the bone had increased at this time, which was before his admission here. In fact, the union, which might have been sufficiently good to support the weight of the body if the two parts of the femur had united so as to preserve the proper axis of the limb, and to allow the shaft to bear naturally the pressure from above, was insufficiently firm to resist the same pressure when brought to bear upon a shaft in which the fracture was joined at an angle; and the uniting tissue, under these circumstances, yielded, and in time, it is not unlikely, had he persisted in moving about, would have given way altogether, and we should have had a case of what would be termed delayed union. It would have been said that the patient had moved about before the union was firm; or that, from some cause or other, firm union had been delayed; whereas the union was probably as good as in ordinary cases suffices to support the body, but in this instance was overstrained by the weight pressing at an angle instead of in the proper long axis of the thigh-bone. This case, then, represents a class in which longer time should be given for the consolidation of the repair than is needed when the long axis of the shaft can be properly preserved during the progress of union.

It is a somewhat similar condition which exists in cases of oblique fracture. The tibia—the bone of the leg which sustains the weight of the body—is often fractured so that two very oblique surfaces are left in contact. These specimens from the museum are examples of this injury. When such a hurt has to be treated, I always allow an extra fortnight—say six weeks in all—before the apparatus is disturbed and the patient allowed to put his foot to the ground; and for this reason. In these fractures, the whole weight of the man falls on and strains the new material, which has joined the fracture, in such a fashion that there is always a tendency for the upper fragment to glide off, or to be launched as it were, from the lower; the latter part of the bone giving little if any support, whilst in a transverse fracture, or a fracture only slightly oblique, it largely assists in bearing the superincumbent weight. Imperceptibly, it may be, in such a case, the union gives way; pain is felt by the patient; and, on re-examining the limb, repair is found to be incomplete. All the cases of non-union of the bones of the leg which have been brought under my notice, have been instances of oblique fracture; and every now and then, in these wards, we have found it necessary to put up afresh a fracture of this kind, upon which a patient has been allowed too soon to bear his weight.

Of fractures near a joint, that of the humerus in its lower third is, perhaps, of all the one most often followed by non-union. Various explanations have been offered with reference to this particular failure of bony union; but certainly in one case we could trace it to this, and I suspect, in most cases, it is in this direction that the cause is to be looked for. A fracture of the humerus is put up; and the forearm, at least for convenience, is placed at a right angle, and slung. As the case progresses, a question is raised as to the possibility of the elbow being stiff. The hand and forearm are flexed and extended a little, and the joint is said to move freely. If this examination be repeated, you must not be surprised if you find, when in due course you come to examine the fracture, that it has not united. The elbow being for the time somewhat stiff from disuse, it was not the joint which moved, but the fracture which was rocked in flexion and extension, as it easily may be when you act on it with the great leverage given you by the forearm, and so was hindered from becoming firm. I never knew an elbow-joint to be anything but temporarily stiff after a fracture of the humerus not extending into the joint; so that in these cases there is no excuse for handling the forearm. The arm should be left perfectly at rest during the entire process of repair; and no movement of the elbow should be permitted until it is certain that the bone is firm.

If you remember these as typical of certain classes of cases, you will be prepared to prevent the occurrence of non-union in most of the comparatively few cases in which special care will be called for. Of another class—that of failure of bony union from the difficulty of keeping the separate fragments in juxtaposition, as after transverse fracture of the patella—you may learn from this patient, recently discharged from Harley Ward, who has sustained an injury of this nature, that it is not impossible to obtain osseous union even under what at first may appear unfavourable circumstances, if only you give time for the process to complete itself, and take care that in the meanwhile no strain is allowed to stretch the fibrous tissue which at first unites the pieces of the broken bone.

HÆMORRHAGE AFTER TONSILLOTOMY.—As a means of preventing the sometimes severe and obstinate hæmorrhage which attends excision of the tonsils, M. Broca recommends compression of the carotid artery against the transverse process of the sixth cervical vertebra, so as to allow the formation of coagula in the small tonsillar vessels.

ON THROMBOSIS AND EMBOLISM: CASES WITH COMMENTS.*

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WITHIN a comparatively short period, I have met with a considerable number of cases of disease in which fibrinous coagula, large or small, either fixed or movable and floating within the circulatory system, have played a conspicuous part; and I trust that a brief narrative of some of these cases, together with some reflections suggested by them, may not be without interest for this Society. I will preface the recital of the cases by a very few general remarks.

The chief among the conditions which are known to determine the formation of coagula within the circulatory system are the following.

1. Stagnation of blood, or retardation of the blood-stream—as, for example, in an aneurism after ligature or compression of the artery; in the right side of the heart when there is an impeded escape of blood through the lungs or through the left side of the heart. Amongst the causes of an impeded flow of blood through the lungs, may be mentioned the state of apnoea which results from pneumonic consolidation from the pressure of liquid on the pleura, or from obstruction in the larynx. The operation of tracheotomy too long deferred may fail to save life, in consequence of coagula within the pulmonary artery, the right cavities of the heart, and the systemic veins. A coagulum may form in a dilated left auricle, consequent on constriction of the mitral orifice. Of this there is a good example in one of the specimens on the table. Again, coagula may form in the right side of the heart when, in consequence of weakness of the walls or dilatation of the cavities, there is an incomplete emptying of these cavities and a partial blood-stasis.

