THE GULSTONIAN LECTURES

ON
ANÆMIA.*Delivered before the Royal College of Physicians.*BY SIDNEY COUPLAND, M.D., F.R.C.P.,
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LECTURE III.—PART II.

Fatty Degeneration: The most important Result of Anæmia—of Heart—of other Muscles—of Vessels—of Viscera.—Liability to Hemorrhages explained by Vascular Deterioration.—Treatment of Anæmia: Hygienic—Dietetic—Medicinal—Transfusion.

THE frequency with which fatty metamorphosis of the tissues and an anæmic state of the blood are associated is such that the connection between the two must be an intimate one. Whenever a tissue is deprived of nutriment, it undergoes this change, its albuminous particles becoming converted into fatty granules and oil-globules. It is the simplest instance of retrograde metamorphosis of the tissues, and occurs as a physiological event in their natural decay. Pathologically, although no tissue is exempt from this form of degeneration, the muscles are particularly prone to it, and this proneness is no doubt due to the muscles being so actively employed in the metabolic processes of the body. Deprived of their due supply of oxygen, their protoplasmic elements are unable to go through their normal changes; and, instead of complex stable products, they break up into fatty molecules. Such a conversion may be effected with remarkable rapidity: witness the wide-spread metamorphosis in acute poisoning by phosphorus, which may be established within a day or two;* and in such case I believe the change is effected by the deprivation of oxygen, and the disturbance to the normal tissue metamorphosis thereby established. More often, there is reason to believe that the process is of slower duration; but, important as it is to determine this question, I do not think it possible to do so yet. There may be links still wanting in the chain of knowledge; but it is, to my mind, one of the most reasonable facts in pathology, that, whenever the blood is impoverished, the tissues must suffer degeneration, and it is important not to disregard the fact in the treatment of anæmia. The muscle of the heart—which of all muscles requires a constant and definite supply of oxygen for its renewal, its unceasing action denoting continual metamorphosis—is particularly liable to suffer. Nourished probably in two ways, by the blood circulating through its cavities and by the arterial supply to its walls, is there not reason enough why this muscular organ should be especially singled out to undergo this metamorphosis when the blood is deficient in oxygen-bearing elements? Hence it is that the occurrence of true fatty degeneration of the heart is recognised as the most constant and most striking effect of a grave and prolonged anæmia. Out of seventy-six cases of fatal idiopathic anæmia, where *post mortem* examinations were made, in sixty-four it is expressly stated that the heart was thus degenerate, sometimes extremely so; in six other cases, no mention is made of its condition; and in only six is it said to have been healthy. This degeneration of the heart is, in fact, as I pointed out in my last lecture, the only constant anatomical feature of cases of idiopathic pernicious anæmia. It was originally pointed out by Addison, and the observation was abundantly confirmed by Wilks; and its occurrence has been noted, as above seen, with few exceptions, in every case of fatal anæmia since attention was drawn to it. Wagner, in his treatise on Fatty Degeneration of the Heart, attributes some cases to anæmia, and Ponfick described in detail the “anæmic form” of fatty heart. There is, then, general concurrence of opinion upon the association; and it is probable that it has wider bearings than we generally admit. The recognition of this sequence, as a pathological fact, may possibly, in time, throw light upon cases of cardiac degeneration at present thought to be idiopathic—cases, that is, which do not arise secondarily to inflammation of the pericardium and myocardium, from disease of the coronary arteries, from alcoholism or from fever, or, as a final event, in long-standing valvular disease. It is hardly conceivable that an impoverished state of blood should follow upon an enfeebled fatty heart; at any rate, such an impoverishment as to fall under the head of anæmia. It is far more probable that the degeneration is the result of the blood change rather than the cause of it.

