BRITISH MEDICAL JOURNAL:

BEING THE JOURNAL OF THE BRITISH MEDICAL ASSOCIATION.

EDITED FOR THE ASSOCIATION BY ERNEST HART.

LONDON: SATURDAY, JULY 5, 1890.

OBSERVATIONS

ON

THE TREATMENT OF PERNICIOUS ANÆMIA BASED ON A STUDY OF ITS CAUSATION. WITH NOTES OF A CASE.

By WILLIAM HUNTER, M.D., M.R.C.P.,

John Lucas Walker Research Student, University of Cambridge; Arris and
Gale Lecturer, Royal College of Surgeons.

NOTES OF A CASE.

THE patient, a man, aged 59, in good circumstances, came under my observation on May 9th, 1889, suffering from anæmia and profound weakness, which had already confined him to his bed for three months.

Previous Life History.—With the exception of an attack of ague which he had had as a youth while studying on the Continent, and some gastric affection for which he was under treatment for a short time about six years previously, his health had up to the onset of his present illness been exceptionally good. His present illness dated from the autumn of 1886. During a holiday in the country he was exposed for a time to unhealthy sanitary conditions in the house in which he stayed, and he suffered at the time from sore throat and diarrhoea. From that time onward he never entirely regained his former health. He continued to suffer at times from an inflammatory condition of throat and tongue, and his strength began gradually to fail, so much so that late in 1887 he was induced to seek medical advice.

In January, 1888, anæmia was already very marked, but, with the exception of some congestion of the throat and certain pale red spots on the tongue, there was nothing further objective to be made out. At times there were retching and vomiting, especially in the morning. Although still able to go about, his condition at this time was such as to occasion anxiety to his friends. A month later the number of corpuscles was found reduced to 860,000 per cubic centimetre (17 per cent.), and the corpuscles themselves showed all varieties of change both in form and size.

Under treatment he recovered considerably during the spring and summer of that year. I had an opportunity of seeing him in June, 1888. He complained much of great weakness and exhaustion on the slightest exertion. There was no emaciation, but the pallor was marked, without any trace of lemon tint, or of jaundice of conjunctivæ. The condition of his tongue troubled him much. Great tenderness on mastication, especially when hot or stimulating food or drinks of any kind were taken. The tenderness on swallowing he described as extending down the throat to the stomach. Tongue extremely raw and flabby, deeply indented by the teeth, presenting a red and fiery appearance, with here and there scattered patches of a more inflamed character, the intervening portions of mucous membrane being smooth as if devoid of papillæ. The inflammatory redness extended to the anterior pillars of the fauces. There was no uneasiness or tenderness of, nor any symptoms connected with, the stomach itself.

Number of red corpuscles at this time 3,200,000 per cubic millimetre, with 56 per cent. of hæmoglobin, the corpuscles showing no specially marked changes, either as regards their size or shape. This condition of the blood represented, therefore, a considerable improvement on that found four months previously.

He went to the country for a time, and returned early in August

somewhat improved in health. The improvement however, was more apparent than real. The condition of the blood on his return I found to be almost the same as before. From this time onward he began to lose ground, and became weaker and weaker till he had to take to bed early in December—about three months before he again came under my observation, on March 9th, 1889.

Present Condition.—He presents an extremely pale, slightly lemon-coloured appearance, without jaundice of conjunctivæ, or cedema, or other obvious signs of disease. No emaciation, although patient has lost all the appearance of stoutness which he formerly possessed. No fever; pulse 108, soft and regular; weakness, very great; unable even to sit up for any time. Muscles of arms and legs very soft and flabby.

Alimentary System.—Appetite very poor, no desire for food, and much discomfort in taking it, especially if of a hot or stimulating character. Gums very spongy, and some of the teeth loose. Condition of tongue much the same as that previously described; it is not quite so raw looking, but presents a more atrophied appearance, the mucous membrane being smooth and free from papillæ. At parts over the dorsum and along the edges there are patches of more fiery redness, some under the tip of the tongue showing small inflamed vesicles full of serum. The whole tongue is tender, and mastication is both painful and difficult. No acidity or uneasiness in stomach after food; occasional flatulence; bowels irregular, requiring the use of mild laxatives (castor-oil in capsules); looseness at times, especially at nights, alternating with constipation. While the former weakness him, the latter occasions him even more discomfort. He always feels very uneasy when the bowels are not moved daily.

