much interested in the writing of Dr. Dukes[13] on the albuminuria of adolescents which is, as he points out, associated with a pulse of high tension.

The causation of this form of albuminuria on the theory that I am here working out is sufficiently simple. Thus—1, high pulse tension, due to uric acid or other poison in the blood, and many of the symptoms seem to me to point strongly to uric acid; 2, deficient skin function from the deficient blood-supply of contracted arterioles; 3, hetero-albuminæmia causing albuminuria, and if this is long continued leading to nephritis.

On the other hand, the explanations given by Dr. Dukes that the albuminuria is due to heart failure is, to my mind, quite inadequate to explain all the conditions. Thus, he says, the albuminuria is removed by lying in bed and a milk diet; or, if kept in

[9] Previous reference p. 312.
[10] JOURNAL, Feb. 9th, 1889, p. 291.
[11] Hermann, *Physiol., Trans. of*, Ed. v, p. 121.
[12] JOURNAL, February, 1889, p. 291.
[13] JOURNAL, vol. i, 1889, p. 625.

bed, any diet will do, but if up and about the patient must have milk only; and from this it will be seen that the heart is equal to its duties on any diet while the patient is in bed, but if he is up and about it is only equal to its duties when he is on milk. Is there any reason to think, then, that the heart of a growing lad is better nourished and innervated on a diet of milk than on an ordinary diet of meat, bread, potatoes, etc.? I know of no reason, and it seems to me that this is a serious objection to the theory of causation by heart failure.

But on the theory that we have to do with hetero-albuminæmia from deficient skin activity, all these difficulties disappear. The patient has no albuminuria while in bed, no matter what diet he is on, for the warm and equable temperature kept up by the blankets all over the body overcomes the action of uric acid on the arterioles, allows of a free circulation of blood in the skin, and an efficient performance of its functions; and I have previously pointed out[14] that exercise causing perspiration, or a hot bath, will overcome the effects of uric acid on pulse tension and the skin in just the same way.

The patient has no albuminuria when he is up and on milk, because, as mentioned above, this is the food which, according to Professor Semmola, requires least elaboration to make it assimilable. Again, in those cases the albuminuria is most marked after meals, and especially after breakfast; and if it is due to heart failure this seems an extraordinary thing, for it is at its worst just at the time one would expect the heart to be strongest, that is, in the morning after a good night's rest, and when it has just been further stimulated by a meal. If the albuminuria were due to heart failure it would probably be worst at the end of the day's work, and apart from the stimulus of food and drink.

If, however, the albuminuria is the result, as I am suggesting, of high tension pulse and consequent hetero-albuminæmia there is no difficulty in explaining the facts observed for the few hours succeeding breakfast, that is, "the alkaline tide," is just that period of the day at which there is generally most uric acid in the blood and the largest excretion of it in the urine, and as a consequence of its presence in the blood most excess of pulse tension;[15] and the same occurs after other meals, as the alkali in the food is being taken up and enters the circulation; but it is always most marked after breakfast, because the acid tide of the night has almost run itself out, and is easily overpowered. And this corresponds exactly with the observed facts regarding this albuminuria, while the heart failure theory does not. Further, after meals there are, no doubt, more albuminoids coming into the circulation that require elaboration by the skin to make them assimilable than at other times.

I have previously pointed out[16] the relation which a uric acid storm, with its contracted arterioles and cold skin and extremities, seemed to me to bear to Reynaud's disease, and also to paroxysmal hæmoglobinuria. And with regard to this latter disease, Professor Semmola considers not only that albuminoids may, owing to deficient skin action, fail to be rendered fit for assimilation by the tissues, but also that albuminoids, previously elaborated, may fall away from the condition necessary for assimilation; so that in an attack of paroxysmal hæmoglobinuria, when the skin vessels are contracted and the surface blue and cold, we may have not only albuminuria, caused in the way I am suggesting, but the process of albuminoid degeneration may be carried still further and affect the hæmoglobin itself.

And there is an experiment which to my mind seems to support very strongly this mode of causation, namely, the hæmoglobinæmia, which can be produced in some people by ligaturing and freezing a finger.