See case recorded by myself in the *Lancet*, vol. ii, 1879, p. 309

There are many cases where the degenerative powers have been apparently of rapid evolution—cases of acute anæmia following loss of blood. As an example of this, I might instance the case related in my last lecture (No. 6), where profuse and uncontrollable uterine hæmorrhage supervened in a chlorotic subject, and where the heart was found to be most extensively degenerated. Here is another case, where one factor, at least, in the production of the degeneration was a similar loss of blood. A woman, aged 46, was admitted under my care into the Middlesex Hospital, on October 23rd, 1880, suffering from general dropsy dependent upon a dilated and degenerate heart. She had approached the climacteric, having previously passed a healthy life; but, for the past five years, the catamenia had been most profuse; she called it “flooding”. Three months before admission, the menses apparently ceased; to reappear in great excess during the past fortnight. Edema of the legs began about one month before, followed by swelling of the belly; but, prior to this, she had been getting pale, and suffering from palpitation and breathlessness on exertion. She was somewhat obese, very anæmic, and the conjunctivæ were yellow-tinged. Accounting for the dropsy, there was evident dilatation of the heart, with epigastric impulse, a soft systolic apex *bruit*, and irregular intermittent pulsation. She suffered from edema of the lungs; and treatment had no effect in reducing the dropsy or removing the anæmia, which even grew more intense, although no menorrhagia occurred whilst she was in the hospital. After her death, which took place on November 12th, the heart was not only overlaid with fat, but its muscular walls were flabby and extremely degenerate. The kidneys were also fattily degenerated. Here, then, was a case (and it is one of a class) where the cardiac degeneration was the most pronounced feature, and was the immediate cause of death, but where it seemed probably related to, if not wholly dependent upon, the menorrhagic attacks to which she had been subject. Dr. Green has recorded a striking instance of the effect of profuse menstrual loss in inducing fatty degeneration, in the eighth volume of the *Clinical Society's Transactions*. The case is that of an anæmic ill-nourished girl, the subject of mitral stenosis. The occurrence of menstruation, after an interval of six weeks, and the profuseness of the flux (possibly made so profuse by the presence of the mitral disease), so lowered the standard of the blood, that, within five days, she died in a state of exhaustion. In addition to the mitral disease and an abnormally narrow aorta, the muscular tissue of the heart was found to be in an advanced state of metamorphosis, and similar regressive changes were met with in some of the voluntary muscles. Similar examples might be quoted. The degeneration of voluntary muscles observed in Dr. Green's case is very interesting; and it is probable that, if such muscles were more frequently examined in anæmia, they would be found occasionally affected.

One may, without transgressing the rightful limits of pathological speculation, attribute the origin of the areas of fatty degeneration observed by Virchow in the lining membrane of the aorta in cases of chlorosis to the same cause—the impoverishment of the blood. The well-known experiments of Cohnheim bearing upon the embolic process demonstrated the fact that the integrity of the walls of the blood-vessels depends upon their being filled with a due amount of healthy blood. The tendency to capillary hæmorrhages—purpuric spots, ecchymoses beneath serous membranes, and sometimes parenchymatous hæmorrhages—in pernicious anæmia is, I believe, due in great measure to such vascular deterioration. It has not always been found, however, in such cases; but I may say that the only opportunity of which I availed myself of examining the retinal vessels in a case of this disease, where there were marked hæmorrhagic foci in the retina, enabled me to distinguish clearly that the endothelial cells of the small arteries, and those of the capillary walls themselves, were studded with minute oil-globules. That such ecchymoses are due to the condition of the blood only is far less probable than that they are caused by actual damage to the vessel-walls through which the altered blood circulates; and, when there is evidence of such vascular deterioration, this explanation seems to be all-sufficient. This, however, like that of the condition of the voluntary muscles, is a point which calls for further investigation.

Fatty degenerations of the gastric and intestinal glands, of the liver, and of the kidneys, are met with in a certain number of cases of profound and fatal anæmia; but how far, and in what way, whether as results or as causes of the anæmia, it does not seem possible yet to say. Certainly, not one of these organs may be so affected; they are not so often found degenerate as the heart-muscle. The gastric glands are not often examined, and their derangement is possibly a factor in the production of the anæmia; whilst the intestinal and hepatic cells are liable to undergo a physiological fatty infiltration, which must not be confounded with a morbid state. In one of the cases previously related (Case 5), the liver, though certainly fatty, was very remarkable, the cells affected being those nearest to the centre of the lobule, and

not at the periphery, as in the ordinary fatty infiltration-process. Some of the most advanced cases of visceral fatty degeneration which I have seen have occurred in cancerous subjects, especially uterine cancer, which more or less rapidly progresses in ulceration, impoverishing the blood more and more as it advances. Such instances of course differ from cases of phthisis, where there is pyrexia and direct respiratory disorder to aid in the production of the fatty change.