Nothing abnormal to be found on physical examination of the abdomen. Liver dulness natural. Spleen enlarged; dulness extending from upper border of eighth to lower border of eleventh rib.

Blood shows most marked changes. Number of red corpuscles 1,730,000 (34 per cent.), with 30 per cent. of hæmoglobin. The red corpuscles well preserved, but showing marked changes both as regards size and shape; the former, however, being much more marked than the latter. The corpuscles vary in diameter from three to ten or fifteen μ ; most of them under examination spherical, including those of small size; some, however, oval, flask-shaped, and pointed. In addition, a certain number seen throwing off buds, varying in diameter from one-third that of the original corpuscle. In all respects except size these buds resemble the original corpuscles—their shape, colour, and appearance being the same. A certain number of the red corpuscles, surrounded by paler more colourless spheres, derived apparently from the corpuscles themselves. The material of the corpuscle seems to ooze out, and then assume the spherical form, frequently remaining attached for a time by slender processes to the main body of the corpuscle. No increase in the number of blood plates, nor of white corpuscles. No small extremely high coloured spherical corpuscles—Eichhorst's corpuscles—to be seen. No nucleated corpuscles.

Urine very high coloured, clear, very acid; quantity, thirty-five ounces, in addition to some unavoidably lost. Specific gravity 1015. Free from albumen; no bile pigments—the possible presence of which was suggested by its colour—hæmoglobin, or acid hæmatin. Urea, 1.7 per cent. It deposits on standing a flocculent cloud of mucus. On microscopic examination a number of desquamated renal cells, in small irregular cast-like groups holding granules of blood-pigment. The yellowish granules of blood-pigment are confined to those cells, none being seen free or within any of the epithelial cells of bladder present.

Vasomotor and Nervous System.—Mental faculties absolutely unaffected. Troubled a little with sleeplessness at night, also with occasional and somewhat profuse perspirations, especially in the early morning. These latter usually connected with the

¹ For the notes as to his condition at this time I am indebted to the kindness of Dr. Lauder Brunton, to whom I desire here to express my thanks.

attacks of looseness of the bowels, from which he at the same time suffers. The other systems present nothing abnormal.

Subsequent History.—The patient had been steadily losing ground since the preceding December. His temporary recovery the previous summer had taken place under the administration of phosphorus and strychnine. Arsenic had failed then to do him any good, and it had not since been tried. He was now receiving iron in the form of small doses of the tincture of the perchloride, without apparently any benefit. He was taking various meat and beef extracts in addition to such solid meat as fish, egg, chicken, etc., as he could be induced to take or the condition of his tongue would allow him to take. It was the latter that occasioned him most trouble, taking of food being, in fact, a painful effort, owing to the tenderness of the tongue.

Mild astringent lotions were ordered for the mouth, the diet was changed to a less nitrogenous one, milk and farinaceous food being substituted, and, in addition to the iron he was already receiving, arsenic in 2-minim doses of the liquor arsenicalis was ordered. Under this treatment a rapid and distinct improvement took place as regards the condition of the mouth and tongue. The change of treatment took place on March 10. The effect was already obvious on March 20. The tongue was then flabby and smooth on the surface, but much less so than before. It had lost its former angry red appearance and was now much paler, the inflamed patches were less marked, and the vesicles had entirely disappeared. From this time onward he ceased to have any further trouble with his tongue. Its condition was better than at any time during the previous year and a half. The sponginess of the gums disappeared, the teeth became firm, the tongue always continued to present a smooth flabby appearance, but the tenderness on eating was entirely lost; instead of being a painful effort to take even warm bland food and drinks, he could take all kinds of food and even condiments without the slightest discomfort.