I have for a long time been impressed with the strong resemblance of a paroxysm of hæmoglobinuria to a uric acid storm, as seen in migraine, and described by me in previous papers. And, indeed, I have seen attacks practically indistinguishable from those of paroxysmal hæmoglobinuria occur in the subjects of migraine, apparently in place of an ordinary attack; and in a case of paroxysmal hæmoglobinuria, in which I was able to get the urine excreted at the time of the attack, there was a markedly excessive excretion of uric acid, and the family history, previous health, and many of the symptoms preceding and accompanying the attack were just those of a uric acid storm, as seen in migraine.[17]

[14] JOURNAL, February, 1889, p. 290.

[15] It is interesting to note that in undoubted Bright's disease also more albumen is excreted at this period of the day (Saundby, p. 15 and elsewhere).

[16] JOURNAL, February 9th, 1889, p. 291.

[17] *St. Bartholomew's Hospital Reports*, vol. xxiii.

I do not consider that one or two such cases prove much as to the causation of paroxysmal hæmoglobinuria, but I think that it may be of some interest to consider them here in connection with what I am saying with regard to the causation of albuminuria; for I believe it has been shown[18] that paroxysmal hæmoglobinuria is preceded and followed by albuminuria, so that we have some ground for assuming that both are due to the same cause, and that the hæmoglobinæmia which is the cause of the hæmoglobinuria is only a further more severe stage of the hetero-albuminæmia which is the cause of the albuminuria, not in this case only, but in Bright's disease in general; but I hope to publish a full account of the above-mentioned cases at a future time.

I would also refer to a very interesting paper by Drs. Bristowe and Copeman read at the Medical Society of London on April 29th, 1889, and especially to what is said there of the relationship of paroxysmal hæmoglobinuria to Raynaud's disease. The authors of this paper lay stress on the action of cold in producing these troubles, but they do not explain why cold should affect the blood of their patient differently from that of other people; their patient, however, is an omnibus conductor, aged 41, and liable, if I may so put it, to beer and the gout which I have shown that it produces,[19] and I would like to suggest, in the light of my previous remarks, that uric acid, through its effects on the arterioles and their circulation,[20] was a factor in the production of Raynaud's disease and hæmoglobinuria in this individual by cold; and in the description of his attack[21] the cold extremities, dying fingers, shivering, and headache all correspond to the common symptoms of a uric acid storm as seen in ordinary migraine.

Further, Dr. Bristowe is led to the conclusion[22] that there is no relation between hæmoglobinuria and Raynaud's disease other than that the paroxysms of both are apt to be induced by the same cause, namely, exposure to cold; but, if my explanation is correct, both are due to the contraction of the vessels of the skin and extremities by uric acid, its action being aided and intensified by cold, and thus their frequent association is easily accounted for.

It is also possible that, in addition to the deficient elaboration of albuminoids by the skin of which I have been speaking, the presence of much uric acid or other nitrogenous product of metabolism may have a direct destructive action on the red corpuscles, and this is, I think, no new suggestion; indeed, it has been shown that creatinin has this destructive effect on the corpuscles.[23]

And there are several facts which bear as collateral evidence on the question of the causation of albuminuria by deficient skin action and hetero-albuminæmia, as the frequent connection of albuminuria with scarlet fever, measles,[24] and other diseases affecting the skin.

The relation to dyspepsia,[25] too, is of interest, for dyspepsia has, as I have often pointed out, most intimate causative relations to a uric acid storm, and so to high pulse tension and deficient skin action; but dyspepsia may have a double action, for, apart from its influence on uric acid, it may render some of the albuminoid products of digestion more than usually unfitted for the nutrition of the tissues, and may thus throw extra work on the liver, skin, and other organs of metabolism, already hampered perhaps by uric acid and its effects on the circulation.

Some difference has been noticed between the albumen excreted in the albuminuria of adolescents or in "functional" or "cyclic" albuminuria and that of organic disease; as Dr. Pavy[26] has pointed out that the albumen of cyclic albuminuria is precipitated by an organic acid, while serum albumen is not so, and Dr. Maguire[27] says that the presence of much paraglobulin in the urine is in favour of a functional origin for the albuminuria, but much serum albumen is a sign of organic disease.