I am not, however, now concerned with the subject of fatty degeneration as a whole, but only in relation to anæmia. We have, I think, ample evidence that there is a link between the blood-condition and the tissue-change; and everything points to the latter being the result of the former. At the same time, we must not lose sight of the other alternative to which I have above alluded, for I do not think it is absolutely capable of disproof, at any rate in every case. I refer to the view that the anæmic condition is itself dependent upon what may be called an "inertia" on the part of the tissues, whereby they do not perform what is required of them, and, failing to go through their normal metamorphosis, pass into the fatty regressive state. In such a case, there would be less demand for oxygen, and this would react upon the supply, and fewer corpuscles and hæmoglobin would be formed. Such an hypothesis requires, however, far more than the former, which has also received support from experimentation, a continued loss of blood being found to produce fatty metamorphosis.

The treatment of anæmia, apart from the removal of its cause, when this can be ascertained, may be summed up under the head of hygienic, dietetic, and medicinal measures. On the first and second of these, I need hardly dwell; their importance is self-evident. The necessity for rest, alternated with moderate exercise, graduated according to the severity of the anæmia, is important. Even slight exercise is irksome and harmful until the blood is sufficiently reinforced to permit of it; and absolute rest is called for at first. The influence of air and light is of as much value in aiding restoration from the anæmic condition as their deprivation is powerful in inducing this state; and such hygienic measures as bathing act by promoting tissue-metabolism. A valuable contribution to the treatment of anæmia by these means has lately been made by Drs. M. P. Jacobi and White (*Archives of Medicine*, August and October, 1880), in what is a scientific exposition of the effects produced by those who practise hydrotherapy. These writers relate cases, accompanied by analyses of the urine, where wet packs and *massage* were employed, in addition to ferruginous medication. The effect of such treatment was to cause increased elimination of urea, to improve appetite, and to procure sleep; and the explanation afforded is, that the treatment, acting through the nervous system and the circulation, promoted tissue-change, and increased the capacity of the tissues and the blood for oxygen, the passive exercise of the muscles aiding also, by quickening the circulation, in the removal of the products of nitrogenous waste.

In diet, the indication is to allow meat in considerable quantity as soon as the digestive organs are capable of the work imposed on them; but where, as so often happens, the digestive power is at a minimum, then recourse may be had to meat-essences, peptonised foods, and (as an extreme measure) nutrient enemata. There is frequently a great distaste for nitrogenous food among these subjects, many of whom owe their anæmia in large measure to their preference for a diet of non-nitrogenous materials; and it follows, therefore, that chief attention should be paid to this point, especially as the organs of digestion are more or less deranged by the anæmic state. As I have before pointed out, digestive derangements both induce and are induced by anæmia; and, by dealing with them, we often strike at the root of the condition, which, however, requires also measures directed specially to it. These measures comprise the administration of certain drugs, which are comprised under the term "hæmatinic". Of these I pass now to speak briefly.

