

ACUTE SUFFOCATIVE PULMONARY OEDEMA.

T H E S I S

for the

DEGREE OF M.D. GLASGOW

submitted by

M. COHEN, M.B., Ch.B., 1923.

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ACUTE SUFFOCATIVE PULMONARY ŒDEMA.

The subject of acute suffocative pulmonary œdema has received a good deal of attention in France and Germany. In preparing my thesis I have availed myself freely of the literature of those countries, especially of the former.

In the following dissertation after defining the condition, I shall deal with its history, etiology, pathogenesis, morbid anatomy, clinical features, differential diagnosis, prognosis and treatment. The pathogenesis and treatment will receive particular attention. Finally, I shall give my conclusions and append a number of instructive cases.

D E F I N I T I O N .

The term œdema signifies a pathological condition in which fluid accumulates excessively in the tissues or cavities of the body. In œdema of the lungs serous fluid with leucocytes in a more or less considerable quantity is present in the connective tissue spaces of the pulmonary tissues, and in the air-vesicles and smallest bronchi.

Acute pulmonary œdema may be defined as a morbid

syndrome constituted by a sudden serous transudation from the lung-capillaries into the pulmonary interstices, alveoli and bronchioles. This serous inundation of the cavities of the lungs impedes the respiratory function and may become an obstacle to the pulmonary circulation by compressing the capillaries. The resulting severe dyspnœa with the feeling of impending suffocation accounts for the designation of "Acute suffocative pulmonary œdema" as usually applied to the condition in this country. This form of title appears to have been first employed by Dr. (afterwards Professor) Lindsay Steven in a clinical lecture delivered by him in the Glasgow Royal Infirmary on March 8th, 1901.

Clinically, acute œdema of the lungs constitutes a definite entity characterised by the sudden onset of intense dyspnœa, a copious, frothy, serous expectoration of albuminous nature, and abundant moist râles all over the chest. It may rapidly lead to death from asphyxia.

It may be pointed out here that pulmonary œdema of insidious onset is usually terminal and indicates that the patient is dying, or, as Cohnheim puts it, a man does not die because he gets œdema of the lung: he gets œdema of the lung because he is dying. But acute suffocative pulmonary œdema may suddenly appear in an individual who was, at the time being, apparently in good health and may actually be the cause of death.

H I S T O R Y.

The condition is mentioned for the first time by Laennec in 1819. His description is supplemented by Andral who, in his annotations to the fourth edition of Laennec's treatise on mediate auscultation (1837), says that besides chronic or passive oedema of the lungs must be described an acute or active oedema of the lungs characterised by a rapid course comparable to that of oedema of the glottis. He distinguishes an acute form and a hyperacute form of abrupt onset with extreme orthopnoea and a rapid termination. Before that, in 1834, in volume III of his "Clinique médicale", Andral had reported four cases in connection with different diseases: chronic bronchitis, pneumonia, pleural effusion, and aneurysm of the heart. He already said that it depended on some unknown congestive factor in its mechanism, and that it should not be confused with inflammation.

Fournet in 1839 simply points out the diagnostic difficulty between acute pulmonary oedema and pulmonary congestion. Castelnau (1845) mentions the condition as a complication in a case of rheumatic fever.

In 1846 Legendre describes it in the course of scarlatina, but is partly deceived about the anatomical seat of the oedema which he localises exclusively in the cellular tissue of the lung.

It was carefully studied by a Lyons doctor, Devay (1855), and Béhier and Hardy (1855) also admit hyperacute pulmonary oedema capable of rapidly causing death by asphyxia.

Barthez and Rilliet (1861) recognise the existence of an acute or hyperacute symptomatic form of pulmonary oedema.

In 1869 Grisolles expresses a doubt about the existence of this clinical entity. But Fernet (1865), Ball (1866), Houdé (1869), Besnier (1873), and D'Espine and Picot (1877) admit the clinical reality of this affection.

In 1873 Souin de la Savinière devotes his thesis to the study of acute pulmonary oedema in scarlatina, measles, emotional states, pneumonia, bronchitis, following thoracentesis, in the drowned, and reports some similar cases in the course of Bright's disease.

Cazalis (1874) and Schweninger (1876) quote respectively cases of acute pulmonary oedema in the course of typhoid fever and following fatty embolisms of the lung. Bernheim (1877) has had the opportunity of observing it in acute articular rheumatism. Lasègue (1879) in his study on albuminuric bronchitics describes pulmonary oedemas in the course of Bright's disease.

Lund of Christiania (Oslo) reports in 1878 four cases relating to patients who were found dead in the morning in bed and in whom autopsy revealed the existence of a very intense oedema of both lungs.

In 1881, de la Harpe of Geneva reports the case of a patient who died within a few hours, and of another who had ten to fifteen attacks without succumbing and during each attack assumed the

aspect of a dying man suffocated by a frothy liquid which quickly filled his respiratory passages.

Lebreton (1884) describes among the pulmonary complications in rheumatics an oedematous form which corresponds to acute oedema of the lungs. Jaccoud in his "Cliniques de la Pitié" (1883-1884) describes a case of fulminating pulmonary oedema in the course of typhoid fever. A case observed by Renaut of Lyons is described in the thesis of his pupil Honnorat (1887). Bouveret of Lyons (1890) in a very interesting memoir describes well acute pulmonary oedema in Bright's disease. In the same year Huchard communicates to "La Société médicale des hôpitaux" eight cases of acute oedema of the lungs in aortic affections.

Then appear the interesting communications of Professor Dieulafoy (1892) on hyperacute oedema of the lungs in Bright's disease, and the study of Vinay (1896) on this accident in the course of pregnancy.

Works relating to this subject have also been published by Hertz (1874) and by Eppinger (1898) in Germany; and in France by Fouineau (1898), by Chemery (1908) who pays particular attention to the pathogenesis of this condition, and by Le Calvé (1925).

One may also mention the thesis of Méyohas (Montpellier, 1901) on acute oedema of the lungs in articular rheumatism, and that of Fabre (Toulouse, 1905.)

In England, interesting articles appear in the Lancet (1902)

by Professor Lindsay Steven of Glasgow and by Lissaman. The latter describes the case of a woman who had seventy two attacks in two and a half years. A case is reported by Waldo in the Lancet (1895) and another by Rowland in the same journal in 1902. Leonard Williams has called attention to this condition in the Lancet (1907). Correspondence following his article appears in the issues of the Lancet of December 14 and 21, 1907. I reported a case in the British Medical Journal (1926), and this provoked many contributions to the subject in subsequent issues, and indicated that the condition was less rare than was previously thought. Altogether I have met with, personally, three cases of acute pulmonary oedema, and describe them on pages 106, 151, and 155 of this work.

Collective series of communications on this subject describing many cases appear in the issues of the British Medical Journal of April 17, June 19, and October 30, 1926. A concise review by Professor Wynn appears in the Medical Annual, 1927.

A. Birrell publishes an account of some examples of this syndrome in the Bristol Medico-Chirurgical Journal, 1927. He has found that venesection brings relief from the attack, and notes that the blood flows centripetally - that is, from the opening in the vein on the distal side of the incision.

In Feuchtwanger's historical novel, "Jew Süß", the Duke, Karl Alexander, has a sudden attack of "choking catarrh" and quickly succumbs "not to an apoplexy, nor to an inflammation,..... but to a catarrh which choked him so that he was suffocated."

The pathogenesis of this accident has given rise to numerous

works. Fraentzel (1889) explains the development of acute oedema of the lungs in Bright's disease by a disturbance in the equilibrium of the left and right ventricles. This interpretation seems to be supported by the experimental researches of Welch (1878) carried out under the direction of Cohnheim, from which it seems that the most efficient cause of pulmonary oedema lies in a weakening of the left heart. But Sahli (1885) from his experiments concludes that pulmonary oedema is not produced by this mechanism.

Grossmann (1887), von Basch (1887), and Löwit (1893) carried out experiments with muscarine which, according to the two former, promptly produces in animals a pulmonary oedema. Owing to the physiological antagonism between atropine and muscarine Grossmann has proposed to combat this oedema with atropine.

Bouveret (1890) has studied this complication from the pathogenic point of view in Bright's disease, and he thinks that this enormous congestion of the lungs, which starts so abruptly, results from a trouble in the vasomotor innervation of the pulmonary circulation. The difficulty is to establish the point of origin of the morbid stimulus which provokes the paralytic dilatation of the pulmonary vessels.

Vinay (1896) has studied acute oedema of the lungs in pregnant cardiopaths, and attributes it to a toxic origin. He compares acute pulmonary oedema with eclampsia and the other

convulsive disorders which may appear in pregnancy, the variability in the localisation of the poison being responsible for the difference in the symptomatic manifestations. Tonnel (1897) also ascribes to this oedema a toxic origin.