Under the change of diet a corresponding improvement—more gradual, however, and less obvious in its character—was also noticed for a time in his general condition. The arsenic was found to disagree, and was stopped five days later (March 15th). Its administration had been pushed too rapidly, with the result that the patient suffered from frequent micturition and some symptoms of strangury. At the same time the urine threw down for the first time a heavy deposit of uric acid crystals and amorphous urates. This condition entirely disappeared and never recurred after the arsenic was stopped. As arsenic had been found in an earlier stage of his illness to be without effect, I did not again try it.

The improvement that occurred for a time may be judged of by a note made a month later (April 13th). Notwithstanding that during the intervening period he had several exacerbations of weakness of the kind presently to be described, and on two occasions had had slight bleedings from the nose, his general condition was on the whole considerably improved. His appetite was better, and he was taking much more food than before.

The pallor was still extreme, mixed with a slightly lemon hue, but there was a slight return of colour to the mucous membranes and finger nails. He felt considerably stronger, and was now able to sit up for half an hour daily. He was still troubled with occasional looseness of the bowels, especially at night, accompanied by sweating. The improvement evident in his general condition was most manifest in the urine. The latter was of more natural colour than at any time observed throughout the latter period of his illness, and presented nothing abnormal on microscopic examination.

The improvement which had thus set in, slight though it was, had not, however, been an uninterrupted one. He was subject from time to time to peculiar exacerbations of weakness, usually more or less sudden in onset and of comparatively short duration, which always left him in a more exhausted condition than before. They occurred without any recognisable exciting cause, and were marked by certain highly characteristic features well fitted to arrest attention.

The first of these attacks I had occasion to note occurred on March 22nd. For two days previously he had presented a much more lemon tint than usual, and the urine at the same time had become still more high coloured. The night of the 21st had been disturbed by perspiration, but on the morning of the 22nd he felt comparatively well, and took breakfast as usual. Shortly afterwards he was seized with a feeling of intense weakness, ushered in by drowsiness so marked in character that at the time of my visit the

patient lay in a dreamy soporific state, as if under the influence of a slight narcotic, answering questions slowly and languidly, and taking no interest in his surroundings. This was all the more marked, as his natural disposition at all times was lively and excitable. While the attack lasted the patient presented a ghastly hue, the lemon tint becoming so pronounced that the medical attendant at one time feared an attack of jaundice. The pulse was slightly increased in rapidity, and more compressible; temperature 99.4°; pupils contracted. The drowsiness continued till late in the afternoon, gradually passing off. During the night there were several free motions of the bowels, the stools being much darker in colour than usual.

The urine passed at the same time was of remarkably high colour, clear, very acid in reaction, specific gravity 1016, quantity obtained (excluding some unavoidably lost) being 36 ounces. On microscopic examination, I found a large number of renal cells containing granules of blood pigment, some of the cells being full of such granules. The appearance of these renal pigment cells in the urine at this time was all the more striking, as they had been entirely absent from the urine for more than a week previously (March 13th to 22nd).

The day after such an attack as has been described the patient always expressed himself as feeling relieved, although it always left him much weaker. Slight attacks occurred from time to time, constituting what came to be termed his "bad days." Their onset was always heralded by a more yellow or lemon hue of skin; when at their worst, his weakness was such that the worst was occasionally feared. Nevertheless, he recovered from these in the most marvellous way, so that, as already mentioned, on April 13th considerable improvement had taken place in his general condition since the time he came under my observation. On April 17th he had another sharp exacerbation of weakness, from which he never again recovered. It was marked as before by drowsiness, increase of lemon colour, profound weakness, slight rise of temperature, flushing of head with perspiration, and contraction of pupils; and was followed by several free motions rich in pigments, and by passage of very high coloured urine, extremely acid in reaction, with renal cells and casts containing granules of blood pigment. He recovered from this in part, and was able to sit up for an hour on April 23rd. Next day, however, he had a return of the weakness, and was languid and drowsy the whole day. Examination of the blood the following day showed number of red corpuscles 950,000 (19 per cent.), with 20 per cent. of hæmoglobin; poikilocytosis extremely marked; the red corpuscles presenting the most marked changes in shape and size; some of them throwing off buds, others surrounded by pale colourless discs still adhering to the corpuscles by slender processes. The pallor of the patient at this time was extreme; lemon colour very marked, the appearance presented by the conjunctivæ being that of slight jaundice. Tongue soft and flabby, slightly red at margins, smooth on the dorsum. He had not been troubled so much with his bowels during the past week. Perspirations had also been checked by 1/32-grain of sulphate of atropine at bedtime. Pulse 100, soft and regular; temperature normal, slightly raised during the last three nights—never, however, above 99.5° F. At times a feeling of great heat over the head. Systolic murmurs over the heart. Splenic dulness still increased.