It seems to me that in these observations we have in outline the natural origin of nephritis which Professor Semmola's experiments have so closely imitated; in the so-called functional albuminuria we have the hetero-albuminæmia and excretion of a foreign (that is, insufficiently elaborated) albumen, as in Semmola's injection experiments, which is at first irregular and only occurs, as

[18] Dr. Ralfe, *Lancet*, vol. ii, 1886, p. 764.

[19] M.D. Thesis, prev. ref., pp. 6-10, and *St. Barthol. Hosp. Reports*, vol. xxiv.

[20] JOURNAL, February, 1889, prev. ref.

[21] *Lancet*, vol. ii, 1889, p. 256.

[22] *Lancet*, vol. ii, 1889, p. 308.

[23] *Comptes Rendus de la Soc. de Biologie*, 1877, p. 159.

[24] *Lancet*, vol. i, 1889, p. 1001.

[25] Sir W. Roberts, *Urinary and Renal Diseases*, p. 197, and Dr. Ralfe, *Lancet* vol. ii, 1886, p. 764, and JOURNAL, vol. ii, 1886, p. 1012.

[26] *Lancet*, vol. i, 1888, p. 712.

[27] *Lancet*, vol. i, 1886, p. 1052, *et seq.*

shown above, at those times when the excess of uric acid in the blood and the high pulse tension are likely to be most marked, later on if the first stage is not recognised, treated, and cured, we get serum albumen as well as the albumen which is precipitated by an organic acid, that is, as Professor Semmola would put it, the continued passage of a foreign albumen through the kidney has at last caused irritation and structural change, so that it now allows the passage of the normal albumen of the serum as well as the foreign albumen, and if the process is not stopped Bright's disease begins; and this is in accord with the experience of Dr. Johnson and also of Dr. Dukes that many of these functional cases end in Bright's disease.[28]

Professor Semmola's writings and experiments must speak for themselves, but it seems to me that if his views on the functions of the skin in the elaboration of albuminoids are correct we have here a connecting link between the high tension pulse of uric acid and albuminuria and eventual nephritis, which may be of considerable importance.

In this short paper nothing beyond a mere outline of the main points has been attempted, but if the suggestions now made are correct much further evidence of their truth will no doubt be forthcoming.

RESULTS OF THE PORRO-CÆSAREAN OPERATION IN ALL COUNTRIES, FROM ITS INTRODUCTION TO THE CLOSE OF 1888.

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[A Second Contribution.]

A chronological record in progress was given in this Journal for March 30th, 1889, page 708, counting 250 Porro operations for the thirteen years ending with December 31st, 1888. Additions and corrections made in the past six months have enlarged the list of cases for the same period to 264. These have recently been retabulated in the order of their occurrence, and a new analysis made with much care, to show the work of each respective country into which the operation has been introduced. The value of a chronological method of recording is proven by the clearly-established fact shown therefrom, that in all countries, with the exception of France, in which there have been twelve or more operations there can be exhibited a growing improvement in the results attained. Thus, there were six deaths following the first seven Italian operations, and only seven deaths after the last thirty. Italy saved 20 out of her first 46 cases, and 27 out of the next 45. Austria saved 17 out of her first 30 cases, and 25 out of the balance; her second, 30. Following her last 20 operations there were but 2 deaths, or 10 per cent. The early record of Germany was so discouraging that out of it grew the Sänger-Cæsarean method and its modifications. The first 7 cases were all fatal, and but 6 recovered out of 22, or the first half of all those in her credit. Of the second 22 there were 17 recovered; and of the last 15 only 2 died from the operation, a third death having been suicidal. England lost 7 out of her first 8 cases, and then saved 4 in succession. Mr. Tait claims to have had three additional operations without a death, but has not reported the cases; which would enlarge the credit of England to 15, and her recoveries to 8. This would have been a very remarkable success in the early days of the operation, but is much less so now. The late Professor Breisky performed his first operation in Prague on July 9th, 1878, and his eighth and last in Vienna on September 28th, 1888. Not one of his eight operations, covering more than ten years, had a fatal termination, and all of the children were living. Dr. Hubert Riedinger, of Brünn, also aided in a remarkable manner in establishing the credit of Austria. His first operation preceded that of Professor Breisky by twelve days, and his seventh was nine days before the eighth of Breisky. The seven women recovered, and but one child was lost. Up to the last of December he had not performed an eighth operation. France, as already mentioned, has not improved her record by experience. On the contrary, she has retrograded. The first five operations saved three women and three children; the second five, one woman and five children; the third, one woman and two children, and the two additional saved one woman and two children. There has not been reported one French Porro case belonging to the past four years, and the operation would appear to have been abandoned by its early advocates.