An important discussion regarding the pathogenesis of acute oedema of the lungs took place at "L'Académie de médecine" in 1897. Huchard maintains that the condition is due to aortic and periaortic affections. Professor Dieulafoy believes that patients attacked by acute pulmonary oedema are sufferers of Bright's disease more or less latent, and does not agree that this accident can be attributed to a cardiac cause, but is dependent on a renal origin. He insists on venesection as the treatment of choice and that milk régime must be absolute. For Debove the condition is always due to Bright's disease; but, on the other hand, as in chronic Bright's disease the heart is always affected, it also may be incriminated in the pathogenesis of acute pulmonary oedema.

Brouardel (1897) has always noted in subjects who have died suddenly from acute oedema of the lungs the existence of renal and cardiac lesions; but the latter were sometimes absent. In these predisposed subjects cold is an important factor. This author raises the medico-legal question of pulmonary oedema. He says: "There are cases which it has been my lot to see fairly frequently. A patient begins to choke. It is evening, and one of the physicians on night-duty is called. He gives an injection

of morphia, following an only too common rule; the patient gets no relief. A second and third injection at length bring quiet; some minutes or hours later the patient succumbs, and the family blame the physician who has given the injections. Autopsy and inquest follow. It is found that death was due to super-acute oedema of the lungs."

Hallion and Carrion in 1899 bring about pulmonary oedema with intravenous injections of massive doses of sodium chloride.

At the International Congress of Medicine in Paris in 1900, the question of acute pulmonary oedema was the object of two interesting reports by Masius of Liège and Teissier of Lyons. Prof. Teissier in his report, and later in the thesis of his pupil Miecamp (Lyons, 1900), expounds a complex pathogenesis including mechanical, toxic and nervous elements.

In 1905 appeared the work of Josué who seeks to establish the rôle of hyperadrenalism in the pathogenesis of certain cases of acute oedema of the lungs.

Chemery in his thesis (1908) attributes the pathogenesis to arterial hypertension, retention of chlorides, and a nervous element causing a sudden vasodilatation of the pulmonary arterial system.

Le Calvé in his learned, encycloplædical study, "L'Oedème" (1925), favours the vasomotor theory. He regards the essential cause of the attack as a pulmonary vasodilator reaction depending on various factors.

In the Journal of Physiology (1926) appears a paper by R.K. Lambert and H. Gremels who, from their experiments, affirm that the real cause of pulmonary oedema lay in the changes which occur in the endothelium of the capillaries.

The history of acute oedema of the lungs following evacuatory tappings of the thorax or abdomen commences with Pinault of Châteauroux who observed in 1853 a case of albuminous expectoration following thoracentesis. He attributed this accident to the sudden activity produced in the pulmonary circulation following the evacuation of the pleural effusion and leading to a considerable afflux of blood, "the most liquid part of which transudes through the membranes to the surface of the bronchial mucosa where there thus appears sometimes a very considerable quantity of serum." This explanation is the one that is now universally accepted. It is well supported by Hérard (1872), and Moutard-Martin (1877) also defends it.

Legroux in 1863 and Woillez in 1872 propose another explanation, that of traumatic perforation of the lung by the trocar, allowing the pleural effusion to escape into the bronchial tubes. Marotte in 1872 also adopts this hypothesis.

Béhier in 1873 advances the theory of a spontaneous perforation. He thought that a bronchial fistula was produced which re-opened with each dilatation of the lung following the tappings.

Another theory, that of the spontaneous reabsorption of

the liquid remaining after the thoracentesis, was only tentatively suggested by several authors.

In 1873 Terrillon in his thesis discusses these diverse theories, and his conclusion, frankly hostile to traumatic perforation, oscillates between spontaneous perforation and congestive oedema.

At a discussion introduced by Ferréol before "La Société médicale des hôpitaux" in 1873, this last theory of oedematous congestion first suggested by Pinault, and so ably sustained by Hérard, received the greatest support from the speakers. During this discussion Lande of Bordeaux published a memoir also in favour of this hypothesis.

Mercier, in his thesis (1876), also deals with the pathogenesis of acute oedema of the lungs after thoracentesis.

Jouglà of Toulouse in 1879 reports a case of acute pulmonary oedema following paracentesis abdominis. He regards the pathogenesis of this phenomenon as analogous to that brought forward to explain cases following thoracentesis, the lungs being able to be compressed by an ascites as well as by a pleural effusion. To the mechanical element of abrupt decompression he adds a nervous factor for the production of this accident.

We are indebted to Professor Renaut of Lyons for the study of the morbid anatomy of acute oedema of the lungs (1897).

E T I O L O G Y.

Acute pulmonary œdema is not uncommon, though very inadequately recognised in this country (Langdon Brown, 1924). It occurs especially in individuals over fifty, without predilection for either sex. It is relatively rare in the child, in whom the causes are the same as in the adult, but, contrary to what is observed in the latter, aortic and renal lesions are exceptionally implicated. In children the attack occurs rather in connection with an infectious disease such as scarlatina, measles, diphtheria, tonsillar affections and influenza, or with bronchitis and broncho-pneumonia. S. Mc. Naughton (1926), who calls attention to its occurrence in children, considers that the so-called acute suffocative catarrh is the same condition, and that there are probably many cases of less acute or subacute oedema of the lungs in children which are not fatal and are diagnosed as acute bronchitis. It is not uncommon to see a child who has suddenly developed a severe cold; examination shows some difficulty in breathing, and loud râles are heard all over the chest; a diagnosis of a very acute bronchitis is made, and a guarded prognosis is given, but next day the child may be found quite well. He considers that this subacute oedema of the lungs is essentially anaphylactic in nature. The mother may state that the patient on a day or two previously had eaten something

unsuitable.

The attack supervenes more frequently during the night than the day, occasionally after an excess of some kind like a hard day's work or a copious repast; but usually there is no history of a preceding intemperance. Cases occur in winter as in summer. However, the cold season, by predisposing to respiratory affections, seems to favour the occurrence of acute pulmonary oedema. Le Calvé maintains that the soil for the attack is often prepared by a preceding affection of the respiratory passages creating in the lungs a *locus minoris resistentiae*.

The two most important causes of acute oedema of the lungs appear to be affections of the kidneys and of the aorta. Dieulafoy gives the preponderating part to the kidneys, whilst Huchard to affections of the aorta. It is then in arterio-sclerotic subjects in whom the aorta is diseased and the kidneys sclerosed that acute pulmonary oedema will be principally observed. The condition has also been described in cardiac affections, nervous diseases, intoxications and morbid blood-states, infectious diseases, pulmonary affections, following thoracetensis, and it has also occurred idiopathically.

1. In cardiovascular and renal lesions.

Huchard in his fundamental memoir (1890) reports eight cases. Seven of the cases relate to individuals affected with aortitis, acute, subacute, or chronic, complicated or not with angina pectoris. The eighth concerns a woman affected with aortic stenosis. Nearly all his cases are similar. They are aged individuals who, in the course of aortitis, manifest or latent, are suddenly without antecedent phenomena, seized with very severe dyspnoea; on auscultation subcrepitant râles are audible over the chest; percussion yields exaggerated resonance of the chest; and they bring up abundant expectoration with much albumin. Two of the cases deserve special mention, as in them the attack of pulmonary oedema supervened at the end of acute cardiac failure. In those two patients the lower limbs became rapidly oedematous, and in one of them it was noticed that the pulse became feeble, showing the arterial tension to have undergone a considerable lowering.

Apart from Huchard's, few other cases are found where the pulmonary complication is attached exclusively to aortitis. We may mention a case of Du Cazal (1890) in generalised arteriosclerosis, another of Fouineau (1898) and one of Lesage (1896) coinciding with aortic atheroma. Lund observed two deaths from acute pulmonary oedema with aortic and coronary atheroma.

Apart from aortic lesions, there is a certain number of cardiac affections which have been regarded as determining

causes of œdematous congestions of the lungs. A patient of Jacquet (1883) and one of Parisot and Spillman (1897) died from a sudden attack of pulmonary œdema coinciding with aneurysm of the heart. Autopsy disclosed in both cases a renal lesion with atheroma of the aorta.

A case of Lund and four cases of Huchard supervened as complications of mitral stenosis.

Fouineau in 1898 reports a case of acute pulmonary œdema following sudden obstruction of the left coronary, another in generalised arteriosclerosis with interstitial myocarditis, and a third also in chronic myocarditis.

Merklen in 1900 gives the case of a vigorous boat-breaker who was seized with acute pulmonary œdema on two occasions when trying to lift a too heavy load which caused sudden cardiac failure.

Huchard at the Congress of Paris in 1900 related the interesting case of a woman affected with mitral stenosis and latent lesions of the kidneys and aorta, suddenly seized with acute pulmonary œdema, and in whom autopsy verified the clinical diagnosis of an auricular thrombosis obstructing the left auriculo-ventricular orifice.