2. Coagula will form and adhere upon any part of the interior of the heart or of the vessels which may have been roughened or abraded by disease or accidental injury. Thus fibrinous concretions form upon the surface of a cardiac valve which has been roughened by inflammation, upon the rough ends of a ruptured tendinous cord, upon the edges of a crack in the endocardium or in the inner coat of an artery; and the blood may coagulate and form a thrombus within an artery which has been roughened by atheromatous or calcareous degeneration, or by syphilitic or other forms of inflammation.

3. A small coagulum often forms the nucleus of a larger concretion. Thus, as layer upon layer of laminated fibrine forms within an aneurism, or on the warty concretions covering an inflamed cardiac valve, so a nucleus of coagulated fibrine within one of the cavities of the heart may rapidly increase by determining a further deposition upon its surface; so minute floating particles of fibrine—capillary embola—may lead to the formation of larger coagula in the systemic veins, in the right side of the heart, or in the pulmonary vessels.

4. There is reason to believe that in certain states of system, such as are found, for instance, in rheumatic subjects, the blood contains either an excess of coagulable material or material which is more than ordinarily prone to coagulate, possibly in consequence of the presence of some morbid material in the blood.

My first three cases are examples of thrombosis in the right side of the heart—a result, as I suppose, of weakness of the heart's walls, dilatation of the cavities, and consequent partial stasis of blood in holes and corners of those cavities.

CASE 1.—Mr. H., aged 64, had not been in his usual good state of health for about four months before his death, which occurred on October 25th, 1871. In June, he had an attack of what was called "gastric disorder", after which he suffered from shortness of breath on walking uphill. On one occasion in particular, while walking up a steep hill at Dover, his breathing became very difficult. This was at the end of September. Immediately after this, he returned to his home in London, where I saw him occasionally with my friend Dr. Lavies, who was in daily attendance upon him. Mr. H. was then confined to his room with a feeling of general weakness, a quick and feeble pulse, hurried breathing on any exertion, some oedema of the ankles, and crepitation over the bases of both lungs. The heart's impulse was feeble, but there were no abnormal valvular sounds. The urine was normal. One of the most distressing symptoms was a sensation of alarm and impending suffocation on first awaking. There was a considerable amount of subcutaneous and abdominal fat, and we con-

* Read before the Medical Society of King's College.

cluded that we had to deal with a fat and flabby heart. This condition varied from time to time; but, on the whole, he appeared to be improving, under the influence of rest and judicious nursing. On the morning of October 25th, he awoke; told the nurse, who was sitting by him, that he felt comfortable; and asked the time. She said, "A quarter to three." He began to reply, "Oh, a quarter," but suddenly stopped in the middle of the sentence: the nurse turned and saw that he was black in the face; his tongue protruded between the teeth, both arms and legs were convulsed, and in a few minutes he was dead.

At the *post mortem* examination, we had the assistance of Dr. Kelly. There were small spots of ecchymosis beneath the pericardium. The heart was large, with much fatty growth on its surface. The right side, but especially the auricle, was much distended with blood. A firm decolorised coagulum filled the apex of the right ventricle, adhering closely to its inner surface, and extended about three or four inches along the pulmonary artery. Its diameter there did not appear sufficient to obstruct seriously the current of blood; but it was also continued backwards through the tricuspid orifice into the auricle, where it formed a band two inches long, an inch wide, and a quarter of an inch thick. The outer surface of this firm clot had been moulded on the wall of the auricle; but it was now free in the cavity, and it had evidently been driven by the current of blood over the tricuspid orifice, the sudden and complete obstruction of which had caused general venous distension, with lividity of the surface, and arterial emptiness, cerebral anæmia, and consequent convulsions. The lungs were gorged and oedematous at their bases; the bronchi were deeply congested, and contained much frothy mucus.

There can, I think, be no doubt that the abrupt arrest of the circulation in this case was caused by the separation of the fibrinous coagulum from the wall of the auricle, and its consequent valve-like flapping over the tricuspid orifice.

It is probable that this fatal clot had been gradually forming for some weeks before death; and that the immediate cause of its formation was the imperfect contraction of the fat and flabby right ventricle, and the consequent incomplete emptying of its cavity.

CASE II.—On November 7th, 1871, I saw Miss R., aged 19. She was breathing rapidly and deeply. She said, "I feel as if I could not get a sufficiently deep breath." Her lips were livid, and her skin pallid; the pulse small, rapid, and feeble. The hands felt cold; the temperature in the armpit was 98 deg. The chest in front was everywhere resonant, except over the middle lobe of the right lung, where it was quite dull. The respiratory sound was exaggerated (puerile), except over the dull space before mentioned, where the respiration was inaudible. The dyspnoea rendered auscultation at the back difficult; but moist râles were heard over both lower lobes. With these sounds, however, there was a free entrance of air. I expressed my belief that she was dying from obstruction at the right side of the heart or in the pulmonary artery. As she had spat some blood a few hours before, it was probable that the dullness over the middle lobe was the result of pulmonary apoplexy. My first visit was at 3 P.M. At 7 P.M., her condition remained the same. She died at 11 the same evening.