Of all remedies, iron maintains, as it always has maintained, a pre-eminence in the treatment of anæmia. It seems to act directly upon the blood-forming viscera, and probably also upon the digestive organs—*i. e.*, on the gastro-intestinal tract. No wholly satisfactory explanation has, I believe, been given of the mode of action of the iron in either of these directions, or of the reason why the best results in the most marked cases of chlorosis are obtained by the administration of heroic doses far in excess of what is actually required in blood-formation, and much more also than is actually absorbed. Trousseau called iron as much a specific in chlorosis as mercury was in syphilis; and there was truth in the remark, although we may not know in what its specific action precisely consists. The application of methods of blood-measurement enable us to estimate the rapidity with which iron hastens recovery from the anæmic condition. Sometimes it appears to act, at any rate in the first instance, by causing an increase in the number of corpuscles without a corresponding increase in the amount of hæmoglobin

(Gowers, Baxter, and Willcocks); but the reverse has also been noted (Hayem); and, in a series of cases published by Moriez, from M. Hayem's *clinique*, the increase in hæmoglobin was marked from the first, and, of course, greater than the increase in the number of corpuscles, which was high at the commencement. Moriez gives several tables illustrative of the effects of this and other modes of treatment, using the symbols N, R, and G to represent respectively the number of the red corpuscles per cubic millimetre—their richness, *i. e.*, the number of corpuscles in healthy blood to which they are equivalent, and their functional value or quality, G being equal to $\frac{R}{N}$. For the normal standard to be reached, not only should the figures R and N be equal, and of the full amount (*i. e.*, 5,000,000 per cubic millimetre), but they would then contain the same amount of hæmoglobin, when G of course = 1. The mean results of twelve cases thus tabulated gave before treatment (by iron), N = 4,031,745, R = 1,991,318, and G = 0.49; after treatment, N = 4,747,675, R = 3,940,000, and G = 0.83. I give the results of similar estimations in a few cases lately in the Middlesex Hospital (see Table), the most remarkable being No. 4, where the percentage of hæmoglobin was doubled within ten days. This case was treated with sulphate of iron and bicarbonate of potash; and it will be seen that, although the number of corpuscles was but slightly affected, their functional value rose above the normal. Unfortunately, no numeration was made on December 24th, when the actual amount of hæmoglobin was much higher still; so I cannot say whether in point of number the corpuscles in this case still remained so much below the normal.

Cases of Anæmia treated by Iron and Arsenic.

No.	Date.	Number of Corpuscles.	Hæmoglobin.	Quality of Corpuscle.*	Treatment.
1	Jan. 6th	2,090,000	—	—	Ferri sulphas
	" 14th	2,750,000	32 per cent.	0.57	"
	" 27th	3,170,000	42 "	0.66	"
	Feb. 2nd	3,600,000	—	—	"
2	Jan. 12th	4,050,000	45 per cent.	0.55	"
	" 26th	4,430,000	52 "	0.53	"
3	June 16th	2,400,000	—	—	Ferri arsenias
	July 8th	3,300,000	—	—	Quin. cum ferro
4	Dec. 1st	3,700,000	40 per cent.	0.54	Ferri sulphas
	" 11th	3,800,000	80 "	1.05	"
	" 24th	—	104 "	—	"
5	Dec. 3rd	3,800,000	52 per cent.	0.68	Liq. arsenicalis
	" 28th	4,060,000	80 "	0.93	"
6	Feb. 2nd	2,490,000	23 "	0.56	"
	" 8th	2,800,000	39 "	0.70	Ferrum dialysatum
	" 21st	3,700,000	39 "	0.52	on & after Feb. 10th

Under the use of iron the corpuscles, according to Hayem, also increase in size, and the large number of hæmatoblasts which he finds in the blood of chlorotics appreciably diminish during the treatment. These methods of blood-measurement might be applied to discriminate between the efficacy of the different salts of iron, and the different modes in which it is administered—from the waters of a chalybeate spring to the concentrated doses in which we often give it medicinally. Indeed, as to the form and mode of the administration of iron, I can quite confirm the opinion of those who regard the sulphate as the most rapid in its effect, and who urge its administration in large doses. It is remarkable how well it can be borne, and for my own part I am content to prescribe it in almost every case of pure chlorotic anæmia. At the same time, we possess so many different preparations of different degrees of astringency, that there are scarcely any cases in which some form of this metal is not tolerated.