The commonest cardiac lesion in cases of acute pulmonary œdema is aortic regurgitation. M. Rockfelt (1926) reports the case of a woman of 52 with aortic regurgitation who had nine attacks in a year. I had under my care a patient, a

young man of 33, with severe aortic and mitral incompetence, who had four attacks in one and a half years. All his paroxysms occurred at night awakening him from his sleep.

Most cases as seen in general practice occur in connection with advanced cardiovascular sclerosis with high blood-pressure or with valvular disease of the heart. A.E.Hodder has studied a series of cases from 1921 to 1925, and found the one factor common to all to be an advanced degree of cardiovascular sclerosis. Valvular disease was present in some, but not in others; in all there was, just prior to the attack, evidence of auricular fibrillation. S.M. Hebblethwaite (1926) also considers paroxysmal auricular fibrillation to be a cause.

According to the French observers, it is in chronic interstitial nephritis that acute pulmonary oedema is most frequently encountered. Numerous examples are reported by Legendre (1846), Devay (1855), Deckherr (1872), and de la Harpe (1881). Jaccoud points out its frequency in albuminuria. Fraentzel (1889) mentions it in connection with cardiac hypertrophy due to Bright's disease.

Then have appeared the works of Bouveret in 1890, and of Huchard, Tonnel, Dieulafoy and Giraudeau in 1897. We shall quote some of their cases. This oedema of the lungs in Bright's disease resembles, as Legendre was the first to remark, oedema of the glottis in Bright's disease. Troisier has observed acute pulmonary oedema in a case of pneumonic nephritis.

Although this accident is so frequent in chronic interstitial nephritis, it is rare in acute nephritis. In the few cases where it has occurred, the acute nephritis was not an isolated affection. Béhier (1858) observed a death from acute oedema of the lungs in a man who had acute nephritis. Autopsy revealed also pneumonia with diffuse bronchitis and an old pericarditis. Fouineau reports a case in acute nephritis following influenza. Merklen at the Congress of Paris in 1900 cited the case of a patient who had chronic interstitial nephritis till then latent, suddenly seized with acute pulmonary oedema, due to an exacerbation of acute nephritis occasioned by influenza.

2. In nervous disorders.

Fouineau describes the accident in the course of a subacute ascending myelitis, and attributes it to an attack of the vasomotor centre of the lung. Morel Lavallée reports a case in the course of tabes. Lévi has observed it in a hysteric in whom he ascribes it to a purely nervous origin. It has occurred as a complication of an epileptic fit. L. Langeron (1925) describes a case of epilepsy developing at 40 years of age in which each attack was accompanied by acute pulmonary oedema. He believes that an irritation, inducing changes in the vasomotor roots of the sympathetic, may have produced an intense vasodilatation which caused the

œdema, and that the same irritation in the cerebral cortex gave rise to the epileptic attack.

In his thesis, Souin de la Savinière devotes a chapter to acute œdemas of the lung supervening under the influence of powerful emotional states. He concludes by asking: "Why may not the lung become hyperaemic under a psychic influence as well as the face which blushes under the influence of an emotional state?" He reports the case related by Breschet of a prisoner who, a few hours after having heard pronounced a happy sentence which had pleasantly surprised him, was seized with severe dyspnoea and succumbed.

In the same group of cases may be ranked those of Tyrrel Edye and H. Müller published under the name of paroxysmal angioneurotic œdema of the lung. But, as Renaut observes, these particular cases have really a more complex pathogenesis, for the patients had mitral stenosis and one had undergone thyroidectomy.

The cases of pulmonary œdema supervening under the influence of intense and prolonged cold enter into the same category, as the cold may influence the nervous system directly, or indirectly by provoking toxic products due to nephritis a frigore. But in the majority of cases of pulmonary œdema provoked by cold, the latter only plays the part of determining cause in individuals in imminence of œdema because of aortic disease or chronic Bright's disease.

We may include here the cases observed in the course of intestinal obstructions, and those of Falk, where the sudden œdematous congestions also appear primarily due to a nervous disturbance. In one of his cases it followed the ingestion of an iced drink, and in another it followed a wound of the heart leading to paralysis of the left ventricle and determining a reflex on the vasomotors of the lung.

The tapping of an abundant ascites is also capable of leading to acute oedema of the lungs. Such a case is reported by Jouglà (1879).

Obstruction to the entrance of air into the lung, as by a foreign body or by stenosis of a bronchus, has also caused the appearance of acute pulmonary œdema (Fouineau).

Woillez (1872) has seen two cases following simple traumatism of the chest. Amblard (1906) describes a case supervening in the course of a phlegmonous peritonsillitis, and attributes this accident to compression of the vagus by an inflamed gland.

3. In intoxications and dyscrasias.

Arthritics, so subject through their dyscrasia to nervous disorders and congestive complications, are frequently attacked by acute œdema of the lungs. It also occurs in gouty individuals, especially those affected with biliary or renal lithiasis. In such gouty persons and arthritics it is subject to recurrences, and the rôle of intoxication may be invoked. It also occurs in

diabetics. Moreover arthritics, chronic rheumatics, gouty patients, and diabetics are most often arteriosclerotic with arterial hypertension, in whom the kidneys sooner or later become more or less incompetent and so lead also to intoxication.

Under this heading may be included Bright's disease, as owing to renal incompetence toxic products may be retained in the organism.

Pregnancy seems to predispose to serous infiltrations of the lungs. Grisolle (1865) says: "Death may supervene suddenly in pregnant women; at autopsy is found a serous infiltration of the lungs dependent on organic disease of the kidneys." Vinay (1896) observed two cases of acute pulmonary oedema in pregnant women affected with cardiac lesions. In both cases the urine contained albumin. He says: "It is evident that those two patients succumbed to acute pulmonary oedema rather than to actual cardiac failure, and in my opinion the alterations of the kidney played a more important part than the mechanical troubles of the cardiac circulation." Natale, in 1898, also reports a case of acute oedema of the lungs occurring in a pregnant woman with cardiac disease.

Acute pulmonary oedema has occurred in debilitating conditions, such as severe anaemia.

Some exogenous intoxications determine attacks of acute pulmonary oedema. Individuals in a state of acute alcoholism and exposed to cold may die suddenly from the appearance of

this accident. However, in such cases autopsy often revealed renal lesions (Brouardel). Intoxication by the venom of serpents may determine an analogous attack (Hertz and Eppinger).

The intravenous injection of an iodine-containing solution causes the death of dogs by a mechanism of considerable oedema of the lungs. Huchard has seen pulmonary oedema supervening in a case of aortic aneurysm following the administration of two grams of potassium iodide. The employment of iodoform in surgery has occasionally determined similar accidents in those affected with Bright's disease. Pilocarpine has also caused this accident (Jaccoud). So has morphine. It has also followed the employment of strophanthin in too large a dose, and arsenical preparations.

Muscarine produces in animals an acute congestive oedema of the lungs. Grossmann attributes the oedema supervening in these cases to a spasm of the left ventricle, the right ventricle continuing to send blood into the pulmonary circulation. Experimentally acute oedema of the lungs has also been produced by means of concentrated solutions of sodium chloride (Carrion and Hallion, 1896 and 1899), amyl nitrite (Winkler, 1898), methyl salicylate (Chatin and Guinard, 1900), and amyl-salicylic ether (Chanoz and Doyon, 1900). Adrenalin has been utilised for the production of acute pulmonary oedema in experimental animals by Baylac of Toulouse (1900), Takamine (1901), Bouchard and Claude (1902), Josué (1905), and Hallion

and Nepper (1910).

Delamare and Descazals (1897) have shown that acute pulmonary oedema may follow intravenous saline injections. Pozzi (1896) has shown that intravenous injections of artificial serum may be blamed for having produced acute oedema of the lungs by sudden augmentation of blood pressure.

It may be produced by the inhalation of irritating gases, acute oedema from gassing with chlorine or phosgene having occurred during the Great War. Cases of death from acute pulmonary oedema have been described in connection with ether anaesthesia (Poppert, 1894) and during chloroform inhalation (Smith, 1899).

Pedersen of New York contributes to *Annals of Surgery*, 1906, a valuable study on "acute pulmonary oedema secondary to ether narcosis," in which he reviews the cases reported in literature in this connection. He describes his case, which recovered, in detail, and writes: "The behaviour of the heart was most instructive, because, while the oedema was developing, its action was, broadly speaking, normal, although accelerated, and only after the oedema was so fully established, that the respiratory function had begun to fail, were unfavourable signs from the heart noticed. It seemed probable, therefore, that the oedema was primary in the lungs and not in the right heart." But he adds that ether occasionally has a selectively depressing action on the right heart, and, having excited this phenomenon, may set up oedema in the lungs secondarily.

4. In infectious diseases.

Acute pulmonary oedema has been reported in many of the infectious diseases.