From this time onward he rapidly lost ground, his weakness steadily increasing, uninfluenced apparently by diet—mainly milk—or medicine— β naphthol—on which he was now put. The urine remained of persistently high colour, notwithstanding that the quantity was undiminished—varying from 40 to 52 ounces—and its specific gravity was low (1014). Slight attacks of drowsiness became more common, with mild delirium at nights. On May 3 slight epistaxis occurred, the blood, of a pale yellow colour, merely tinged with hæmoglobin. The heart's action became weaker, murmurs louder, weakness greater, then finally death occurred on May 22nd, preceded by wandering delirium. Consciousness was retained till within a few hours of death.

MORBID ANATOMY.

Extreme pallor of body, subcutaneous fat of a bright lemon colour present in considerable quantity. All the organs of the body, with single exception of spleen, pale and bloodless. Heart showed slight fatty degeneration of its muscle. Liver, on section, presented a rusty and somewhat mottled appearance. Centre of lobules pale, fatty; periphery of a more brownish tint, due to the presence of much pigment. On micro-chemical examination the tissue found exceedingly rich in blood pigment.

A piece of it placed in sulphide of ammonium became coal black in a few seconds—the characteristic reaction of free iron. On microscopic examination the pigment seen in the form of granules, for the most part of fairly uniform size and appearance, lying within the lower cells and most abundant in the outer two-thirds of the lobules. Similar pigment granules, although much fewer in number, also seen within the capillaries, lying enclosed in leucocytes. These changes best seen after developing the Prussian blue reaction in the iron of the pigment by placing the sections first in solution of ferrocyanide of potassium and afterwards for a short time in dilute hydrochloric acid. *Spleen* weighed 11½ ounces, enlarged and soft, presenting a purplish appearance. Pulp soft and diffident. Its appearance that of a congested spleen. On microscopic examination of fresh pulp, red corpuscles very sparse, in no greater number than in other fresh organs. Rich purple colour, due to the presence of free hæmoglobin. Large irregular rhomboidal crystals, probably of hæmoglobin, seen here and there. A piece of the fresh tissue placed in sulphide of ammonium became rapidly coal black. Blood pigment present in very large quantity in the form of minute spherical granules lying within the cells of pulp. After hardening of tissue this reaction altogether less marked. *Kidneys* pale and bloodless. Large quantity of blood pigment in the form of fine granules lying within the cells of the convoluted tubules, in certain also of the ascending loops of Henle.¹ *Stomach*: Walls of stomach and intestine very thin and almost transparent. No trace of malignant disease. Lymphatic glands lying along the main lymphatic trunks on the smaller curvature of the stomach enlarged, soft, pinkish, and translucent in appearance, the largest of them five centimetres in diameter. Stomach empty, its mucous membrane covered with a thin layer of mucus, slightly bile-stained near the pylorus. Mucous membrane very thin, but presenting to naked eye no obvious morbid appearance. After hardening—portions of it in Müller's fluid and then in spirit; other portions in spirit—most extensive changes are revealed, affecting both the submucous and mucous coats. The cardiac end of the stomach is the seat of chronic, subacute, and acute inflammatory processes, the changes varying in degree and character at different parts. At parts the gastric glands have entirely disappeared, and are replaced by fibrous tissue; at others the glands are still seen surrounded and pressed on by connective tissue, the glandular cells themselves in a state of proliferation. At parts the changes even more acute, large groups of actively proliferating connective tissue and glandular cells replacing the original gland structure. Around these inflammatory foci, capillaries greatly distended, and extending in from the submucosa. Changes of this kind present throughout the greater part of the mucous membrane; only here and there are portions seen where the glandular structure remains intact. The changes in the submucous coat as striking as those in the mucous membrane itself. The submucous coat greatly thickened, so much so that it is difficult at parts to trace the transition between it and the mucous membrane; this thickening due to increase of fibrous tissue, resulting from chronic inflammatory processes. The change involves in a special degree the vessels, the walls of which are greatly thickened; at parts, however, the process is more subacute in its character, the walls of the vessels being seen infiltrated with groups of proliferating connective tissue cells. In the pyloric portion of the mucous membrane the chief change is the increase in the interstitial connective tissue between the glands; the more subacute and acute inflammatory changes are absent. *Small Intestine*: Walls very thin. Mucous membrane of duodenum covered with a thin coating of slightly bile-stained mucus. No swelling or other obvious change. On microscopic examination nothing abnormal found.