Next to iron, and in some forms of anæmia to be preferred to it, is *arsenic*. This metal is a powerful hæmatinic. It acts on the tissues as well as on the blood and blood-forming organs; and its effect upon the cutaneous epithelium has been actually demonstrated. It is almost the only drug which has been successful in the treatment of severe idiopathic anæmia, which more often resists all medication. The patient under Dr. Cayley's care, where the anæmia was most profound and the corpuscles had fallen to a little less than one-half the normal, notably improved under arseniate of iron. A case was recently recorded from St. Mary's Hospital, under the care of Dr. Broadbent (*BRITISH MEDICAL JOURNAL*, September 25th, 1880, p. 508), which is a good illustration of the efficacy of arsenic. A woman, aged 42, who had

* Calculated by dividing the corpuscular richness by the actual number of corpuscles, the amount of hæmoglobin being the indication of the former.

been progressively anæmic for about four months, and was in an extreme condition when admitted (estimated number of corpuscles two days later being 560,000 per cubic millimetre, or 11.2 per cent.), was placed upon liquor arsenicalis with digitalis. The dose of the liquor was increased from two to four minims, and was administered six times daily. In two months' time the number of corpuscles had increased to 64 per cent. In the remarks appended to the report of the case, it is held "that there can be little doubt that it was of essential or pernicious anæmia; the patient had the appearance characteristic of this disease, and the subfebrile temperature; while the red corpuscles of the blood were not only reduced in number to an unusual degree, but deformed. Whether this diagnosis be accepted or not, the failure of iron to do good, and the rapid improvement during the administration of arsenic, are remarkable. In little more than two months the patient passed from extreme anæmia to apparently perfect health, with wonderfully good colour of the cheeks and mucous membranes, and she continued well and strong for some months after leaving the hospital, up to the time when she ceased to present herself for examination". Such cases as

this speak strongly in favour of the value of arsenic. I have sometimes prescribed it in simple anæmia (see Table, Nos. 5 and 6) with good result, but as a rule one prefers the use of iron, which acts more promptly and fully. Phosphorus has been sometimes administered with success in essential anæmia; as, for example, by Dr. Broadbent in a case where iron had failed; but it has, on the whole, but few, if any, advantages over arsenic, and its long-continued administration is to be deprecated.

It was at one time thought that manganese could replace iron as a hæmatinic, when that metal had failed to influence anæmia; but it never gained much favour, and has fallen into disrepute. Indeed, from some recent observations of M. Hayem, referred to by Moriez, manganese would appear to have no influence in increasing either the red corpuscles or the hæmoglobin, at any rate in the right direction. Its administration appeared to diminish the effect of iron, which was given subsequently. Another treatment that has been sometimes prescribed is that of inhalations of oxygen. The same observers have studied the effects of this treatment also, and declare that although it

CASES OF PERNICIOUS ANÆMIA TREATED BY TRANSFUSION.