Measles.— Laennec describes it in 1819; Barthez and Rilliet in 1861. Devay (1855) observes it during a severe epidemic of measles. Souin de la Saviniere (1873) thinks that this complication in measles may be attributed to renal lesions. Brouardel (1897) believes that exposure to cold plays some part.

Scarlatina.—Barthez & Rilliet (1861) report the case of a child of eight years who in the course of nephritis with anasarca following scarlatina, had a suffocating attack of pulmonary oedema and died in a few moments. A similar case is described by Legendre in 1846, in a child of four, whilst it was desquamating from a mild scarlatina. Devay in 1855 quotes the case of a patient who had anasarca from a scarlatinal nephritis and who succumbed quickly from acute oedema of the lungs. Souin de la Savinière (1873) and Renaut also describe cases of acute pulmonary oedema in scarlatina.

Small-pox. Miecamp, who in the service of Professor Courmont made many autopsies on variola patients, reports in his thesis (1900) that he found in some cases evidence of oedema of the lungs. He thought that those cases might have been bronchoplegic forms of the condition where death

occurred before the appearance of the characteristic expectoration.

Typhoid Fever.— Jaccoud in his "Cliniques de la Pitié" (1883) describes the case of a patient suffering from mild enteric fever, who was suddenly seized with an attack of pulmonary oedema one morning on rising to go to the W.C. He explains his death through a reflex of intestinal origin transmitted to the pulmonary vasomotors. Fouineau (1898) reports the case of a man of 42 years with aortic incompetence of rheumatic origin dying suddenly from acute pulmonary oedema complicating enteric fever.

Influenza.— According to Professor Teissier influenza is the infection of choice as a provoking agent of acute oedema of the lungs. In 1893 he wrote: "What characterises influenza is a mixture of congestion and oedema. If the cases of acute pulmonary oedema are not apparently very numerous, it is because here the phenomenon is often associated with exudations of congestion and bronchopneumonia." However, Rendu has observed two typical cases of acute pulmonary oedema in the course of influenza, free from any pneumonic or bronchopneumonic condition. During the epidemic of influenza of 1918 all were familiar with the intensely oedematous condition of the lungs seen in some cases of that disease (Wynn). Moreover, influenza is very frequently accompanied by nephritis, and to the latter

have been attributed the pulmonary complications which may supervene. But the influenza toxin itself may also play a part in the production of acute oedema. Fouineau reports in his thesis several examples in nephritis following influenza.

Encephalitis Lethargica.— Le Calvé states that acute oedema of the lungs has been described as a complication in lethargic encephalitis.

Pneumococcal infection.— Guillain and Laroche (1910) have described a primary form of acute oedema of the lungs of pneumococcal origin. They observed this in an individual of 45 years of age, without any renal or aortic lesion, who was seized by an attack of pulmonary oedema in the course of which he brought up an expectoration containing pneumococci. Blood-letting arrested the serious signs of asphyxia.

Rheumatic Fever.— Castelnau publishes a case in 1845 of hyperacute oedema of the lungs in acute articular rheumatism. Devay (1855) describes another case, and quotes in this connection Trousseau and Pidoux who say: "The oedemas of rheumatism do not occur only at the periphery of the body; they may occupy deep-seated organs, e.g. the lungs, where veritable exudations are seen to develop with the suddenness of invasion of rheumatismal congestions."

Legroux (1863), Fernet (1865), Ball (1866), and Besnier (1873) report some cases. D'Espine and Picot (1877) in

their treatise describe sudden pulmonary congestion and œdema in connection with the sudden death of children in rheumatism. Bernheim (1877) concludes that the oedematous localisations in the lung in rheumatism may precede the articular localisations, appear simultaneously with them, or supervene later.

Landouzy reports two cases in rheumatic fever followed by cardio-aortic trouble. He observed in these patients several attacks of acute œdema supervening suddenly in the night. Méyohas (1901) describes several definite cases in acute rheumatic fever.

Choléra nostras. Rommelaere (1897) reports the case of a patient who succumbed from acute pulmonary oedema in the course of a choleraic infection. But as there existed at the same time interstitial nephritis with albuminous urine, atheroma of the aorta and discrete tuberculous lesions at the apices, the choleraic infection may not have been the principal or determining cause.

Erysipelas.— Joseph Franck states that acute pulmonary œdema may occur in the course of convalescence of erysipelas.

From the study of acute pulmonary oedema in the various infectious diseases, it may be concluded that infection is a very favourable condition for the production of this accident.

5. In Pulmonary affections.

In many cases of acute œdema of the lungs evidence of some kind of previous affection of the pulmonary parenchyma or bronchi is to be found (Le Calvé).

Bezançon, de Jong and Jacquelin (1921) attribute in cardio-renal cases a great importance to the existence of antecedent broncho-pulmonary lesions which play a predisposing rôle. They regard a chill as the frequent exciting cause, leading to a slight acute febrile infection, following which bursts out the paroxysm of pulmonary œdema.

Andral in 1834 collected three cases of sudden œdema of the lungs in different diseases of the respiratory apparatus, viz., chronic bronchitis, pneumonia, and pleural effusion. Laennec had previously noted the occurrence of this accident after pneumonia. He says: "This affection (pneumonia) appears to leave after it a great disposition to infiltration of the pulmonary tissue."

Devay describes the occurrence of acute pulmonary œdema in connection with tuberculosis, gangrene of the lung, and bronchitis. He recalls that Cruveilhier explained rapid death in the aged by a mild pneumonia leading suddenly to an intense pulmonary œdema with death from asphyxia.

Souin de la Savinière in his thesis regards pneumonia as a powerful cause of pulmonary œdema. He quotes a case of Béhier where acute œdema of the lungs supervened in a patient with acute bronchitis and nephritis a frigore.

Masius at the Congress of Paris in 1900 emphasized the production of oedema of the lungs by collateral congestion around pneumonic areas. Merklen (1903) says: "Acute pulmonary oedema constitutes in pneumonia a terminal, and often agonising accident; but in a person with manifest cardiac disease or in subjects of latent cardiac insufficiency (aged, obese, alcoholics) it may appear as a previous complication or occur along with the pneumonia. From the pathogenic point of view may be invoked the double influence of the pneumococcal infection, the direct cause of the pneumonic oedema, and the cardiac insufficiency which favours its diffusion and generalisation."

Lascazas of Saint-Martin reports in his thesis (1906-1907) a case observed by Carnot in the course of pneumonia. The complication has been seen to supervene in pneumonias of normal appearance, and as a terminal phenomenon in some grave pneumonias. As already mentioned, Guillain and Laroche have described a primary form of acute pneumococcal oedema.

Bouchut (1844) reports the case of a woman recently confined dying from acute pulmonary oedema following a thrombosis of the left pulmonary veins. A case following fatty pulmonary emboli is described by Schweninger (1876).

Mercier quotes in his thesis a case of Desnos where autopsy disclosed only discrete tuberculosis of the apices with right-sided pleurisy. In the thesis of Riory (1898)

is described a case of Professor Teissier where an attack of acute oedema supervened in a patient with an interlobar empyema. On withdrawing 800 grams (26 ozs.) of pus by thoracentesis the attack ceased in about an hour, thus demonstrating its reflex nature.

In 1898, Poulain, from observation of a clinical case, concludes that a simple modification of the resistance of the pulmonary parenchyma suffices to determine acute pulmonary oedema in predisposed individuals.

In the present thesis, I record the case of an elderly woman, a sufferer from chronic bronchitis and emphysema, whom I attended in 1926 for an attack of acute oedema of the lungs. Atropine and a liberal venesection overcame the seizure.

6. Following thoracentesis (oedema a vacuo).

Cases of acute pulmonary oedema following thoracentesis have been described by many observers. The different opinions as to the mode of production of this accident following thoracentesis have already been indicated:

- (1) perforation of the lung by trocar (Legroux and Woillez);
- (2) the reabsorption of the fluid remaining after the thoracentesis;
- (3) spontaneous perforation (Béhier); and
- (4) the transudation of the sero-albuminous fluid across the

alveolar walls owing to a rapid pulmonary congestion (Pinault).

The objections to the first theory are: Acute oedema does not supervene after thoracentesis except in cases where the effusion is very abundant; in these cases the lung is very distant from the thoracic wall, reduced to a small volume, and therefore it is very unlikely that the trocar would reach it. Also there is an absence of blood in the expectoration or in the liquid obtained by thoracentesis. Finally, auscultatory signs indicating a hydropneumothorax, which would result if the lung were wounded, are absent.

The second theory is also untenable. If there remains any fluid after the thoracentesis it cannot be reabsorbed all of a sudden. The inflamed pleura, covered with false membranes, absorbs little and slowly. Admitting that the pleura may be charged with this reabsorption, it is surely not into the alveoli that the fluid would go but rather into the circulation, in virtue of the physiological law which demands that all liquid absorbed by a tissue should pass into the circulatory current.