PATHOLOGY.

During the last two months and a half of his illness the urine of this patient was made the subject of a daily series of observations relating to the nature (1) of the changes in the blood, (2) of the changes in the gastro-intestinal tract responsible for the extensive blood destruction, characteristic² of the disease.

The former included observations on the excretion of urinary pigments, of blood pigment, and of iron. The results obtained seemed to me of such interest and importance, both in their bear-

ing on the pathology and diagnosis of the disease, that I have recorded them at length in a series of papers elsewhere.³

1. They showed, first, a largely increased excretion of urinary pigments, evidenced by an extremely high colour of the urine without any diminution in quantity, and with a low specific gravity. During the last three weeks of his illness, for example, when there was little intestinal disturbance and the whole of the urine could be obtained, the quantity varied from 40 to 52 ounces, with an average specific gravity of 1014. The reaction was at all times extremely acid. At no time were bile pigments found in the urine. Uro-erythrin appeared from time to time during the slight febrile attacks from which he suffered. The chief interest attached to the presence in large quantity of a urinary pigment, having all the characters (chemical and spectroscopic) of that termed by Dr. MacMunn "pathological urobilin." These characters I have described and represented in the paper referred to. This pigment was not in itself the cause of the high colour of the urine. After its removal from the urine by means of chloroform the urine retained much of its original colour. Furthermore, it was present in very varying quantity at different times, even in urine presenting the same high colour. Thus, while its characteristic absorption band was sometimes lost on dilution of the urine twice, at other times it was still recognisable after the urine had been diluted as much as six or seven times. My conclusions as to the character of this pigment were based on numerous observations, both chemical and spectroscopic, made from day to day. I have since repeated these observations with the pigment then separated, and still present in my hands in considerably quantity. I have satisfied myself once more that it differs in important respects, both chemically and spectroscopically, from any pigment present in the urine in health, or obtainable by the method I employed from high coloured urine of fever. So far as my observations go, the distinction drawn by Dr. MacMunn between the pigment present in the urine in health and in high coloured urines generally, to which he gives the name of "normal urobilin"—and that obtainable from the urine under certain special conditions, for example, absorption of large extravasations of blood, to which he gives the name "pathological urobilin," is a valid one.

While a high significance attaches to the presence of high coloured urine of low specific gravity and in undiminished quantity, an altogether special significance attaches, in my opinion, to the presence of pathological urobilin in such cases. It is most usually found in the urine during absorption of large extravasations of blood, under conditions therefore I would define as primarily those in which an undoubted extensive destruction of blood is going on unattended by hæmoglobinuria.