Dr. Kelly made an inspection with me, thirty-four hours after death. The middle lobe of the right lung was solidified by impacted blood. In other parts of the lungs, there were a few smaller hæmorrhagic patches, and also some ecchymoses beneath the pleuræ. The bronchi were congested, and contained much bloody tenacious mucus down to their smaller branches. This condition of the bronchi was general over both lungs. The cardiac area was rather larger than usual; a little clear fluid in the pericardial cavity. The right side of the heart was full of black coagulum; the left side moderately full. Each ventricle formed an apex, so that the heart's apex appeared double. The valves were all healthy. The right ventricle was dilated; its columnar carneæ and musculi papillares was thicker than usual. A large loose *post mortem* coagulum lay in the right auricle, ventricle, and pulmonary artery. In the appendix of the right auricle was an adherent, pretty firm, decolorised clot of fibrine, entangled in the musculi pectinati. Similar but smaller clots were found in the apex of the right ventricle, where the deep fossæ between the prominent musculi papillares form a complicated network most favourable for the formation of a thrombus. On opening the pulmonary artery, a clot was found at the commencement of each main branch; and firm decolorised clots were met with in the smaller branches as far as they could be dissected, so that these vessels were actually distended by coagula which had been driven into them by the right ventricle. The pulmonary veins were healthy, and contained a little fluid blood.

In the early part of the year, Miss R. had been much frightened by a domestic occurrence. From that time she suffered from occasional palpitation, cough, and difficult breathing. She was a zealous dancer;

but always, after waltzing, she looked pale, and suffered from palpitation. About three weeks before her death, she was much distressed by the loss of a locket, the gift of her deceased mother. She became feeble, and her breathing was oppressed. On October 31st, she had great difficulty in attending a wedding as bridesmaid; and, after the ceremony was over, she went to bed thoroughly prostrated. She suffered from increasing difficulty of breathing and palpitation, occasional sickness and cough, with frothy mucous expectoration, which had an occasional rusty tinge. On the morning of the day on which she died, she spat up about a tablespoonful of pure blood. This was followed by an increase of dyspnoea; and at 3 P.M. I found her in the condition which I have already described, the symptoms continuing unchanged until her death at 11 the same night.

In this case, dilatation of the right cavities of the heart—a result, probably, of the combination of feebleness of the muscular walls, emotional excitement, and over-exertion in dancing—led to the formation of coagula in those portions of the auricle and ventricle where, in consequence of incomplete contraction of the walls, the blood is liable to stagnate. Then branches of the pulmonary artery were gradually and successively blocked by embola detached from clots within the right cavities. At length, the obstruction to the pulmonary circulation was so great as to excite urgent dyspnoea, which was still further increased by the hæmorrhage and the consequent impaction of blood in the terminal bronchi and air-cells. It is probable that the blood escaped from ruptured bronchial capillaries, the result of a retrograde passive engorgement of the bronchial veins consequent on the block in the pulmonary vessels. The ecchymoses beneath the pleuræ were explained by the same backward engorgement of the bronchial veins and capillaries.

CASE III.—Mr. B., aged 33, a merchant, of somewhat intemperate habits, had hæmoptysis about the middle of March 1872. For this he was seen by Dr. Mumford, who found that he was passing copiously urine having a specific gravity of 1035, and containing much sugar. I saw the patient with Dr. Mumford on March 28th. In addition to the diabetic symptoms, there was crepitation at the apices of both lungs. On April 17th, the symptoms were somewhat mitigated. The patient was dressed and downstairs. On April 23rd, I was called to see him at 8.30 in the evening, in consequence of a serious aggravation of his symptoms. I found him in bed, breathing hurriedly and deeply. The hands were very cold and clammy; the pulse small, feeble, and rapid; the lips blue; the face pale and anxious. The respiration was puerile everywhere except at both apices, where loose crepitation was audible. The sounds of the heart were normal, except that the first sound was shorter than usual. I was told that difficulty of breathing had been gradually coming on for two days, and that the dyspnoea had been urgent for about twelve hours. I expressed my opinion that he was dying from a coagulum at the right side of the heart. He died at 11.30 A.M. the following day.

Thirty hours after death, Dr. Kelly examined the body with Dr. Mumford and me. A firm colourless clot was found adherent to the apex of the right ventricle; this extended along the pulmonary artery, becoming small and black beyond the bifurcation. At the pulmonary orifice, the clot was equal to about one-third the diameter of the vessel. It extended also backwards from the ventricle through the tricuspid orifice into the auricle, where, on the auricular side of the tricuspid orifice, it was enlarged to the diameter of nearly an inch; so that the tricuspid orifice was much obstructed, and the auricle was distended with dark liquid blood; while the ventricle was little if at all distended. The walls of both the auricle and the ventricle were thin, soft, and covered by fatty growth. The left cavities were empty. There was a moderate quantity of serous fluid in the pericardium, and some spots of ecchymosis beneath the serous layer. There were cheesy deposits in the apices of both lungs, and two small cavities in the left apex. The lungs were gorged with serum; the mucous membrane of the bronchi was congested, and covered by frothy mucus. This engorgement with mucous exudation must have come on after my visit—i.e., during the last fourteen hours of life, a result of passive congestion of the bronchial veins and capillaries, in common with the whole systemic venous system, consequent on the obstruction at the right side of the heart. The serous effusion into the pericardium and the ecchymoses beneath the membrane were results of a similar passive engorgement of the coronary veins and capillaries.