The hypothesis of spontaneous perforation of the lung is also out of the question. It is very unlikely that the spontaneous perforation which is not produced when a voluminous effusion distends the pleural cavity and exerts a continuous pressure on the lung, should appear when there is less fluid in the pleura and consequently less pressure. But the most

serious objection that may be offered is the absence of a pneumothorax; this alone suffices for the rejection of this theory.

However, the fourth theory is the one that is now generally accepted. There occurs a rapid pulmonary congestion which leads to the serous intra-alveolar exudation. Hérard says: "When the lung has been for a long time compressed by an effusion, at the moment when, following the expulsion of the liquid, the organ resumes its normal size, there occurs in it a kind of serous or sero-sanguineous outpour,... it is this serum which is expelled by the bronchi."

In the cases where acute pulmonary oedema has followed thoracentesis a pathological condition was usually found superadded to the pleural effusion. In Girard's case the patient had acute articular rheumatism with double pleurisy. Béhier's case had a tuberculous broncho-pneumonia. Aortic incompetence and stenosis was present in Terrillon's case. In Gombault's case the lung on the opposite side of the pleurisy was fibrous and adherent because of an old pleurisy, whilst Dumontpallier's case had bronchitis and adhesions of the left lung with right-sided pleurisy. In that of Bouveret there was an old tuberculous pleurisy with adhesions. In other cases was obtained a history of an infectious or toxic condition like scarlatina, rheumatism, or tuberculosis.

Moreover, Dieulafoy remarked that when no complication

was associated with the pleurisy, the accidents have coincided with the withdrawal of a large quantity of fluid or with a too rapid issue. It may be concluded that acute oedema of the lungs following thoracentesis is associated either with pathological complications or with the rapid withdrawal of a too large quantity of fluid, and more often with these two conditions combined.

The sudden decompression and the concomitant complication enable the appearance of acute pulmonary oedema to be explained. As Moutard-Martin says: "When a limb has for a long time been compressed by a fracture-apparatus, it becomes oedematous when the apparatus is removed, because the tonicity of the tissue has disappeared and the compression has paralysed the capillaries." Similarly in the case of the lung, the compression by the pleural effusion produces a kind of paralysis of the capillaries. This paralysis, following the decompression, permits the sudden afflux of blood, which will be all the greater the more sudden the decompression, the capillaries being so-to-speak taken by surprise.

The production of oedema is favoured by the increased permeability of the walls of the pulmonary capillaries brought about by the long inaction of the capillary circulation owing to the compression, and also by the concomitant or previous affection, toxic or infectious, which has damaged the nutrition of the compressed capillary walls and modified

the molecular concentration, and therefore the osmotic tension, of the fluids which bathe them.

7. Idiopathic.

Occasionally acute pulmonary oedema occurs without any apparent underlying cause. I performed an autopsy on a man, aged 48, who had jumped out of bed in the middle of the night and expired. He was somewhat obese. There was a very intense œdematous congestion of both lungs with frothy fluid in the bronchi and trachea. On section of the lung, much frothy fluid escaped, even without pressure, from the cut surface. All the other organs appeared quite normal. This man had gone to bed in his usual state of health having made no complaint to anybody. His attack of pulmonary oedema may be regarded as of the bronchoplegic variety, being of such severity that it killed him before he could bring up any expectoration. Attacks of acute œdema of the lungs without obvious cause may recur, as in Lissaman's patient, who had seventy-two attacks in two and a half years.

P A T H O G E N E S I S.

Various theories have been suggested to explain the production of acute pulmonary oedema. The German school have proposed the mechanical theory, regarding acute oedema of the lungs as the result of cardiac disorders. Huchard, who maintains that the condition is due to aortic and periaortic affections, explains the mechanism by an intense pulmonary vasodilatation resulting reflexly from a stimulus from the cardio-pulmonary nervous plexuses. Supporters of the toxic or renal theory, among them being Brouardel, Debove and Dieulafoy, consider that the accident is the result of autointoxication nearly always symptomatic of chronic interstitial nephritis. Some explain the phenomenon by a combination of several elements — mixed theories. Whilst Josué (1905) has put forward the suprarenal theory, which applies, according to him, only to certain cases of acute oedema of the lungs.

More recent suggestions as to the pathogenesis are anaphylaxis and auricular fibrillation, but there is not sufficient evidence that either of these is the essential cause. Wynn writes: "Many of the cases resemble attacks of angio-neurotic oedema, and are possibly anaphylactic in nature."

1. Mechanical theory.

Grossmann and von Basch were able to determine attacks of pulmonary œdema in animals by injections of sulphate of muscarine. This causes a spasmodic contraction of the cardiac muscle, the contraction being more marked in the left half. This is the theory of "cramp of the heart." The blood-stream thus hindered in its flow to the left ventricle, there results an increase of pressure in the left auricle. By retrograde propagation of this elevated pressure across the pulmonary circulation there results hypertension in the pulmonary artery, which is increased by the right ventricle continuing to expel through the pulmonary artery a nearly normal quantity of blood which accumulates in the pulmonary circulation owing to the obstruction to the flow in the region of the contracted left ventricle. The latter expels into the greater circulation only a small quantity of blood, and thus there results a considerable fall of pressure in the arterial system.

Lówit repeated Grossmann's experiments and obtained contrary results. At this Grossmann undertook a series of more detailed experiments which confirmed his original findings.

Lichtheim and Cohnheim do not agree with Grossmann's opinion. By ligaturing the aorta above the diaphragm and

all the pulmonary veins they tried to produce oedema of the lungs, but in spite of the enormous increase of pressure thus engendered in the pulmonary circulation they could not obtain this result.

Welch, from experimental work on the rabbit, carried out under the direction of Cohnheim, considers that it is not a question of spasm of the left ventricle but a paralysis of this ventricle - a sudden failure of the left ventricle. The right ventricle continuing to function in spite of the failure of the left ventricle, there results a rupture of equilibrium between the energy of the two ventricles, the left heart being unable to expel in a unit of time as much blood as is expelled by the right heart. Consequently there results rapid excess of tension in the pulmonary arterial system, engorgement and dilatation of the pulmonary capillaries, transudation of serum across their walls into the alveoli and surrounding connective tissue spaces, and acute oedema of the lungs. After some time when the pulmonary arterial system has become disgorged, the left ventricle recovers and by increased activity may manage to restore the equilibrium.

In addition to experimental work supporting Welch's assumption, there is pathological evidence in favour of it. Fraentzel supports Welch's theory from a clinical case he observed. Langdon Brown writes: "The most probable sequence

of events is this: the left heart is already loaded to its full capacity; the proverbial last straw is too much for it, and it breaks down, while the right heart goes on beating still, forcing blood into the lungs until they become engorged, since they are unable to shut off any of the blood-supply by vaso-constriction. An outpouring of serum occurs into the alveoli in such quantities that the patient is drowned in his own secretion. Two facts support this view: the commonest cardiac lesion in these cases is aortic regurgitation, which is known to terminate not infrequently in sudden stoppage of the heart; and venesection (10 to 12 ounces), according to French authorities, is the only effective treatment, and this would relieve the overloaded right heart and the stagnant pulmonary circulation."

Various objections have been raised against Welch's theory and that of Grossmann. By experiments on the dog, Sahli was unable to confirm their hypotheses. By ligatures placed at different levels on the left ventricle of a rabbit in such a way as to progressively reduce its capacity, Montanari decreased the ventricle by two-thirds of its volume without result. Oedema occurred, but in an inconsistent manner, with a greater diminution. In France, Teissier and Guinard, from experimental work carried out on dogs, have shown that very intense and sudden mechanical obstructions are unable by themselves to determine pulmonary oedema, in

spite of the enormous tension induced in the pulmonary circulation.

Furthermore, there is a lack of clinical evidence showing a fall of blood-pressure at the beginning of the crisis, whilst that ought to occur with a ventricular cramp or paralysis of the left ventricle. Many observers, including Bouveret, Vinay and Merklen have definitely noted the maintenance of normal tension of the pulse at the outset. Pedersen observed this in his case following ether narcosis, and I was able to confirm this in two of my own cases.

2. Vasomotor theory.

Bouveret considers that acute oedema of the lungs results from a disorder of the vasomotor innervation of the pulmonary arterial system. He, however, does not indicate the starting-point of the morbid stimulus which provokes the paralytic dilatation of the pulmonary vessels. This is left to Huchard. The latter, having observed the frequency of aortic and periaortic lesions in subjects who had attacks of acute pulmonary oedema, believes them due, like Bouveret does, to a pulmonary vasomotor disorder. He says: "In aortic incompetence, in coronary angina, in interstitial nephritis, in mitral stenosis of arteriosclerotics, there is always between these different affections and acute oedema of the

lung a necessary intermediary: it is aortitis, or especially periaortitis, with its inflammatory or reflex reaction on the cardio-pulmonary nervous plexuses. This fact is confirmed by the researches of Ranvier, who has so well demonstrated the rôle of nervous and vasomotor disorders in the production of œdemas. That is not all: acute œdema of the lung is often preceded by a considerable decrease of aortic tension and by an enormous and sudden increase of the pulmonary tension. Against the latter the right ventricle struggles and hypertrophies, and as long as it can struggle the œdematous inundation of the lung is prevented; but should its power suddenly become weaker for one reason or another, then acute œdema of the lung supervenes with great rapidity. So it is not the incompetence of the left ventricle that must be incriminated, but that of the right ventricle."