Dr. Mott has recently recorded⁴ a case of this disease in which the pigment found by himself and Dr. Halliburton was urobilin; and, in reference to the foregoing observation, Dr. Mott expresses the opinion that it is of comparatively little importance which variety of pigment is present, the chief interest attaching to the presence of "urobilin" in such large quantity being that it serves to explain the presence of iron in the liver in such cases. I have shown in my former investigation that it is the peculiar character of the preceding blood destruction that determines the presence of iron in the liver in such cases. While I found it impossible to reproduce a condition of the liver similar to that found in pernicious anemia, by injection of such reagents as distilled water, glycerine, or even pyrogallie acid, I succeeded, on the other hand, with toluylene-diamine. With regard to this drug I found that it had not only a peculiar destructive action on the blood, but had also "a specific, not necessarily poisonous, action on the liver cells." One result of its action on the blood was that the hæmoglobin remained combined with the albuminous constituents of the corpuscles or plasma. As the result of its action on the liver cells part of the hæmoglobin reduced *in situ* to the form of an albuminate of iron, which remained within the liver cell in the form of pigment granules. That under these circumstances the by-products of pigment nature split off from the original hæmoglobin molecule should differ from those split off in health need occasion but little surprise. In health such by-products are the bile pigments, and the pigments and chromogens of the urine including normal urobilin. Under other conditions, for example, changes in large extravasations of blood, special by-products may appear, differing, it may be slightly, but still recognisably, from the pigments usually formed. As such a by-product I regard

¹ For full description of these changes in the kidney in cases of pernicious anemia as well as of their significance see my paper "On the Excretion of Blood Pigment," *Practitioner*, November, 1889.

² "An investigation into the Pathology of Pernicious Anæmia," *Lancet*, ii, 1888.

³ "Observations on the Urine in Pernicious Anæmia," *Practitioner*, September November, December, 1889.

⁴ *Lancet*, vol. i, 1890.

pathological urobilin. It is formed, not only by processes of reduction or oxidation from the bile pigments within the intestinal canal, but also I find evidence to show from hæmoglobin through the agency of cells. Between it and normal urobilin important differences exist. Its appearance in the urine in pernicious anaemia points therefore—far more than any excess of ordinary pigments—to a preceding extensive destruction of blood of a special nature. It does not explain the presence of iron in the liver, any more than the excess of iron in the liver explains its occurrence. Both alike are, however, explained by the same special cause—a destruction of blood proceeding along lines, and due to causes, other than those found in health. I concluded in my former investigation that the destruction was caused by the action of certain poison or poisons of specific nature. It was the object of my investigations to discover in the present case whether any such poisons were present.

2. My observations on the urine in the above case showed, secondly, occasional presence of blood pigment in the form of granules enclosed within cells of renal origin. For a time these pigment cells were absent, and their absence corresponded with the period during which ground was being regained. They reappeared, however, in increased numbers on each occasion of an exacerbation of his weakness. The cells and pigment granules present in the urine during life were identical with those I found in the tubules of the kidneys after death; and their origin from the latter cannot, therefore, be doubted. The significance to be attached to their presence I have elsewhere fully discussed.

3. They showed, lastly, an increased excretion of iron. In three cases of chlorosis I found the average excretion of iron in the urine *per diem* 1.76 milligrammes, and in health 5.65 milligrammes. The average daily excretion of iron in the urine in health I estimate to be from 3 to 5 milligrammes. Three weeks before the patient's death in the above case, at a time when the anaemia was most intense, I found the excretion to be so high as 32.26 milligrammes; a week before death it was 6.52; and two days before death it was still 1 milligramme.

So far as I am aware, no previous observations have been made on the excretion of iron or the presence of renal cells containing blood pigment in this disease. Taken together with the large increase in the urinary pigments, and especially with the presence of pathological urobilin in the urine, they pointed clearly to a preceding excessive destruction of blood, the amount of destruction, as evidenced by the varying colour of the urine and excretion of pigment, varying from time to time.

These clinical observations in the present case bear out fully the conclusions it was one of the main objects of my former investigation to establish as to the essentially hæmolytic nature of this disease. The changes found after death in liver, spleen, and kidneys point equally to the same conclusion. The situation and character of the pigment within the liver were such as I have formerly described. The pigment was more abundant in the present case than in any I have yet met with; and I have now examined the liver in thirteen cases of this disease. The enlargement of the spleen recognised during life and confirmed *post mortem* is of some interest in connection with the important part played, as my observations show, by that organ as one of the chief seats of blood destruction both in health and disease. The congested purplish appearance presented by the pulp was the same as that found by me in another case I have described. It was also due apparently to the same cause—not to the presence of red corpuscles, few of which were to be found, but to the presence of free hæmoglobin. The presence, also, of well-formed crystals, presumably of hæmoglobin, is also of special interest in connection with the interesting observations of Dr. Copeman⁵ as to the readiness with which crystals of hæmoglobin are obtained from the blood in this disease.

