shown, the animal be in full digestion at the time. The same applies to the liver of the dog, the cat and of man. Here, in passing, I may note that throughout my observations I have always attached great importance to the examination of the fresh tissue. The best micro-chemical reagent for revealing the presence of free iron, or iron containing pigment in fresh tissues, is, according to my observations, sulphide of ammonium. A piece of fresh liver tissue placed in sulphide of ammonium takes on a colour varying from light greenish-yellow to coal black, according to the amount of free iron it may contain. If a piece of this tissue be teased out in sulphide of ammonium and examined at once microscopically, the pigment will be seen as coal black spherical granules, or as a diffuse greenish stain, according as it is in the form of granules or diffused in more soluble form amidst the protoplasm of the cell. For the fresh tissue sulphide of ammonium appears to me to be a far more delicate and reliable reagent than, for example, ferrocyanide of potassium and hydrochloric acid (Prussian-blue test). The reaction, if any, is developed at once, and without any injury to the cell protoplasm, whereas with the Prussian-blue test the degree of colour always depends to some extent on the length of time the tissue is allowed to be in contact with the reagent. Hence if, as sometimes happened, the two reagents gave results apparently not in agreement with each other—if, for example, the tissue after hardening gave little or no reaction of iron, whereas in the fresh condition it gave a considerable reaction—it was on the former and not on the latter that I relied most.

This difference in the behaviour of tissues before and after hardening is by no means uncommon. It is most common when the pigment is diffused throughout the cell, and it is due, in my opinion, to the circumstance that in the process of hardening the soluble pigment has diffused out. For this reason the best hardening agent is alcohol, Müller's fluid and other similar preserving agents are to be avoided. Tested in this way, the liver of young healthy mammals gives evidence of containing very little blood pigment. According to Drs. Brunton and Delépine² this amount is considerably increased during digestion. If then, as I think is the case, the presence of pigment within the liver cells in health is in mammals by no means a striking feature, the case is very different in certain forms of disease.

In Disease.—I show you sections from the liver in cases of pernicious anæmia. You note how very abundant is the pigment; how striking is the iron reaction it gives; and how characteristic is its distribution within the lobule. It lies within the liver cells, but not uniformly distributed amongst all the cells of the lobule. It is most abundant in the cells at the periphery of the lobule, while little or none is to be found in the cells of the inner third of the lobule. These latter are for the most part fatty, and are quite free from pigment. The pigment is in the form of granules, fairly uniform in size, although larger than those we saw in the liver cells in pigeons. It appears to occupy the axis of the rows of liver cells; but on careful examination with high powers it is in some cases seen to occupy a very definite relation to the bile capillaries. It lies close to the edge of the cell, as if in process of being excreted into the bile capillary. In no disease, as I have elsewhere shown,³ does the amount of pigment found in the liver ever exceed—it rarely ever approaches—that present in pernicious anæmia. A large increase of pigment within the liver cells can, I find, be experimentally brought about by injection of certain substances into the blood. Such an increase I show you in the case of three animals, rabbit (Experiment 42), dog (Experiment 64), and of a pigeon (Experiment 72), induced by the action of toluylendiamine. You will note how exceedingly abundant the pigment is, and how the appearance of the liver contrasts in this respect with that of the liver of the normal animal.

The conclusions, then, to be drawn from this portion of our study are as follows:—1. That another situation in which pigment may be found within the liver apart from extravasation or congestion is the liver cell. 2. The frequency with which it is met here in health, and its amount, differ in birds and mammals respectively. In birds it is usually present, sometimes in large amount. In mammals, on the other hand, while rarely ever absent, it is, according to my observation, not present in any great quantity. 3. The pigment presents certain peculiarities, more especially in respect of the small

uniform size of its granules, which distinguish it broadly from that met with in health in the capillaries of the liver. 4. The conditions under which it arises also differ from those already seen in the case of the latter in the capillaries. (a) The small uniform character of the granules is as striking in birds, where the red corpuscles are large and oval, as in mammals, where they are small and round. (b) It is not increased in old animals. It is, on the contrary, more likely to be found in young animals than in old. (c) Abstinence from food and exercise tends rather to diminish its amount. Thus my observations prove that it is not increased by withholding food from animals, or during inanition in man. Drs. Brunton and Delépine's observations show that it is increased during digestion in animals (rabbits). In all these respects therefore the conditions favouring the deposit of pigment in the capillaries and liver cells respectively are essentially different. 5. The only respect indeed in which they are the same is that in both cases I find an increase may take place as the result of disease, or may be induced experimentally by agents acting injuriously on the blood. Even here, however, there is a difference, for the increase which may occur under such circumstances within the liver cells is far greater than anything ever observed in the capillaries. This is true at least of disease.