Thus according to Huchard the pathogenic sequence is: disorders of the cardio-pulmonary innervation by aortitis or periaortitis, considerable increase of tension in the pulmonary circulation, acute or rapid failure of the right ventricle.

However, Sahli and Teissier have shown in their experiments that, when the conditions for producing pulmonary œdema are fulfilled, incompetence of the right ventricle hinders it from supervening. Furthermore, if aortic and periaortic lesions have been frequently observed in patients

subject to attacks of acute pulmonary oedema, many cases have been observed where there was not the slightest trace of such lesions. That aortic lesions are not essential for the production of this accident is also seen from the fact of its occurrence following thoracentesis.

Moreover, the above doctrine of Huchard is not exclusive even for himself, for he says: "Renal sclerosis and aortitis are two manifestations of the same affection. It is therefore very natural to admit as the cause of pulmonary oedema the renal lesion and the aortic lesion." This opinion is shared by Brouardel who has often noted in subjects dying from acute oedema of the lungs the existence of renal and cardiac lesions; but the latter, he adds, are sometimes lacking.

The experimental researches of Teissier and Guinard may be invoked in favour of this aortic and renal theory. But these authors have not managed to produce pulmonary oedema unless they combined with intense mechanical obstructions or nervous excitations a toxic element, the intravenous injection of methyl salicylate.

On the other hand Debove, Landouzy and Renaut have seen acute pulmonary oedema in patients who had only aortic lesions and no alterations of the kidneys. Whilst Dieulafoy and Brouardel describe cases of acute oedema of the lungs having renal lesions without any other defect.

Huchard attributes a rôle to the pneumogastric.

For him the implication of this nerve proceeds from an alteration of the first part of the aorta or from pericarditis of the base. The periaortitis or the pericarditis of the base act by irritating the nerves of the neighbouring plexuses, and this irritation transmitted to the origin of the pneumogastric determines by a reflex way the vasomotor disorders and congestive oedema.

As Chemery says, it is to be too exclusive to limit to the periaortic or basal pericardial region the seat of the initial nervous disorder. Miecamp thinks likewise. The latter affirms that the causes capable of producing the sudden pulmonary congestion through nervous disorders are very numerous and varied. The connections of the pulmonary innervation with that of other organs of the economy and with the central nervous system would ensure the possibility of distant impressions or lesions ending in the last instance, by different routes, on the vasomotor apparatus of the lungs. He thinks that the starting-point of the reflex may be in a strong moral impression, in a cutaneous stimulus, or in a visceromucous impression; and he ranks the aortic and periaortic irritations among the reflexes of a visceromucous origin.

Following the report of Teissier (see "Mixed theories") at the Congress of Paris in 1900, Huchard observes that he

has been ranked by mistake among the supporters of the vasomotor theory exclusively. He says: "We have insisted on the mechanical element (pulmonary hypertension and sudden incompetence of the right ventricle), and on the nervous element often favoured by periaortitis and pericarditis of the base. It is also necessary to add a toxic element, which, certainly, plays a rôle in acting on the nerves and putting the vascular system in a state of œdema, according to the judicious expression of Renaut (of Lyons). There you have a very complex pathogenesis, and that, very happily, I shall add, for the necessary combination of all the pathogenic elements, explains and assures the relative infrequency of the accident in nephritis."

According to Le Calvé (1925) the essential cause of acute œdema of the lungs is a pulmonary vasodilatation which depends on various factors: a sudden rupture of equilibrium or rapid disturbance of pressures in the lungs (e.g., in decompression from thoracentesis), toxæmia (e.g., in intoxications and infectious diseases), or chloride retention (e.g., in renal disease). In his opinion a previous pulmonary or bronchial affection creating in the lungs a locus minoris resistentiæ is of prime importance in determining the localisation of the attack. The localisation in the lungs is also frequently due to a reflex irritation from the cardio-pulmonary nervous plexuses.

3. Toxic theory.

Most supporters of this theory attribute to disease of the kidneys the origin of the intoxication provoking the oedematous attack of the lungs. Jaccoud is struck with the frequency of acute pulmonary oedema in cases of albuminuria. He says: "Examine the urine, you will find in it the cause and explanation of the phenomenon." Fraentzel (1889) describes the coexistence of cardiac hypertrophy of renal origin, dyspnoea, and albuminous expectoration. But it is Bouveret who, in 1890, definitely establishes the relationship between acute oedema of the lungs and Bright's disease.

Vinay (1896) who has studied acute pulmonary oedema in pregnant women with cardiac disease attaches in its pathogenesis a more important rôle to the alterations of the kidneys than to the mechanical disorders of the intracardiac circulation. He considers albuminuria as the principal cause, whilst the heart-disease he regards only as an accessory factor. He even compares acute oedema of the lungs with eclampsia and the other convulsive phenomena which may occur in pregnancy: they all arise from identical cause, the different symptoms being due to the variability in the localisation of the poison.

Tonnel (1897) considers acute pulmonary oedema as the result of a simple vasomotor inhibition, attributable to

poisons not eliminated from the organism.

As we have already seen, Brouardel and Debove attribute a considerable part to renal intoxication in the production of the oedematous attack, whilst Professor Dieulafoy, who is the principal protagonist of this theory, holds that the kidneys alone are responsible for this accident. He states that it is one of the most serious complications of nephritis, acute or chronic, and that it is not necessary to invoke aortic lesions which most often do not exist. At the Congress of Paris in 1900 he reaffirmed that disorders of the kidneys alone, with the resulting toxæmia, may engender typical attacks of acute oedema of the lungs. This theory is supported by numerous clinical cases. At autopsies in young subjects who have died from acute pulmonary oedema he has noted that "the vessels are healthy, the heart normal, the aorta non-atheromatous, the kidney alone is affected with parenchymatous nephritis and the lungs are engorged with oedema." However, he does not state by what mechanism the intoxication produces the attack.

Merklen (1900) who favours the toxic theory, allows a certain rôle to the nervous system and to myocardial lesions in the production of pulmonary oedema. He states: "Pulmonary oedema may be a direct consequence of renal insufficiency and uræmia, of the same nature as cerebral oedema.... Acute pulmonary oedema of chronic interstitial

nephritis appears therefore to be a uraemic oedema....

It is possible and even probable that the pulmonary localisation of the oedematous congestion may be prepared by a certain degree of stasis of the lungs; but the toxæmia is the essential factor, without it being possible to say whether it acts directly on the pulmonary vessels or on their innervation. The accessory rôle, but real, of the nervous system is likewise well established by the evidence of occasional causes of the attack of pulmonary oedema: alimentary disorders, cold, emotions."

"Besides alterations of the kidney and aorta, it is necessary to attribute a big part to lesions of the myocardium, particularly of the left ventricle...." The latter may reveal its incompetence through overwork, a febrile attack, or a toxæmia being superadded.

Debove also incriminates the heart in the pathogenesis of acute oedema of the lungs. He declares: "Acute pulmonary oedema is frequently of renal origin; pulmonary oedemas also occur in pure cardiac cases; and, besides, in chronic nephritis the heart is always affected."

Renaut has shown that "certain conditions of habitual intoxication, most often auto-intoxication, place the vascular system in imminence of oedema."

Experimentally, acute pulmonary oedema has been produced

by means of various toxic agents. They have already been mentioned in the etiological section. As we have already seen, muscarine, according to Grossmann and von Basch, plays only a mechanical rôle, causing a "cramp of the heart."

Winkler (1898), studying the properties of amyl nitrite, has seen acute oedema of the lungs supervene in experimental animals. He introduced the amyl nitrite directly into the animal's trachea, and at the same time recorded the blood-pressures. The appearance of oedema was always accompanied by a rise of pressure in the left auricle and pulmonary artery with lowering of the general arterial pressure. These facts would seem to confirm the doctrine of Grossmann and von Basch.

Carrion and Hallion (1899), by intravenous injections of increasingly concentrated solutions of sodium chloride, have produced in animals acute oedema of the lungs similar to that described in man. They say that "the cells of the vascular endothelium are modified as the injection is made; at first they constitute a dialysing membrane; then, bathed by abnormal blood, altered in their nutrition, shrivelled up by the contact of an excessively concentrated plasma and stretched at the same time by the increased quantity of blood, they allow a considerable number of pores to be produced... and the dialysing membrane becomes

a filter allowing even red cells to pass through." Widal blames the increase of sodium chloride in the blood in the pathogenesis of acute pulmonary oedema. Owing to renal inefficiency there is retention of sodium chloride which accumulates excessively in the pulmonary parenchyma, where it attracts for its dilution water from the blood.