No. of Case.	Sex.	Age.	Duration of Symptoms.	Leading Symptoms other than Anæmia.	Method of Transfusion.	Effects of Transfusion.		Result.	Reference.
						Immediate.	Remote.		
1	F.	34	6 months	Great debility; anasarca; slight ascites; diarrhoea; albuminuria; retinal hæmorrhages	—	—	Splenic and renal infarction	Death	Quincke; Volkmann's <i>Sammlung</i> , No. 100, Case 1.
2	M.	35	18 months	Apathy; anorexia; vomiting; constipation; dyspnoea; retinal hæmorrhages	Lamb's blood	Appearance of numerous small corpuscles in blood	Albuminuria; pepticiæ; embolism of Sylvian artery	Death 3 days after transfusion	Iditto, Case 3.
3	F.	34	6 years ailing, 1 year worse	Vomiting; constipation; moderate pyrexia; retinal and cutaneous hæmorrhages; epistaxis	Defibrinated human blood into radial artery	—	—	Death 6 hours after	Iditto, Case 9.
4	F.	56	4 months	Great debility; delirium; vomiting; diarrhoea; albuminuria (slight); retinal hæmorrhages	Iditto (patient moribund)	—	—	Death ¼ hour after	Iditto, Case 10.
5	F.	33	5 months	Amenorrhœa 4 years; great debility; dyspepsia; constipation; albuminuria (slight); retinal hæmorrhages	Iditto 85 cubic centimetres into left radial artery	Collapse, and sense of cold for 3 hours; abdominal pain and diarrhoea	Pyrexia (see Chart No. 2); hæmoglobinuria	Recovery	Iditto, <i>Deutsches Arch. f. Klin. Med.</i> , xx, Case 11.
6	M.	43	2 months	Dyspnoea; palpitation; tinnitus; pepticiæ; epistaxis; retinal hæmorrhages; occasional diarrhoea	Iditto 50 cubic centimetres	Pain and diarrhoea for short time; temperature 102.5°, 2 hours after	—	Recovery	Iditto, Case 12.
7	F.	47	15 months	Great wasting; general œdema; no hæmorrhages; diarrhoea	Iditto 80 to 100 cubic centimetres	Temporary rally	—	Death 12 hours after	Iditto, Case 13.
8	F.	16	1 year	Occasional vomiting; diarrhoea; general œdema; amenorrhœa; retinal hæmorrhages; stomatitis; temporary albuminuria	Iditto 105 cub. centimetres	No marked disturbance	—	Recovery, but subsequent relapse, and death from nephritis	Iditto, Case 15.
9	F.	34	4 months	Emaciation; slight œdema; anorexia; diarrhoea; no hæmorrhages	Iditto 100 cub. centimetres	Rigor; involuntary defæcation	Temp. 102.2°, two hours after; pulse 160 (from 84)	Recovery	Iditto, Case 16.
10	F.	21	6 months	Great œdema following parturition; diarrhoea; occasional vomiting; retinal hæmorrhages	Iditto 25 cubic centimetres	None	—	Death 10 weeks after	Iditto, Vol. xxv, Case 22.
11	F.	39	?	Wasting; profuse diarrhoea following abortion (sixth month), four days before transfusion	60 grammes into brachial vein	?	—	Death 2 days after	Iditto, Case 24.
12	F.	30	Some months	No notes	80 cubic centimetres — arterial	—	Great weakness, and loss of appetite	Recovery	Iditto, Case 25.
13	F.	36	3 months	Weakness; vertigo; palpitation; diarrhoea; retinal hæmorrhages	Iditto 60 cubic centimetres	Retardation of pulse & respirations during and after vomiting; pain and involuntary stools	Rigor, with rise of temp. to 104.9°; hæmoglobinuria	Recovery	Iditto, Case 26.
14	M.	21	2 months	Weakness; vomiting; epistaxis	"Transfusion to 7 ounces"	—	Peri- and endocardial ecchymosis	Death	Taylor, <i>Guy's Hosp. Rep.</i> , 1878, Case 21.
15	M.	50	4 months	Weakness; irregular pyrexia	On two occasions, with 6 days' interval; venous—5½ ounces	Retardation of pulse, followed by chills; rise in temp. to 104.5°; vomiting. 2nd time, vomiting, involuntary defæcation	Delirium came	Death 15 hours after second transfusion	Pepper, <i>Amer. Jour. Med. Sci.</i> , 1875, Case 3.
16	F.	29	8 months	Eight months pregnant; dyspnoea; thirst	180 grammes of human blood	—	Abortion day after, with loss of 330 grammes of blood	Death 2 days after	Gusserow, cited by Lëjine, <i>Rev. Mens.</i> , 1877, Case 22.
17	F.	24	5 months	Eight months pregnant; vomiting	Small amount only transfused	—	—	Death	Iditto, Case 23.
18	F.	28	?	Seven months pregnant; vomiting; diarrhoea; abortion at eighth month; irregular pyrexia	Performed in <i>extremis</i>	—	—	Death	Ferrand, <i>Méd. Soc. des. Hôp.</i>
19	M.	29	Rheumatism 8 years before; worse 3 weeks	Edema; diarrhoea; purpura; retinal hæmorrhages; vomiting	11 ounces of defibrinated human blood into cephalic vein	Pulse-rate fell from 124 to 102	—	Death 7 days after	Unpublished, under care of Dr. Wilks, Guy's Hospital.
20	M.	40	1 year	Weakness; pains in limbs; giddiness; no hæmorrhages; improved in two months under arsenic; returned five months later, with vomiting, dyspnoea; anasarca towards close	On second occasion was twice transfused	—	—	Death	Unpublished, under care of Dr. Pye-Smith, Guy's Hospital.