(To be continued.)

THE BLOODVESSELS OF THE THYROID GLAND IN GOITRE.

By R. M. HORNE, M.B., C.M. EDIN.

THIS communication is a short account of some work carried on during the present year in the Pathological Institute of the University of Strassburg, under the direction of Professor v. Recklinghausen, to whom I desire to express here my grateful thanks for his uniformly kind assistance and encouragement. Histologists have long pointed out the great richness of the blood-supply to the thyroid gland, and have demonstrated the existence of a comparatively thick media in the walls of its arteries. The morbid histology of the blood vascular system of this gland has been but little worked at,¹ Gutknecht alone of all the inquirers into the pathology of this organ having devoted much attention to it. Working on goitrous thyroids extirpated by Kocher of Berne, he claims to have demonstrated widespread hyaline changes in the walls of the bloodvessels, and also the presence of masses of colloid material in the lumina, both of those with degenerated walls and of those with still normal walls. These colloid masses he derives from the red blood-corpuscles of the circulating blood, in which a complicated series of changes leading to the formation of colloid is described. One is led to infer that these changes are induced by some influence exerted by the diseased thyroid. In the course of this research twenty-eight thyroids have already been examined. These, with one exception, were obtained post mortem. They include seven foetal thyroids removed from foetuses varying in development from between the third and fourth to between the seventh and eighth months; two from new-born children who had died at birth, one being premature about the eighth month, the other a full-time child; one from a child who had died a few days after birth, suffering from bronchitis and icterus neonatorum. The remaining eighteen were derived from individuals varying in age from six months to sixty-eight years, whose deaths had been induced by a variety of causes, comprising, for instance, such widely separated diseases as diphtheria, labyrinthitis purulenta and meningitis, tuberculous affections, asphyxia from drowning &c.

The glands were, with one exception, fixed in Müller's fluid and subsequently hardened in alcohol. The exceptional gland was treated with alcohol only. From these hardened specimens pieces were taken of a size convenient for embedding in celloidin or for cutting with the freezing microtome, which was, however, only occasionally used. Each piece, so far as practicable, was cut so as to contain more normal, preformed thyroid tissue along with

² Proceedings of the Royal Society, 1891.

³ THE LANCET, Sept. 22nd and 29th, 1888.

¹ Die Histologie der Struma: Virchow's Archiv, Band xcix., 1885.