The formation of the clot in this case probably began in the apex of the right ventricle—a result of partial stasis of blood there, consequent on degeneration and impaired contractile power of the muscular walls. The patient, for some weeks before his death, had been subjected to frequent and violent emotional excitement, which may probably have increased the distension of the right cavities, while it tended to lessen the contractile power of their walls.

It is not without interest to remark that the symptoms which result

from a plug of fibrine at the right side of the heart or in the pulmonary artery bear a striking resemblance to some of the most characteristic symptoms of cholera collapse. Dr. Alfred Carpenter has published a remarkable case of embolism of the pulmonary artery, in which, as he says, "the only symptoms wanting to make the case apparently one of cholera were alvine discharges and cramps in the limbs." (*Lancet*, September 23rd, 1871.) The symptoms actually observed in that case were blueness of the surface, coldness of the uncovered parts of the body, cold clammy perspiration, coldness of the breath, sinking of the eyes, feebleness of the voice, a feeble thready pulse with hurried breathing, excessive thirst, and almost complete suppression of urine, two ounces of urine only being passed one day, and another day less than two ounces.

An embolon passing into the pulmonary artery, and not causing a fatal obstruction there, may excite inflammation of the tissue of the lung.

CASE IV.—I am indebted to my friend Dr. I. for the outline sketch of an illness of which he was himself the subject. He is in his fifty-second year, and has had repeated attacks of gout. While travelling in Switzerland, he sprained his left knee severely on August 3rd, 1872. On the 7th, he had gout in that joint and in the ankle on the same side. Next day, the left foot was inflamed; and on the following day, the right foot. On August 12th, the gout was gone, and the knee better. On the 15th, he went downstairs. On the 16th, 17th, and 18th, the calf of the left leg was increasingly painful. On the 16th, while sitting quietly, he felt a curious sensation in the left calf and along the thigh, and felt sure that a clot had started on its travels. In about two minutes, he felt it settle in the right lung, two inches below the nipple. He was not at first conscious of any dyspnoea; but a friend called, and in conversation he found that he was short-breathed. A short irritative cough came on. Pulse 76. On the 21st, the pain in the left calf was so severe as to compel him to lie in bed. He then found that the leg below the knee was much swollen and hard. The dyspnoea was so great that he could neither read aloud nor talk. On the 24th, there was pain along the course of the femoral vein, and the vein felt hard and distended. On the 28th, the whole left limb was from two to two and a half inches larger in diameter than the right; dyspnoea more considerable. The temperature of the left leg was 104 deg.; that of the right, 98 deg. He had extreme debility, and complete loss of appetite. On August 29th, he left Zermatt in a litter at 7.30 A.M., and reached Visp at 6.30 P.M. August 30th, he was in bed all day, with pain in the right side. On August 31st, he was travelling by carriage and rail from 7 A.M. to 1 P.M. The dyspnoea was so severe that he could with great difficulty take half a tablespoonful of soup at a time. In the afternoon, there was intense pain below the right mamma, and the physical signs of pleurisy were present. On Sept. 1st the cough was more severe; he expectorated one large solid mass of blood and mucus, followed by three smaller masses; and the cough ceased for the day. Sept. 2nd. Cough and expectoration returned from 4 A.M. to 6 A.M., then ceased; pleuritic pain continued. On Sept. 3rd, he slept soundly, for the first time since the sprain, thirteen hours; sweated profusely; the morning cough and bloody expectoration were as before. He went to Geneva in an invalid carriage; on arrival, the dyspnoea was extreme, so that he could not talk in his usual tone of voice. From this period, all the pulmonary symptoms gradually abated, and his appetite and strength returned; but the limb remained enlarged and painful. I saw Dr. I. in London on September 30th. There was then no trace of mischief within the chest; and, with the exception of the remaining swelling and weakness of the left lower extremity, he was in his usual good health and spirits.

In this case, the original source of the mischief was a clot which had formed in a vein in the leg, consequent on a partial blood-stasis connected with the gouty inflammation.

CASE V.—On October 26th, 1871, I saw, with Mr. Erichsen and Mr. Jones of Epsom, a gentleman aged 64, who, having had much mental anxiety for several months, suffered from sciatica on the left side. This came on in May, and continued for several weeks. Then, one day in July (?), he was seized with sudden faintness at the water-closet, and nearly died. A second faint occurred the following morning. Then there followed the signs of pulmonary engorgement, wheezing over the chest, hæmoptysis, and signs of consolidation of a portion of one lung. The lungs gradually became clear; then followed a painful swelling of the left leg and thigh; and afterwards the right limb was similarly affected. When I saw him, both lower limbs were still swollen, and the left was discharging freely from punctures in the skin. The heart's sounds were normal, but the impulse was feeble. The pulmonary sounds were normal. There was a moderate quantity of albumen in the urine. I saw this gentleman again with Mr. Jones on February 16th. I then learnt that the swelling of the legs had gradually passed away; the urine had ceased to be albuminous. About a fortnight be-

fore, he had a sudden faintness, continuing for about twenty minutes; since then, he had a conviction that, if he swallowed food, it would not pass through him. He consequently refused nourishment, and became very feeble. The pulse was 120, weak, sometimes irregular; the heart's impulse feeble; the first sound sharp and clear. He gradually sank, and died from exhaustion.