increases the appetite and the power of assimilation, it has no influence in increasing the richness of the blood in hæmoglobin (Moriez). Lastly, quinine and strychnine, although not hæmatinics in the strict sense of the word, are very valuable as adjuvants; and the same may be said of the mineral acids and bitters.

The subject of *transfusion* of blood is one which demands the most thorough examination in reference to the treatment of anæmia. In some forms of symptomatic anæmia, especially when the cause is a sudden loss of blood, there can be no question either as to the propriety of transfusion or its efficacy in saving life. It is another matter, however, when we have to deal with the insidious and progressive anæmia, which has been denoted by the term "pernicious". Transfusion has not yet been employed to a very large extent in such cases, and it has often failed of success. I find that it was performed in 20 out of 110 cases, to which I referred in my last lecture, and that the six recoveries (one of which was not permanent) all occurred in Quincke's practice.

I think his success in these cases may be attributed to his having resorted to transfusion at a less advanced stage of the disease than is usually the case. His first cases were invariably fatal, and in his commentary upon them he pointed out the inutilty of deferring transfusion until the heart is already advanced in degeneration, and restoration is well nigh hopeless. In one of his papers, where he summarises the results of five such transfusions, he says that defibrinated human blood was used, and from 80 to 100 cubic centimetres injected into the radial artery. (Professor Schäfer, in his valuable report to the Obstetrical Society of last year, advocates immediate injection from artery to artery as far preferable to intravenous injection, whether mediate or immediate. It will be seen that Quincke performs an operation the same as regards the recipient of the blood; but he uses defibrinated blood, and does not practice the immediate transfusion.) Usually, during the transfusion, the face becomes flushed, the pulse slow, and sometimes very weak, almost a state of collapse occurring. In three cases a rigor, followed by fever, occurred a short time after the operation. On two occasions the transfusion was followed by hæmatinuria and casts in the urine, but not by the escape of actual blood-corpuscles. In one of these cases the amount of hæmoglobin passing off by the urine was estimated at one-sixteenth of that injected; and Quincke points out that similar effects follow, according to Ponfick, the transfusion of an animal with blood from another of different species. In some cases petechial and other hæmorrhages follow transfusion. Quincke seems to be firmly of the opinion that by transfusion the blood is reinforced with functionally active elements, and the results of his cases apparently bear out that opinion. Transfusion, then, is a correct and rational procedure, and one which we are bound to adopt in these cases of progressive idiopathic anæmia, if no ordinary treatment makes any impression on them. But its results can never be expected to be crowned with such success as in cases of acute anæmia following hæmorrhage, for the simple reason that we are dealing with a disease the origin of which lies deep in the organism, and the effects of which are already manifest in grave degenerative changes in the heart. Perhaps the less heroic measure of administering enemata of defibrinated ox blood, which was advocated by the Therapeutical Society of New York as of great value in conditions of exhaustion and anæmia, may be found of service. At any rate, it deserves the careful trial which Dr. Sansom in this country has claimed for it.

It only remains for me to thank you for the kind attention you have given me during this imperfect attempt to illustrate the leading features of anæmia. I have consciously left many points untouched, whilst many of which I have spoken are capable of much fuller analysis than I have attempted. For all the shortcomings of what has been little more than an abstract of the present state of knowledge on this subject, I ask your indulgence.

VASELINE AND ARTIFICIAL EYES.—Dr. Chas. S. Turnbull recommends pure vaseline to all wearers and fitters of artificial eyes. It is infinitely superior to pure water, oils, or unguents, as a lubricant, not only for daily use, but also for facilitating the tedious and irritating process of fitting and matching. It lessens wear and tear. It allays the burning sensation often experienced toward night. It is of particular value in preventing the drying of mucus upon the surface of the eye, an occurrence most annoying to wearers, as well as to their surgeons; and last, but not least, it serves as a convenient and desirable vehicle for the application of anodyne or astringent remedies. It should be applied once or twice daily, according to the employment of the wearer; and by mechanics, mill hands, persons working out of doors, or in dusty or dry atmospheres, has been found of more than ordinary value.