one or more of the smaller adenomatous nodules. In cases where the nodules were too large to admit of this being done the piece was taken from the remains of the original tissue. In specimens not possessing nodules it is unimportant from what part the piece be taken. The pieces were generally obtained from the periphery of the gland with a portion of the capsule attached, and were cut at varying angles to the long axis of the lobe. The method adopted in the examination of these pieces, with the exception of the few earlier ones, was as follows. The piece was stained *en masse* in alum carmine, and, the excess of colouring matter having been carefully washed away in water, was subsequently hardened in absolute alcohol and then impregnated with celloidin in the usual manner. By this means the confusion so apt to arise between colloid material and celloidin is entirely avoided, as the latter, in contrast to the former, remains quite uncoloured and transparent. Each piece thus treated was cut in series. Serial sections were found to be necessary for the purpose of distinguishing definitely arteries from veins, veins from lymphatics and veins and lymphatics from distended follicles. The recognition of these various structures, often difficult and sometimes impossible from the examination of single sections, is readily accomplished if the structure under examination can be traced through a sufficiently large series of sections. The most important changes found in the course of this investigation are those developed in the arteries. These may best be described by a more detailed reference to one particular case in which they were very pronounced. The gland was removed post mortem from a young man aged nineteen. The section revealed the cause of death to be labyrinthitis purulenta with meningitis. Both lobes of the thyroid were small, and on longitudinal section were seen to be studded with numerous adenomatous nodules of various sizes, and distinctly encapsuled. Some were as large as a cherry, some were scarcely visible to the naked eye, and microscopic examination revealed others not seen macroscopically. Four pieces were taken for examination; three of these were cut in series after being embedded in celloidin; the other was also embedded in celloidin, but not cut in series. One piece only was stained *en masse* before being embedded and cut. Under the microscope the lobules of the gland are well defined and separated from each other by septa of normal fibrous tissue. Many of the gland follicles are composed of a solid group of cells without colloid; some contain a small quantity of colloid with a lining of low cubical cells, others are slightly distended from accumulation of colloid, which has led to a slight flattening of the lining cells. In addition to this—the original and more normal thyroid tissue—are seen circumscribed nodular areas of various sizes, which strike one at once from the depth to which they have taken up the alum carmine. They stand out in strong contrast to the less deeply coloured thyroid lobules. They have pushed aside the thyroid follicles in their growth, from which the older ones, at any rate, are separated by a capsule of fibrous tissue often containing many elastic fibres. The tissue composing the nodules is thyroid in its nature and represents the tissue of that gland in its various stages of development, from the branched, elongated, solid groups of cells through the stage in which the cells are arranged in round solid groups to the appearance of ordinary follicles containing colloid. In the older nodules the follicles become much distended and rupture of the walls between adjacent follicle is frequent, the cells, however, still retaining their cubical shape. The cells which build up these various structures in the nodule are much more deeply coloured than the cells of the normal thyroid follicles, and it is to these of course that the deep colour of the nodule as a whole is due. There are many other small, deeply stained areas to be seen which with low powers might in many cases be regarded as nodules in their youngest stage, but which with higher powers are found to be altered arteries. The alteration which these arteries have undergone consists of a well-marked proliferation of their endothelium in localised areas, so as to form solid buds of cells projecting into the lumina of the vessels in which it occurs. Such a condition has not previously been described in the arteries of the thyroid, or, so far as I am aware, in the arteries of any other organ. A typical example of this bud-like proliferation is seen in Fig. 1 (*a*), in which an artery is shown in longitudinal section. The three coats of the artery are easily traced throughout its whole course. The media is particularly well marked, consisting of several layers of muscle cells in transverse section, and here and there on the intima flat endothelial

cells are to be seen. At one point of the intima (*a*) there is a very evident projection into the lumen of the vessel. This is composed of a group of small, round or slightly polygonal, closely-packed cells, which stain well with alum carmine. The muscular coat passes entirely outside of this projection and is almost unaffected by it; perhaps slightly deflected to one side. The group of cells lies without doubt quite internal to the media in the intima itself, and can only have been developed by proliferation of the endothelial cells at that point. The innermost layer of cells—i.e., those towards the lumen—still retain their elongated, flattened endothelial character, but are larger and more distinct than normal, and form a continuous

FIG. 1.

covering over the projecting bud. The effect of this projection is evidently to considerably narrow the lumen of the vessel at the point where it is situated. In this section the lumen is reduced to a very narrow channel. The circulation has not been brought to a standstill thereby, as the vessel behind and beyond the bud is still filled with normal red blood-corpuscles. In a branch given off by this artery several such buds are found just at its commencement, although, owing to the obliquity of the section, their relation to the various coats of the vessel is not so satisfactorily shown. One bud (*b*) is seen lying free in the lumen, circular in shape and consisting of

FIG. 2.

small round or polygonal cells surrounded by a layer of more endothelial-like cells.

In Fig. 2 another artery is shown in longitudinal section in which the bud-like proliferation of the endothelium is well displayed. This artery is remarkable for the number of buds present in the short length of vessel depicted. The buds themselves in their structure and position resemble that of Fig. 1. At several points also a commencing proliferation of endothelium is visible, which has not yet developed into a well-defined projection. The diminution in the size of the lumen is very evident. Transverse and oblique sections of

arteries with similar bud-like endothelial proliferations are naturally to be met with much more frequently than such beautiful longitudinal sections already figured, but are none the less characteristic. Two transverse sections are shown in Figs. 3 and 4. In the first of these the resolution of the wall into its three components is easily made, and there are seen to project from the intima two small rounded groups of cells resembling the buds already seen. In Fig. 4 the media is not so readily distinguished, but by following the vessel through a few sections all doubt of its arterial nature is removed. There are present in this section two projecting masses of cells: one is large and consists of cells similar in size and shape to those of the buds seen in longitudinal section, the innermost also partaking more of the nature of true endothelium, although in part of the margin they are more rounded than elongated. The other bud is much smaller, contains fewer cells, and is probably a younger stage of development. The endothelium lining the rest of