In this case, it is probable that a thrombus, originally formed on the right side of a feeble and perhaps dilated heart, passed as an embolon into the pulmonary artery, where it caused pulmonary congestion and hæmoptysis; then coagula, passing into the systemic arteries and capillaries, led to the formation of coagula, first in the veins of one leg, and subsequently in those of the other. It is probable that the albuminuria may have been a result of embola in the renal vessels.

There is, I think, good reason to believe that coagula, which have originally formed within the pulmonary capillaries, may thence pass through the systemic arteries and cause capillary and venous obstruction in the lower extremities.

CASE VI.—In the autumn of 1869, I saw the following case. A married lady, aged about 40, the mother of a large family, healthy, but of a somewhat delicate frame, during the early part of September had a troublesome dry cough; and one day she began to cough up a large quantity of florid blood. So rapid was the hæmorrhage, that at one time she was nearly suffocated; and the active bleeding continued for several hours. I first saw her at Eastbourne, on September 17th, a week after the onset of the bleeding. She was then expectorating small quantities of semi-solid dark blood, which had evidently been for a considerable time out of the vessels. Percussion and auscultation over the front of the chest gave quite normal results; and, as the chest-walls were thin, the air could be heard entering the lung with great clearness. Over the right lower lobe at the back, there was marked dulness on percussion, and, on auscultation, rather fine crepitation, with diffused blowing expiration. The inference was, that the right lower lobe had been partially consolidated by blood driven into its tubes and air-cells. Percussion and respiratory sounds over the left back were quite normal. The pulse and breathing were somewhat quickened, but there was no febrile excitement. A few days afterwards, I heard that the chest-symptoms were better, but that the left leg below the knee had become painful and swollen, though not inflamed; and I suggested that probably some coagula from within the pulmonary capillaries, having entered the circulation, had led to the formation of coagula within the capillaries and veins of the leg. On October 4th, I again saw the patient. I found that, during the last few days, the painful swelling of the leg had entirely subsided; but this had been succeeded by urgent dyspnoea. The breathing was still upwards of 40 in a minute; the pulse rapid and feeble; the countenance anxious; the face pale; the lips livid. There was now normal resonance over the lower lobe of the right lung, the air entered freely, and there was scarcely a trace of crepitation left. Over every other part of the chest, percussion and respiration were quite normal. The blood which, for a time, had blocked a portion of the lung-tissue, had been expectorated; and evidently there was nothing in the state of the lung to explain the alarming dyspnoea. What, then, was its cause? Probably a fibrinous clot, from the temporarily obstructed vein in the leg, had made its way to the right side of the heart; and there was embolic obstruction of the pulmonary artery. The dyspnoea was due, not to want of air in the lungs, but to want of moving blood. After my visit, the dyspnoea continued and increased, and she died early on the morning of October 6th.

There was no inspection of the body, but there can be scarcely a doubt as to the sequence of events. Pulmonary hæmorrhage led to impaction of blood in the lower lobe of the right lung; this was soon expelled by coughing; but, meanwhile, probably coagula, formed within the pulmonary capillaries while the blood was rendered stagnant by pressure from without, passed into the circulation, and led to the formation of coagula within the capillaries and veins of the leg; thence coagula found their way back to the right side of the heart, where they increased and caused a fatal obstruction to the circulation.

CASE VII.—In confirmation of the explanation which I have given of this and the previous case, I may refer to a case related by Sir James Simpson (*Clinical Lectures on the Diseases of Women*, p. 354). A woman died of phlegmasia dolens of the left arm and side of the face some weeks after an exhausting labour. About a year before she became pregnant, she had an attack of rheumatic endocarditis. After her delivery, she had signs of embolism, first in the right brachial artery, and then in both legs; then came on phlegmasia dolens in the left arm. After death, the aortic valves were covered over with wart-like excrescences, and it is suggested as probable that fibrinous detritus, passing from the arteries into the capillaries and veins of the left arm, caused phlebotic thrombosis and the other phenomena of phlegmasia

dolens. It is notorious that coagula in the right side of the heart and in the pulmonary artery have frequently caused the death of parturient women. This fatal occurrence appears sometimes to have been unassociated with symptoms of blood-poisoning or of constitutional disturbance; and it seems not improbable that coagula from the uterine veins may pass into the returning current of venous blood and so form the nuclei of further concretions at the right side of the heart.

CASE VIII.—Five years ago, I saw, with Mr. Charles Matthews, a woman who, a few days after her confinement, had been suddenly seized with the most alarming symptoms of collapse. Mr. Matthews having been called to her at this time of alarm, heard a bellows-sound over the situation of the pulmonary artery, and he concluded that a clot was partially obstructing the vessel. On the following day, when I saw her with Mr. Matthews, the alarming symptoms had passed away, and the cardiac and pulmonary sounds were quite normal. A few days afterwards, she had phlegmasia dolens in one leg, and it is probable that fibrinous *débris* from the clot in the pulmonary artery may have passed through the capillaries of the lung into the systemic arteries, and thence to the capillaries and minute veins of the limb which was the seat of phlegmasia dolens. The passage of fibrinous coagula from the pulmonary capillaries into the systemic arteries, and the resulting phlegmasia dolens in one leg, would render this case analogous to Cases 5 and 6, which I have before narrated.