[28] Grainger Stewart, prev. ref., p. 168.

Seventeen operations belonging to the last four years, ending on December 31st, 1889, have been reported to me in answer to letters, of which four terminated fatally. The records of these four years are respectively as follows: 1885—24 cases, 7 deaths; 1886—23 cases, 4 deaths; 1887—18 cases, 5 deaths, 1 suicide; 1888—28 cases, 4 deaths. Of the first 50 women out of the 264 cases recorded 30 died as the result of the operation; and of the last 50 but 10, a reduction of the death-rate from 60 per cent. down to 20.

Of the whole 264 women 117 died from the operation, divided as follows: 74 out of the first half, that is, 132, and 43 out of the second 132. Thus it will be seen, under several forms of calculation, that the Porro operation of to-day, under proper management, is far less fatal than that of five years ago, as in 1884, when 17 women died out of 29, or nearly 58 per cent.

As the day of experiment has passed, and the technique of the operation has been established by experience, there is no reason why there should not be a large percentage of cases saved—certainly 80 per cent.—and with due care as to time of operating and condition of strength in the patient, even more than this. The operator ought to be able to make a prospective prognosis in a given case, and should, if possible, so order the preliminary steps that "unfavourable" will not have to be written in rating the prospects of the patient. It is a well-established fact that few cases rated as "favourable" before the operation have died, and that still fewer marked as as "unfavourable" have not. The respective credits of the several countries in which the Porro operation has been performed (not including that of 1889) stand as follows:—

No.	Country.	Cases.	Saved.	Lost.	No.	Country.	Cases.	Saved.	Lost.
1	Italy ...	91	47	44	8	Belgium ...	5	3	2
2	Austria ...	60	42	18	9	Scotland ...	5	1	4
3	Germany ...	44	23	20*	10	Switzerland	6	5	1
4	France ...	17	6	11	11	Australia ...	2	2	0
5	England ...	12	5	7	12	Holland ...	1	1	0
6	Russia ...	10	8	2	13	Spain... ...	1	0	1
7	United States of America	8	2	6	14	Mexico ...	1	0	1
					15	Japan... ...	1	1	0

* And 1 suicide.

It is proper, in this connection, to state the results of the conservative Cæsarean section up to the same date, January 1st, 1889, as many may be under the impression that the Porro operation is quite equal to it in its ability to save life. Like the latter, the new method of performing the old Cæsarean section is also passing through a stage of diminishing mortality, and there is a very marked contrast for the better between the work recently done and that of the years 1882, 1883, 1884, and 1885; and particularly is this the case in the United States of America. Out of the first 20 cases operated upon in all countries, there were 9 deaths; out of the second 20, there were 6; and of the last 40 in 1888, there were only 6. The following table will show the operations in contrast:—

No	Country.	Cases.	Saved.	Lost	No.	Country	Cases.	Saved.	Lost.
1	Germany ...	94	81	13	7	Holland ...	9	9	0
2	Austria ...	32	26	6	8	England ...	3	1	2
3	United States of America	26	11	15	9	India	2	1	1
4	Russia ...	10	7	3	10	Switzerland...	2	1	1
5	France ...	4	2	2	11	Denmark ...	1	1	0
6	Italy ...	4	2	2	12	Norway ...	1	1	0

There were 57 operations in Europe in the year 1888, with 7 deaths; and 13 in the United States of America, with 8 deaths. There have been 34 operations in America, with 18 deaths.

Professor W. R. Smith, M.D., D.Sc., F.R.S.Ed., barrister-at-law, announces a special course of lectures in Hilary Term to barristers, law students and others at King's College on Mondays, Wednesdays, and Fridays at 4 P.M. We referred recently to a series of lectures on forensic medicine by Professor Meymott Tidy to the law students of the Middle Temple. Both the legal and medical professions have much to gain by the adoption of these precedents.