Teissier, utilising the oedema-producing properties of methyl salicylate noted by Chatin and Guinard, determines that acute pulmonary oedema may occur without an appreciable increase of pressure in the left auricle. But a considerable decrease of the carotid pressure is noted. Moreover, the pressure of the pulmonary artery appears to rise slightly as the oedema increases and death approaches. From these findings Teissier inclines to Welch's interpretation of the pathogenesis.

Teissier and Guinard (1901) have determined that the nervous system plays a definite rôle in the production of acute oedema of the lungs. In a dog, previously intoxicated with eserine and methyl salicylate, they could produce pulmonary oedema by excitation of the central end of the vago-sympathetic. Without such intoxication, a nervous lesion was not able to invoke pulmonary oedema. They conclude that previous intoxication is the primary factor in the production of acute oedema of the lungs.

Chanoz and Doyon confirm by experimental study that

under the influence of intoxication are produced in the blood-serum alterations which modify its molecular concentration and, in consequence, its osmotic power; also, changes occur in the walls of the vessels.

Martin H. Fischer in his notable work, "Oedema and Nephritis" (1921), maintains "that an abnormal production or accumulation of acids or conditions predisposing thereto exist in all states in which we encounter oedema." In such cases the capacity of the tissue colloids for holding water is increased above the normal. From experimental findings, he concludes that the production of pulmonary oedema may be explained by saying that an oedema results whenever the oxygen supply to the parenchyma of the lung is sufficiently interfered with. The lack of oxygen leads to an abnormal accumulation or production of acids in the tissues. Furthermore, Fischer finds that the most intense oedemas may be produced in the lungs removed from the body and in the entire absence of blood pressure changes.

Experimental researches have been carried out with adrenalin by Baylac of Toulouse (1900), Takamine (1901), Bouchard and Claude (1902), Josué (1905), and Hallion and Nepper (1910). It induces vascular hypertension, and can determine the death of animals by typical acute oedema of the lungs.

The above experimental work demonstrates the prominent rôle played by intoxication in the production of acute pulmonary oedema. It indicates as well the importance of hypertension, and the great part played by the nervous system. Alterations of the vessel-walls and of the chemical composition of the blood are also immensely important by favouring the serous transudation.

4. Mixed theories.

Fouineau in his thesis (1898) distinguishes acute pulmonary oedema as being of a toxic, reflex, or mechanical order. He regards the initial disorder as being always of a nervous nature, acting on a central or peripheral part of the pulmonary vasomotor apparatus. The mechanical element predominates in the pathogenesis of pulmonary oedema after thoracentesis, and in the sudden failure of the left ventricle in some cases of cardiovascular trouble. The toxic element, acting on the central nervous system, predominates in Bright's disease and in fevers. Whilst acute oedema of the lungs of reflex type is seen in aortic affections. He affirms that be the stimulus either mechanical or toxic, a nervous starting-point is universally admitted.

At the Congress of Paris in 1900, Masius and Teissier

expressed in their reports their opinions of the pathogenesis of acute pulmonary oedema. Masius distinguishes three forms of acute pulmonary oedema:

(1) an inflammatory oedema resulting from a reflex vasodilatation brought about by variable factors, followed by a direct injury of the vascular walls by microbes which normally inhabit the pulmonary parenchyma;

(2) an oedema of stasis which is met with in cardiac disorders (especially mitral stenosis), affections of vessels (aortitis and arteriosclerosis), and in diseases of the kidneys (sclerosis). The pathogenesis cannot be one and the same in all these cases, but a common etiological factor is found in them: it is the damage of the vessel - walls leading to a modification in their permeability. This only acts as a predisposing cause, whilst the immediate causal factor is to be sought for in the mechanical theory, or, in other cases, Huchard's vasomotor theory;

(3) a toxic oedema which is hardly known except in the experimental field.

To sum up, according to Masius, the modifications in the permeability of the vascular walls are a principal cause favouring oedema.

The most convincing example of oedema of the lungs due to an alteration in the lung tissue is that caused by the inhalation of gases such as chlorine and phosgene.

These gases cause oedema principally by altering the tissue, increasing the permeability of the vessels and injuring the endothelial lining of the alveoli.

In the Journal of Physiology (1926) there is a paper by R.K. Lambert and H. Gremels describing some experiments on the factors concerned in the production of pulmonary oedema. By means of the heart-lung preparation, the use of saline injections, and estimating the extent of effusion into the lung tissues by measuring the increase of electrical conductivity the course of the development of pulmonary oedema was followed. It was possible to rule out such factors as the rise of pulmonary pressure and the dissociation of the outputs of the ventricles, and affirm that the real cause of oedema lay in the changes which occur in the endothelium of the capillaries.

Sahli is also of the opinion that in many cases, especially in renal disease, pulmonary oedema seems to depend upon local changes in the vessel walls.

Teissier's conception of the pathogenesis attempts to reconcile the various interpretations that have been previously suggested to explain the phenomenon. He says that acute pulmonary oedema is the consequence of a complex pathogenic process in which infection or intoxication prepares the soil, nervous and mechanical disorders supervene

in the second place, to end in the serous extra-alveolar , and intra-alveolar inundation. The special soil necessary for the production of pulmonary oedema is the infection or intoxication which constitutes it. Moreover, this preponderating influence of infection or intoxication is well shown by the abundant evidence of medicamental toxic oedemas following e.g. the ingestion of potassium iodide, or iodoform, or the intravenous injections of artificial serum (Pozzi), and also by the results of experimentation.

Certain secondary lesions observed in some cases ought not to be neglected, as they present this essential character of affecting by preference the areas of distribution of the cardiac plexus or the autonomic ganglia. This fact indicates the intervention of a nervous element necessary for the production of acute pulmonary oedema.

As for information furnished by experimentation, beyond the definite evidence that intoxication is one of the important causes of acute oedema of the lungs, the noteworthy experiments of the German authors (Welch, Grossmann, von Basch, Winkler) would impose the conclusion that pulmonary oedema is determined, wholly or partly, by disorders of the circulatory pressure in the cardio-pulmonary apparatus. For them, therefore, the poison would only act by provoking disorders of pressure in the peripheral or intra-cardiac circulation.

The extreme importance attached by the German authors to the influence of mechanical disorders, is reconciled by Teissier with the two other etiological factors that clinical evidence and morbid anatomy have disclosed: the toxi-infectious element and the nervous factor. He shows the intervention of this triple etiological combination in numerous cases of oedema following influenza, scarlatina, acute articular rheumatism, and Bright's disease.

It is only the so-called primary acute oedemas (reflex oedemas and oedemas following thoracentesis) which cannot be explained by the same pathogenic conception. However, these oedemas are all related to subjects having a previous defect, an old infection or a constitutional dyscrasia, most often alcoholism. The constitutional trouble has determined alterations of the capillary walls or engendered a relative hydraemia, capable of favouring the osmotic phenomena of acute pulmonary oedema.

Acute oedema of the lungs is equally determined by a reflex peripheral excitation (e.g., violent emotions, powerful impression of iced drinks on the gastric mucosa) which has stimulated the reflex excitability of the pneumogastric and produced in the lungs the congestive vaso-dilatation which engenders oedema. In a similar way, after thoracentesis, the sudden decompression of the lung hampered

by adhesions, will excite the reflex activity of the pneumogastric to provoke an intense vasodilatation in both lungs.

Professor Teissier concludes: "Intoxication and infection remain the essential agents of the oedematous suffusion, the nervous and mechanical disorders intervening only to fix the pathological phenomenon on the pulmonary parenchyma." He also supposes that certain alterations of the blood and of the vascular walls may intervene in the mechanism of pulmonary oedema, and thus he confirms the opinion of Masius who, we have seen, made these alterations the most constant cause of acute oedema of the lungs.

The observation made by Huchard following the report of Teissier has already been mentioned when dealing with the vasomotor theory (page 41).

Chemery who in his thesis (1908) pays particular attention to the pathogenesis of acute pulmonary oedema, tries to establish in the production of the pulmonary attack the influence of this triple element: hypertension, retention of chlorides, and a nervous element. He states: "Acute oedema of the lung is caused by the sudden increase of tension in the domain of the pulmonary artery. This elevation of the vascular pressure in the lesser circulation is the result either of general hypertension or of hydraemia provoked by the retention of chlorides; or of pulmonary

vasomotor phenomena determined by an excitation of the central or peripheral nervous system; and most often of the combination of those different elements. Acute oedema appears therefore to require in the subject attacked the existence of a special soil. The individuals attacked would be subjects presenting arterial hypertension, in a state of chloride retention, in whom the intervention of a nervous element would lead to the sudden attack of oedematous congestion in the case of the lungs."