The late Dr. F. W. Mackenzie, in his elaborate paper on phlegmasia dolens (*Medico-Chirurgical Transactions*, vol. xxxvi), has shown that obstruction of the femoral or iliac vein causes soft oedema of the legs, but that, in order to excite the hard painful swelling which constitutes phlegmasia dolens, the smaller vena radicles must also be obstructed; and he shows it to be probable that this obstruction is often the result of some morbid element in the blood which has gone the round of the circulation. Thus, in one experiment (p. 210), lactic acid having been injected into the right femoral vein of a dog towards the heart, not only were the right iliac veins and some of their branches obstructed by a firm adherent decolorised coagulum, but the right femoral vein, down to the popliteal and several of its branches, were similarly obstructed, and a firm coagulum also occupied the left femoral vein down to the popliteal. The coagula in the left femoral vein and in the right femoral beyond the place of injection are attributed to the action of the lactic acid after it had gone the round of the circulation.

Phlegmasia dolens, although in the majority of cases puerperal, is of by no means rare occurrence in non-puerperal conditions. Thus, out of 100 cases referred to and analysed by Dr. Mackenzie, no fewer than 40 were non-puerperal, and of these 40 about one-third occurred in males. Even in parturient women, it is doubtful whether it is ever a result of a direct extension of disease from the uterine to the pelvic and iliac veins. It seems on the whole more probable that some morbid material, possibly small fibrinous coagula or minute particles of fibrine in a state of decomposition, passing from the uterine veins into the general current of the circulation and returning through the systemic arteries, determines the coagulation of the blood in the capillaries and veins.

The greater frequency of the disease in the lower than in the upper extremities is probably accounted for by the retardation of the blood-stream in the more distant parts, where, too, the venous current has to return against gravity. It has also been suggested that the greater frequency of the disease in the left than in the right leg, in the proportion of three to two, may be, in part at least, explained by the fact that the left common iliac vein has to pass behind the right iliac artery, and when the blood-moving forces are feeble, it is thought that the pressure of the artery upon the vein may somewhat retard the venous current, and so favour coagulation of the blood. It is well known that swelling of one or other leg, sometimes of both—a form of phlegmasia dolens—is an occasional sequela of typhus and of typhoid fever. This complication has occurred in several cases under my own observation. What is the explanation of this phenomenon? While thinking over this subject in connexion with the history of the cases which I have here narrated, it has occurred to me that probably the swollen white leg after fever may be thus explained. Pulmonary engorgement, more or less, is a common result of fever, both typhus and typhoid; during this state of engorgement, coagula may form in the pulmonary capillaries, and, subsequently passing into the systemic circulation, may cause capillary and venous obstruction in a manner analogous to that in which I suppose it to have occurred in Cases 5, 6, and 7.

In a fatal case of typhoid fever (Sarah Gaily, xxvii, p. 69), in which there was much pulmonary engorgement, there was found after death a recent fibrinous patch in one kidney, a result, as I suppose, of the impaction there of fibrinous coagula from the pulmonary capillaries. The valves of the heart were quite normal, but the fibrinous patch was exactly similar to those which are often associated with warty concre-

tions on the cardiac valves.* The probability of this being the true explanation of the swelled leg after fever is increased by the fact that a similar complication is occasionally observed in cases of phthisis, pleurisy, and pneumonia, in which cases it is likely that coagula may have passed from pulmonary capillaries into the systemic vessels. I have met with this complication in several cases of phthisis. The late Dr. Cursham has published four cases of phthisis complicated with phlegmasia dolens (*Medico-Chirurgical Transactions*, vol. xliii). In two of these cases there was ulceration of the intestines, but in the other two no such ulceration was found; and Dr. Cursham attributes the obstruction of the veins to "the presence of pus or some foreign matter in the blood."

Dr. Warburton Begbie, in a recent paper on the swelled leg of fevers (*Edinburgh Medical Journal*, September 1872), refers to four cases of pleurisy in which swelling of the lower limb occurred on the same side as the pleurisy. It is likely that coagula formed within pulmonary capillaries where the circulation has been retarded by the pressure of liquid in the pleura might pass on into the systemic vessels, and there become nuclei of new clots and consequent capillary and venous obstructions. If the occurrence of the swelling in the limb on the same side as the pleurisy be not a mere coincidence, it may possibly be due to the fact that, as a rule, a pleuritic patient lies on the affected side, and this position may involve greater pressure on the undermost leg and some additional retardation of the venous current in the compressed limb.

The lobular pneumonia which so often follows an extensive burn of the skin has for its probable exciting cause the passage of more or less completely coagulated blood from the seat of the burn into the minute pulmonary vessels; while the ulceration, which in these cases has sometimes been found at the commencement of the duodenum, may be explained by coagula passing through the pulmonary capillaries, forming embola in the intestinal vessels, and thus rendering circumscribed portions of the mucous membrane liable to be softened and dissolved by the acid chyme before it is mixed with the alkaline biliary secretion. These duodenal ulcers appear to be analogous to the so-called simple gastric ulcer; the most probable explanation of which is, that plugging of vessels in the mucous membrane by thrombosis or embolism kills a circumscribed patch of the membrane, and thus renders it soluble by the gastric fluid.