ON PARALYTIC CHOREA.*

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My object in this paper is to direct attention to a variety of chorea which sometimes presents a difficulty in diagnosis.

In an ordinary well-marked case of common chorea, we may usually recognise three symptoms: spontaneous movement, inco-ordination of voluntary movement, and muscular weakness. These three symptoms are not always proportioned in degree; any one of them may so predominate as to give a special aspect to the case. The form which I am about to describe is one in which the last element—muscular weakness—predominates, and in which the other elements—muscular spasm and inco-ordination—are so slight, as to be apparently absent; so that the muscular weakness appears to be the only symptom.

I will first describe briefly the characters of a few examples of this form of affection.

CASE I.—John F., aged 14, applied for treatment in July 1879, because he had "lost the use of his left hand". The boy presented the characteristic signs of inherited syphilis: scarred mouth, one notched incisor, cicatrices of old ulceration on the uvula, iritic adhesions in the left eye, and evidence of previous choroiditis in both eyes. The left arm hung by his side. He could move it by an effort, but it was considerably weaker than the right, and this also was weaker than normal. The weakness was said to have come on gradually about a fortnight previously. There was no paralysis of face or leg. On first examination, no twitching or choreic movement could be observed. Voluntary movements were perfectly steady. On subsequent close watching, however, an occasional slight movement of the thumb, or of pronation, was observable in the right hand, but none in the left. When he held both arms straight forward for a time, a slight occasional twitch was noted in each hand. He was given iodide of potassium and strychnia. A week later, the left arm was stronger, and could be used better. It did not prevent a greater degree of twitching. The spontaneous movements in the right arm had increased considerably, and now presented a distinct chorea-like character. He did not, however, present himself again.

CASE II.—Jane P., a girl aged 13, was brought on June 9th, 1879, having, it was said, "lost the use of her left arm" for three months; the onset of the weakness had been gradual. The arm was considerably weaker than the other, the grasp being less than half the strength of the right. Close examination did not reveal the slightest spontaneous movement. It was, however, ascertained that, earlier in the affection, she would occasionally drop things out of the hand, although no history of distinct choreic movements could be elicited. She was said to have had rheumatic fever at four years of age. The heart was healthy. She gradually, but slowly, regained considerable power of using the arm; but with the power, slight spontaneous movements appeared, and then gradually subsided. The symptoms had only disappeared four months after the commencement of treatment, so it may be doubted whether this exercised much influence. First strychnia, and afterwards arsenic, were given.

CASE III.—Emma D., aged 9, was brought on May 1879, on account of weakness of the left arm, which hung useless by her side. She never attempted to use it, although by an effort she could grasp with slight force. The weakness had come on gradually, four months previously. Not the least spontaneous movement could be observed in it. Co-ordination of movement was perfect. Nevertheless, when she grasped the hand, it could be felt that the movement of the fingers was not quite equal. In the legs, especially the left, there was slight twitching. Inquiry elicited that, during the first few weeks of the affection, there had been more spontaneous movement, and that this had lessened as the weakness came on. There was a loud mitral murmur. Strychnia was given, and in a month she was practically well, although the power of the arm was not quite so good as that of the other.

CASE IV.—Ellen B., aged 14, brought on March 15th, 1880, on account of "loss of the use of her left arm", of a few weeks' duration, and gradual onset. The arm hung by her side, and she made no attempt to use it. She could by an effort raise it, but with very little power; and the hand was so feeble, that no sensible pressure could be exerted in the grasp. The leg was strong. The face presented no weakness; the tongue no deviation. At first, no movements

* A paper read before the Section of Medicine at the meeting of the British Medical Association at Cambridge, August 1880.