FIG. 3.

the lumen is well developed and almost resembles a low cubical epithelium. The lumen, although blocked to some extent by these buds, is in neither case completely closed, but still admits the passage of blood, normal red blood-corpuscles being present in both sections. Transverse sections of arteries displaying a more complete occlusion of the lumen than is here represented are readily found. What part is played by collapse of the walls of the vessel in this diminution in the size of the lumen it is difficult to determine. It would seem, however, to be small, for in most cases if the buds be supposed to have been removed there would remain a lumen large in proportion to the size of the vessel in which they are situated.

Perfect occlusion of arteries has not been satisfactorily demonstrated in this gland, but in one other it is frequently met with. In this specimen the lumen is filled with a mass of cells which cannot be resolved into bud-like pro-

FIG. 4.

jections. The cells resemble in size and shape those of the gland follicles, but stain somewhat more deeply. As it is often impossible to determine the presence of muscle cells in the wall of these occluded vessels, it becomes a matter of great difficulty to differentiate them from normal thyroid follicles. The difference in the depth of colour leads one to suspect their nature, but this can only be finally determined by tracing through a series of sections their connexion with the still functional arterial system.

While the proliferation of endothelium is generally directed inwards towards the lumen in the form of rounded buds, it is not always so. The groups of cells may cause little or no projection of the intima, but may develop outwards at the expense of the muscular coat, which is then only with difficulty, and sometimes not at all, to be followed at this point. Or again, as seen in some of the transverse or oblique sections of the smaller intra-lobular arteries, the proliferation is more general

and forms several irregular layers of deeply coloured cells surrounding the whole circumference of the lumen. The size of the buds varies much within certain limits. A size often met with is seen in Fig. 4. Larger buds than these are occasionally found, and buds of all sizes between these and a slight elevation consisting of two or three cells occur. It is probable that the enlargement of single endothelial cells indicates the commencement of the process. In connexion with their significance and ultimate fate it is of importance to learn if the buds become detached from the wall of the artery from which they have sprung, to be carried along in the blood stream. Attention has therefore been directed to discovering any indication of such a narrowing of the base as would be expected to occur as the natural preliminary to a detachment of the bud in its entirety. No evidence of this has been observed. On the contrary, the buds, rounded or conical, possess a base as great, or almost as great, as any other diameter parallel to the base. Attempts have also been made by tracing in either direction transverse sections of buds (such, for instance, as *b* in Fig. 1) lying free, in transverse sections of arteries, to determine if they were free throughout. The presumption of course is that these are merely sections of the projecting ends of buds which have their base of attachment at a lower level than the plane of section. My observations confirm this view. It is therefore concluded that detachment of the buds in their entirety does not occur. It is possible, however, that individual cells are thrown off into the blood stream. Cells larger in size and more regular in shape than normal white blood-corpuscles have occasionally been seen lying free in the lumen of an artery, and may possibly have come from one or more of these buds. This is a point demanding further investigation.

One can scarcely avoid being struck by the resemblance between many of these buds and young gland follicles. The interesting question, then, naturally presents itself: Does colloid ever occur in the interior of a bud? Sections of arteries have been found in this thyroid having buds which contain in their interior colloid not to be distinguished from the colloid in the true gland follicles. From each of three other thyroids also a section of an artery having a bud with colloid in its interior has been obtained. The occurrence of such buds though rare is positive. It would seem therefore that they possess, in common with true thyroid follicles, the power of secreting or of producing colloid material.