Time permits only a passing allusion to the various forms of brain-disturbance which are direct results of embolism or thrombosis. We have had in the hospital some most interesting typical cases.

In the case of a man named Edward Smith, in October 1867, with a diastolic bellows-sound at the base, there was sudden left hemiplegia; after death a plug in the right middle cerebral artery, with softening of the corresponding corpus striatum, warty vegetations on the aortic valves. In Sarah Elizabeth Keen, Dec. 1869, there was sudden coma, with first rigidity, then palsy of the right arm and leg; after death a thrombus was found in the atheromatous left middle cerebral artery, with softening of the whole corpus striatum and the outer part of the optic thalamus. In the last volume of the *Pathological Transactions* (xxiii, p. 94), Dr. Kelly has published a case of acute fatal chorea in a girl aged 9. Amongst other points of interest, there was softening of both corpora striata. Some minute arteries in the softened parts were filled with granular fibrine. There were recent soft vegetations on the mitral and tricuspid valves. It is certain that acute chorea is sometimes a direct result of capillary embolism of the corpus striatum and the neighbouring parts.

Another most interesting case is that of a young woman named Mary Ann Hennessey (vol. xliii, p. 269). She had rheumatism twelve years before admission; palpitation since; a systolic bellows-sound at the apex, sometimes high pitched and musical, then soft and blowing. The changing character of the murmur was probably a result of the alternate deposit and dislodgment of fibrinous concretions on the mitral valve. In addition, there was high and variable temperature; then plugging of the brachial artery, hæmaturia, probably from renal embolism; lastly, cerebral excitement, delirium, and illusions, consequently, as we suppose, on plugging of the cerebral vessels. In this state she insisted on going home, where we heard that the excitement soon passed off and she was able to attend to her shop.

I venture to make one generalisation which appears to me to be warranted by clinical observation. The symptoms to which a stationary clot gives rise by engorgement of vessels extending in a backward direction are usually of a passive character, and are developed slowly; on the contrary, when coagula, large or small, pass onwards with the blood-stream, they often excite active symptoms with great rapidity; the character of the symptoms being almost infinitely various, according to the nature of the part which is

* In some cases of typhoid fever, it is not improbable that coagula or some other form of blood-contamination may start from the vessels at the seat of the intestinal lesion.

implicated—whether lung, brain, kidney, intestine, etc.; and, again, whether arterial trunk, minute arterial branches, capillaries, or veins. Minute particles of fibrine mingling with the blood may probably go the whole round of the circulation with, of course, a liability to be arrested in any set of capillaries, and thus to become the centre and source of fresh mischief. There is reason to believe, too, that, apart from the mere mechanical results of floating masses of fibrine, fibrinous *débris* in the blood may have something of a toxic influence, and thus induce febrile excitement with a high temperature.

I cannot conclude without an expression of admiration for the clinical acumen of the late Dr. Kirkes, who was the first in this country to indicate what, in his paper in the *Medico-Chirurgical Transactions* (vol. xxxv), he calls “some of the principal effects resulting from the detachment of fibrinous deposits from the interior of the heart, and their mixture with the circulating blood.” That paper opened out an entirely new field of pathological and clinical research, the importance of which is yearly becoming more apparent.

NOTES OF A CHOLERA-EPIDEMIC.

By CHARLES A. BRIGSTOCKE, M.R.C.S., Medical Officer and Public Vaccinator in the Calne Union.

ALTHOUGH cholera has visited this country as an epidemic three or four times, its cause and means of propagation are still so enveloped in mystery, that all additional statistics may prove of service, however unimportant in themselves.

The cholera-epidemic of 1866 visited the town of Llanely (where I was engaged in active practice at the time) with great severity, no fewer than two hundred deaths from that cause having occurred in the town and immediate neighbourhood. I am decidedly of opinion that the more than usually fatal character of the scourge in this case may be fairly attributed to the following causes: first, the situation of the town, being in great part built upon marshy badly drained land near the junction of a river with the sea; and, secondly, the nature of the population, composed chiefly of work-people employed in the various collieries—iron, copper, and tin-works in the town and neighbourhood, with a large percentage fluctuating between pauperism and self-support. When to these unfavourable influences we add the fact that pigs were kept by many of the inhabitants in immediate proximity to their dwelling-houses, it would have been difficult to find a town more favourable to the development of cholera. There was scarcely any wandering epidemic but found a nidus there; while fever was an almost constant resident. It seems only fair to the local authorities to state, that since the visitation of cholera more attention, I believe, has been paid to the improvement of the sanitary condition of the town.

On July 4th, I visited a woman, aged 27, suffering from severe choleraic diarrhoea; and on the 7th, the first well marked case of cholera occurred in the district. The epidemic remained apparently quiescent for a short time, but then spread with frightful rapidity. At its acme, from fifteen to twenty-five attacks occurred daily; it then gradually declined, the last death from cholera happening in the early part of October.