(To be concluded.)

⁵ St. Thomas's Hospital Reports, 1887.

MALE NURSES.—The annual meeting of the Hamilton Association for Training and Supporting Male Nurses was held on June 28th, at Park Street, Grosvenor Square. Amongst others who spoke of the capability of the fifty nurses attached to the Society, either ex-army staff assistants or hospital trained nurses, were General C. E. Gordon, C.B., Sir William MacKenzie, and Dr. Prosser James. The balance sheet proved highly satisfactory, and the Society owes much to the liberal founder, Miss Hamilton.

THREE LECTURES ON SOME POINTS RELATING TO INJURIES TO THE HEAD.

Delivered at the Royal College of Surgeons of England.

By WILLIAM HENRY BATTLE, F.R.C.S.,

Hunterian Professor of Surgery and Pathology, Royal College of Surgeons
Assistant-Surgeon to the Royal Free Hospital; and to the East London
Hospital for Children; formerly Resident Assistant-Surgeon at and
Surgical Registrar to St. Thomas's Hospital.

LECTURE I.

MR. PRESIDENT AND GENTLEMEN,—I have taken as a title for the lectures which I am about to give, "Some Points Relating to Injuries to the Head," and purpose asking you to consider with me, more especially, the effect of injuries when fracture of the cranial base has been produced, with some of the complications which are only too liable to accompany these fractures, or follow at a longer or shorter interval after the force producing them is expended.

For several years, in various official capacities, I had the privilege of seeing the surgical practice of St. Thomas's Hospital, and of closely observing, amongst other cases, many of injury to the head; and through the kindness of the members of the surgical staff of that hospital, I am enabled to bring before you a series of the cases which were under care in that institution during a part of that time, and can thus present a comparatively large number of examples for consideration. To these I have added the results of experience obtained at the Royal Free Hospital or elsewhere.

I have made a summary of most of them, and it will be to cases in this group that my remarks will refer, unless mention is made to the contrary.

TABLE I.—Non-fatal Cases of Fractured Base.

	Males.	Females.	Ages in Years.									Total.
			Under 5	Under 10	Under 20	Under 30	Under 40	Under 50	Under 60	Under 70		
Anterior fossa	16	5	—	3	3	6	3	4	2	—	21	
Middle "	59	6	3	4	8	16	13	6	5	1	56	
Posterior "	10	1	—	2	2	—	4	2	1	—	11	
Middle and anterior fossæ ...	15	5	1	4	2	4	5	3	—	1	20	
" posterior "	4	1	—	—	—	2	1	2	—	—	6	
Anterior, middle, and posterior fossæ	1	—	—	—	—	—	—	1	—	—	1	
Total	96	18	4	13	15	28	26	18	8	2	114	

Fractures of the base of the skull may be produced by direct or by indirect violence. Examples of the former are afforded by (1) thrust wounds usually of the orbit or nose; (2) a blow on the chin, driving the condyle of the lower jaw through the glenoid fossa, as shown by a preparation in the museum of St. George's Hospital; (3) a blow on the nose, fracturing the bones which enter into its formation, and the cribriform plate of the ethmoid. The fracturing force may also be conveyed through the condyles of the occipital bone, either from a fall on the buttocks or feet, or as the result of the impact of the body weight on the condyles in a fall on the head. Two opposing forces will also produce a fracture, sometimes limited to the central bones of the base. They may also be caused by *contrecoup*, but are most commonly the result of extension of a fissure from the vault into a fossa on the side to which the violence is applied.

They may also be compound, either as the result of the force acting at the point struck, or from their extension into cavities which communicate with the external air; for this reason the majority of fractures of the base of the skull are compound when they are in the middle fossa, through implication of the middle ear, also a large number of those in the anterior fossa from extension of the fracture through the cribriform plate into the nose.

As a rule, when they extend into the base from a fissure of