It remains for me to decide the frequency of the occurrence of these proliferative changes—(1) in the arteries of this particular gland; (2) in the other glands examined; and (3) finally to determine whether this condition is confined to the arterial system. It has already been stated that four pieces were taken from this gland for microscopical examination. In all of the sections made from these, numbering over two hundred, arteries presenting the condition described were to be found with the utmost facility. It is not an over-statement to say that the majority of the arteries in many of the sections were so affected. The condition is evidently a widespread one. But while this is so, it has also to be remarked that the larger arteries are almost or altogether free from any such proliferative changes. It is in the smaller arteries, whether extra-lobular in the inter-lobular septa, or intra-lobular in the inter-follicular septa, that the condition is most frequently seen. It also occurs in some of the arteries in several of the nodules. The buds may develop at any part in the course of a vessel in which they are liable to occur, but are as a rule most frequent at the points where branches are given off, or in the branches themselves at their origin. In all of the eighteen more fully developed glands the same proliferative changes in the endothelial lining of the arteries have been observed to a greater or less extent, arteries so altered being generally found without much difficulty. Of these eighteen glands, eleven presented one or more adenomatous nodules in one or other lobe. The remaining seven, although not containing nodules, were more or less enlarged, the enlargement in all probability being either due to an accumulation of colloid in the follicles—shown in five of the seven and especially well-marked in one case of extreme enlargement—or to an increase in the number of follicles themselves. Of these, one was removed from a girl two years and a half old, who had died of catarrhal pneumonia. The enlargement was only slight. Sections of arteries having bud-like proliferation of their endothelium have been distinctly demonstrated, but occur rarely. The other belonged to a six months' old child. The gland was enlarged in both lobes. On microscopic examination the alteration in the arteries is frequent and pronounced.

No colloid is present in the follicles of either of these two glands. One thyroid was obtained from a woman aged fifty-eight, in whom a slight exophthalmos was present. The cause of death was pneumonia. Both lobes of the gland were enlarged, the right much more than the left. The large size of the right lobe was seen on section to be due to several large colloid degenerated nodules—one of them cystic—which had reduced the original tissue to a minimum. In the left lobe several smaller nodules were also observed. In most of the sections made from the more normal part of the right lobe, as well as in those made from the left lobe, many arteries with bud-like proliferation of their endothelium were present. In the three thyroids obtained from new-born children the same changes were found, in varying degree. In that of the premature child, and in that of the icterus neonatorum case, it was only after the examination of numerous sections that distinct evidence of endothelial proliferation in the arteries was gained; in the other case, however, arteries so altered were present in the greater number of sections made. This gland was slightly enlarged. It was difficult to decide whether enlargement was present in the other two. Careful section of all three did not display any nodules.

Of the seven foetal glands examined, arteries having buds have been satisfactorily demonstrated in four, doubtfully in one, and not at all in the remaining two. In none of the four glands in which such arteries are found does the condition occur with the frequency associated with most of the other glands examined. One of these, removed from an anencephalic foetus, was decidedly enlarged in both lobes, no nodules were observed macroscopically or microscopically, and no colloid in the follicles. A large number of sections were made from this specimen, but in only a few were altered arteries found. As to the distribution of this proliferative change, great pains have been taken to determine whether it is confined to the arterial system. Without going into details it will be sufficient to state that no evidence of its occurrence in the endothelial lining of the veins has been obtained. My observations on the capillaries do not as yet admit of any definite conclusions.

It is not my intention to enter into a discussion of the bearing which these observations may be supposed to have on our views of the physiology and pathology of the thyroid. A more extended investigation of the various points here cursorily dealt with is necessary before any general conclusions can be based upon them. I am at present carrying on this research in the laboratory of the Royal College of Physicians, Edinburgh.

Slamanan, Scotland.

A REPORT OF TWENTY-TWO CASES OF INTUBATION OF THE LARYNX.

By JAMES B. BALL, M.D., M.R.C.P. LOND.,
ASSISTANT PHYSICIAN, AND PHYSICIAN TO THE THROAT DEPARTMENT,
WEST LONDON HOSPITAL.

OF the twenty-two cases of intubation of the larynx here reported sixteen occurred under the care of some of my colleagues at the West London Hospital, and the remaining six were cases in private practice to which I was called by the medical men who were in attendance. The majority of the hospital cases I was enabled to follow throughout, owing to the courtesy of my colleagues, to whom I am also indebted for permission to publish the cases. All the patients were children, and they ranged in age from thirteen months to eight years. Of the twenty-two cases ten recovered. Intubation was performed in every case for symptoms of acute laryngeal stenosis which threatened the life of the child, and as the alternative of tracheotomy. In two cases the laryngeal trouble arose from traumatic causes—in one from swallowing very hot tea, in the other from swallowing carbolic acid. The rest were cases of laryngitis, simple or membranous (diphtheritic); it was not always possible to determine which. In five cases the disease was secondary to measles. Four of these died. In seven cases tracheotomy was performed after intubation had been tried, principally because the tube seemed to be clogged with membrane or secretions. All of these died.