During its spread, although it displayed a marked preference for the filthiest localities, yet, as if by exception to prove the rule, one or two of apparently the most healthy parts of the district suffered severely.

The following statistics of 192 cases of cholera, which fell under my own personal observation, may prove interesting.

Cases.		Deaths.	
Under 5 years	24	13	54.16 per cent.
From 5 to 20 years	33	9	27.27 „
„ 20 to 40	64	19	29.68 „
„ 40 to 60	49	18	36.73 „
„ 60 to 80	20	8	40 „
„ 80	2	1	50 „
Total...192		68 or 35.41 per cent.	
Age of Males attacked.		Deaths.	
Under 20 years	36	11	30.55 „
From 20 to 40 years	33	8	24.24 „
„ 40 to 60	23	8	34.78 „
„ 60	10	2	20 „
Total...102		29	
Age of Females attacked.		Deaths.	
Under 20 years	21	11	52.38 „
From 20 to 40 years	31	11	35.48 „
„ 40 to 60	26	10	38.46 „
„ 60	12	7	58.33 „
Total...90		39	

From the above tables it will be seen that, although the disease attacked the series in not very unequal proportion, the deaths were

much more numerous in the female sex, the percentage of deaths to attacks being in the case of males 28.43, and in females 43.3.

With regard to liability of attack; while the infirm and badly fed seemed prone to suffer, intemperance, contrary to general belief, did not appear to materially increase the liability; but, when attacked, those of intemperate habits had a much greater mortality: two of the patients, after recovery from severe choleraic diarrhoea, became intoxicated and sank during the algid stage in a few hours. Parturition occurring during cholera seemed (in the five or six cases which I attended) to produce an increased and favourable effort on nature's part to throw off the disease. One woman delivered of twins, however, sank rapidly, although, even in her case, the improved pulse and temperature gave evidence of the endeavour to rally. I need scarcely add that all the children were still-born. There did not appear any increase to miscarriage or abortions during the epidemic. The symptoms of cholera are so well known that I need not recapitulate them, but I may remark, *en passant*, that in the cases of three or four children there was very little vomiting or purging present. Severe cramps were the exception, not the rule. Most of those who died in the algid stage retained consciousness to the last; but in the majority of fatal cases occurring among children, convulsions or coma closed the scene; and, indeed, in the cases above mentioned, where little vomiting or purging was present, coma was present almost from the commencement of the attack, as if the choleraic virus acted narcotically on the system. Consecutive fever of any intensity was met with in but few cases, although two died from the resulting brain-congestion.

With regard to therapeutics in cases of true cholera, no drug seemed of much avail. Those who took medicine, and those who abstained from doing so, seemed to die with great impartiality. One man, who swallowed an ounce of carbonate of soda in mistake for sulphate of magnesia, recovered; another (a very severe case), who drank a quart of beer with brandy in it, rallied from that moment, and attributed his recovery to it. Of those drugs employed, small doses of calomel or of carbonate of soda seemed, if anything, to have a favourable effect. An endeavour to maintain the temperature of the body by means of hot bottles to the axillæ, feet, etc., with a supply of cold water to drink, seemed almost all (with one's present knowledge of the therapeutics of this disease) that one could do in the way of remedial measures. I think, considering the passive condition of the system, as evidenced by the suppression of urine, the dry skin, and intolerance of the alimentary canal, it is not surprising that drugs have no effect. Too much stress cannot be laid upon the importance of attending to the diarrhoea by which it is often, but not always, preceded, particularly as it is believed that, during an attack of diarrhoea, the body is more susceptible of the choleraic virus. Of course the diarrhoea may be treated in the ordinary way; but I may remark that, during an epidemic of cholera, it does not yield readily to treatment; and there would appear to be a much greater tendency to disturbed action on the part of the alimentary canal (evidenced by more or less pain), even where actual diarrhoea is not present. Many confirmed smokers were compelled to temporarily abandon the habit, owing to the resulting bowel-disturbance. As little alteration as possible should be made in one's habits, particularly as to diet. The quantity of vegetables and of stimulants taken might perhaps be slightly diminished; but even this would appear to require great care.

It would have been ludicrous, were not the subject such a serious one, to notice the precautions and nostrums so loudly vaunted at this time. Some invariably carried stimulants with them, to which they applied themselves with such industry that the amount of drunkenness was materially increased. Others looked upon spirits of camphor as an infallible means of safety. Among the workpeople, colliers, etc., in particular, whole families were dosed with this remedy, with the effect upon some of the infant children, as may be imagined, of producing symptoms of poisoning. To such an extent did the camphor mania run, that almost any price was given for the drug in the district. But it would be more easy to ridicule such treatment, were the medical profession able to do more towards the reducing of this disease.

With regard to sanitary matters in infected districts, all proper precautions should be taken when cholera is anticipated; but, during its presence, measures of this sort must be very cautiously carried out, lest fresh food be supplied for its propagation. All the ejecta should be carefully and speedily removed, and covered with earth or lime. The water-supply of the district should be particularly looked to. In the Llanely district, the disease spread rapidly along the course of contaminated water-supplies. Yet so slow are the public to sufficiently estimate the importance of this source of danger, that, in the case referred to, the only means found successful in preventing the use of contaminated water was the emptying loads of lime into the streams.

Measures should also be taken for separating those attacked from those