In the cases which recovered the tube was left in the larynx for periods varying from three to thirteen days, the average period for the ten cases being eight days. The thread was

allowed to remain *in situ* in four cases, and in one of these it is noted that the child dragged the tube out by its means. In the rest of the cases the thread was removed immediately after the introduction of the tube. In all my own cases I removed the thread at once, and I have no doubt it is the best plan. In two cases (Cases 13 and 15) an ulcer was found post mortem on the anterior wall of the trachea, at a point corresponding to the position of the lower end of the tube. It is only right to mention that the tubes used in these cases were not constructed in accordance with O'Dwyer's later patterns. Case 16 is an instance of "death on the table," a rare occurrence with intubation. I have no intention here to make any remarks on the value of intubation or on the relative merits of intubation and tracheotomy. I will merely say that in two of my own cases (Cases 17 and 21), both of which recurred, tracheotomy had been previously recommended as the only chance of saving the child's life, and had been declined by the parents owing to the slender hope which was offered even if the operation were performed. In both of these cases they gladly consented to the mild and bloodless operation of intubation, although I only held out an equally slender hope of recovery. In this way I am sure that some lives may be saved by intubation which would otherwise not be saved, and, moreover, the operation is not so likely to be deferred until the child is *in extremis*, as tracheotomy too often is.

The following is a brief epitome of the notes taken. The sixteen cases first in order are hospital ones.

CASE 1.—Boy aged two years. Admitted to hospital on July 26th, 1889, under the care of Mr. Weiss, for a scalded throat, caused by drinking almost boiling tea. Marked signs of laryngeal obstruction. Intubation was performed on the evening of the 26th; immediate relief. Fed by enemata only. Tube removed on the 29th. No return of dyspnoea. Recovered.

CASE 2.—Boy aged four years. Admitted to hospital on July 27th, 1889, under the care of Dr. Drewitt, with a history of having swallowed about a teaspoonful of carbolic acid. Some hours after admission marked signs of laryngeal obstruction supervened. Intubation was performed. The laryngeal symptoms were relieved, but the breathing became very rapid and the child more and more cyanosed. Died on the following day. At the post-mortem examination there were found ulceration of the pharynx, slight gastritis and broncho-pneumonia.

CASE 3.—Boy aged four years. Admitted to hospital on Oct. 16th, 1889, under the care of Dr. Drewitt. Had been ill a week. Diphtheritic membrane was present on tonsils. There was considerable laryngeal dyspnoea. The urine contained albumen. Intubated on the evening of admission. On Oct. 17th the tube was coughed up, together with a long membranous cast from the trachea. Dyspnoea soon returned and the tube was reintroduced. On Oct. 19th marked signs of broncho-pneumonia appeared, the temperature rising to 102° or 103° on this and subsequent days. The tube was removed on Oct. 20th, but had to be replaced. On Oct. 23rd the tube was coughed up, and as the breathing continued easy it was not again replaced. The lungs gradually cleared and the child recovered.

CASE 4.—Girl aged two and a half years. Admitted to hospital on Aug. 25th, 1890, under the care of Dr. Drewitt, with symptoms of bronchitis and laryngitis, which had existed for two or three days. Intubation was performed soon after admission, with immediate relief of the laryngeal dyspnoea. The tube remained *in situ* until Sept. 1st, when it was removed and was not further needed. The bronchitis gradually cleared up. Recovered. Two months later this child was readmitted with similar symptoms, was intubated and recovered.

CASE 5.—Boy aged one year and ten months. Admitted to hospital on Oct. 19th, 1890, under the care of Dr. Hood, with marked laryngeal dyspnoea. The child was much cyanosed on admission. No membrane visible in the pharynx. Intubated immediately, with complete relief. The tube was coughed up in the night and was immediately replaced by a larger size. During the following days the tube was removed twice, but had to be replaced on account of the dyspnoea. On Oct. 31st the tube was finally removed. On Oct. 30th signs of broncho-pneumonia set in, which ran a course of ten or eleven days. Recovered.

CASE 6.—Girl aged four and a half years. Admitted to hospital on Oct. 21st, 1890, under the care of Dr. Hood. She had suffered from symptoms of laryngitis for two days. On admission she was cyanosed, and had marked recession of lower ribs on inspiration. No membrane in the pharynx. Urine contained a trace of albumen. Intubated at once with