In support of his opinion, Chemery contends: "The majority of cases of acute oedema of the lungs supervene in aortitis or in subjects affected with sclerotic nephritis, all having hypertension."

"Sodium chloride, once retained in the tissues, combines with altered albumin and attracts to itself water which is necessary for its dilution (experiments of Achard and Loeper)." This occurs in oedema in general and "ought to apply to this localised oedema as is oedema of the lung. Acute pulmonary oedema, indeed, supervenes in the course of affections which are nearly always accompanied by retention of chlorides. In Bright's disease, retention of chlorides is the rule; and the cause of this retention is especially the kidneys.... In aortics, arteriosclerotics, it is rare that there do not exist a certain degree of renal insufficiency," and therefore retention of chlorides. "In

the febrile diseases and in the course of infections, particularly in pneumonia, rheumatism, typhoid fever.... Achard and Loeper have noted a great chloride retention."

The importance of sodium chloride is further seen from the experiments of Hallion and Carrion, and of Achard and Loeper, who succeeded in determining pulmonary oedema by intravenous saline injections in animals, with or without previous lesions of the kidneys.

"It may be affirmed, thanks to clinical and experimental facts, in man and animal, that sodium chloride is capable by itself of notably raising the pressure." (Widal, Lemierre and Javal).

For Chemery, hypertension and retention of chlorides go hand in hand; and when to them is superadded a nervous element, acute pulmonary oedema is determined. He writes: "The abruptness of the accidents, this enormous and sudden vasodilatation of the pulmonary arterial system attest the important part taken by the nervous system in the pathogenesis of pulmonary oedema... In all the cases of acute oedema of the lung there exists a nervous element of central or peripheral origin, of direct or reflex action, only intervening to fix on the pulmonary parenchyma the pathological phenomenon. The nervous system only provokes the sudden afflux of blood in the domain of the pulmonary artery."

5. Suprarenal theory.

This theory has been formulated by Josué in 1905.

It applies, according to him, only to certain cases of acute oedema of the lungs. He has observed that acute pulmonary oedema is an accident which threatens individuals with raised blood-pressure, and who have signs of aortic atheroma or chronic interstitial nephritis and often both these affections simultaneously. However, if the association of lesions of the kidneys and aorta constitutes an excellent condition for the production of acute oedema of the lungs, it is not indispensable, for aortics without any renal lesion, and nephritics without any aortic disease may be equally attacked.

"Arterial hypertension, atheroma, and acute oedema of the lung appear to be associated with functional disorders of the suprarenal capsules. These organs secrete and pour into the blood-stream a substance which Takamine has isolated: adrenalin, which possesses very intense hypertensive properties." (Josué). Chronic auto-intoxication by adrenalin, as Josué has shown, has a functional consequence: increase of arterial pressure, and an antomo-pathological consequence: arterial atheroma. According to Vaquez, adrenalin would produce hypertension in the organism either by its over-production or by its absence of elimination, and it does so by causing a peripheral vasoconstriction.

In 1901, Takamine, injecting large doses of adrenalin

intravenously in rabbits, determines their death by acute oedema of the lungs. Hallion and Nepper (1910) successfully repeat this experiment, and admit a direct toxic action on the capillary endothelium which is added to the mechanical action of the pulmonary sanguineous hypertension.

By the intravenous injection of relatively large quantities of adrenalin into a rabbit (10 minims of a 1 in 1,000 solution in a rabbit of 2,000 to 2,500 gms.) Josué has noted that these animals are nearly always seized with typical attacks of acute oedema of the lungs of which they often die. Bouchard and Claude (1902) have also observed this fact. The clinical picture is quite analogous to that observed in man, and autopsy reveals identical morbid lesions to those found in individuals who have died from this accident. Whilst the phenomenon is difficult to determine by other experimental means, it rarely fails to supervene with adrenalin.

"These experimental researches make us understand the pathogenic relationship uniting acute oedema of the lung to lesions of the aorta; it is not the propagation of the inflammation to the periaortic tissues rich in nervous plexuses that ought to be incriminated, as certain authors think. The acute accident and the chronic lesion are common affections from the same auto-intoxication; but the toxic substance is thrown into the circulation in massive

dose in the first case, and in often repeated little doses in the second."

"The experimental and clinical researches therefore demonstrate that acute œdema of the lung has often as a cause the exaggerated activity of the suprarenal capsules. We shall not go so far as to say that acute œdema always results from the same pathogenesis; the example of acute œdema following the tapping of a pleural effusion would suffice to show that that is not the case. But there exist certain acute œdemas of the lung which are the consequence of the same functional disorder: hyperactivity of the suprarenals. The hypersecretion of adrenalin by these glands causes arterial hypertension; when small quantities of adrenalin are poured into the blood during a long time, atheromatous lesions appear in the walls of the arteries; when considerable doses of poison are thrown into the circulation, acute œdema of the lung is seen to supervene." (Josué).

Summing up, according to Josué, hypertension, atheroma and acute œdema of the lungs may result from hypersecretion of the suprarenal capsules.

We have now to consider if there is any relationship between excess of adrenalin in the blood and sclerotic nephritis. Vaquez and Aubertin remark: "Lesions of the suprarenals appear to constitute a part of the anatomical

picture of atrophic nephritis, as hypertension constitutes a part of its clinical picture."

Josué has carefully studied the rôle of renal alterations in their connection with acute oedema of the lungs and atheroma. He says that in patients with arterial hypertension, but no other signs of chronic nephritis, the presence of excessive adrenalin in the blood causes the hypertension and is capable of producing acute pulmonary oedema; could it not also provoke a sclerotic nephritis? Thus would be explained the coincidence between atheroma and renal lesions in subjects attacked by acute oedema of the lungs.

Rabbits were subjected for a long time to repeated intravenous injections of adrenalin, and their kidneys remained remarkably normal. It does not seem therefore that chronic nephritis, seen in many patients who have attacks of acute pulmonary oedema, can be attributed to the hypersecretion of the suprarenals and the action of adrenalin.

It may therefore be asked if the renal lesions would not play the principal rôle in engendering acute pulmonary oedema. Two possibilities are to be considered: firstly, whether the renal lesions would render more easily and more serious the intoxication by adrenalin, or secondly, whether the alteration of the kidneys would be capable of determining the hyperactivity of the suprarenals as an antitoxic

reaction against the poisons retained in the organism owing to the renal incompetence.

Rabbits, in whom the ureters were tied, did not present a notably more marked sensitiveness to adrenalin than did the controls. Therefore renal impermeability does not predispose the organism to undergo more easily the pathogenic action of the suprarenal poison.

There remains the second hypothesis: could not nephritis determine acute pulmonary oedema by the intermediary of the suprarenal capsules, auto-intoxication of renal origin being the cause of their increased activity? The hypertension of Bright's disease would thus be explained at the same time.

The experimental researches of Dopter and Gouraud (1904) are favourable to this hypothesis. These authors, by ligature of the ureters in rabbits, have produced in the suprarenal glands definite histological signs of hyperactivity.

Josué concludes thus: "Nephritis would therefore be able to determine in the case of the suprarenal capsules functional disorders which have as a result the secretion of a too large quantity of adrenalin; now, intoxication by this substance is the cause of all the series of accidents of which acute oedema of the lung forms a part. From this

is understood how nephritis may exist alone in the origin of the acute respiratory accident."

However, the investigations, on which the conclusions of Josué are based, have not been sufficiently confirmed. In many autopsies on cases of pulmonary oedema hyperplasia of the cells of the suprarenal capsules, indicative of hyperactivity, has been looked for in vain.

Conclusions as to the pathogenesis of acute
pulmonary oedema.

Having considered the various theories that have been put forward to explain the mechanism of acute pulmonary oedema, and having studied the published cases and the experimental work done on this subject, I should adopt the following view of the pathogenesis:

Acute oedema of the lungs is ultimately due to an increase in the permeability of the walls of the pulmonary capillaries as a result of their dilatation. This may be produced through mechanical, toxic, or nervous factors. The permeability would be further increased by changes in the endothelium of the capillaries.

It is necessary to reconcile with this view of the pathogenesis the different conditions mentioned in the etiology of acute pulmonary oedema, and the various hypotheses that have been proposed to explain its produc-

tion.

The dilatation of the pulmonary capillaries may be brought about through mechanical obstruction within the circulation. This would be in accordance with the mechanical theories of the German authors, be the obstruction due to failure (Welch) or spasm (Grossmann) of the left ventricle. In these cases blood-pressure effects may be added to the changes in the permeability of the vessels and thus increase the oedema. In certain cases when the mechanical obstruction is very intense and especially if the pulmonary tissue possesses normally a low degree of resistance, the increased pressure itself may suffice to cause oedema of the lungs. The resistance of the pulmonary tissue could be diminished by (e.g.) a toxæmia or anaemia or previous lung disease.

The commonest cardiac lesion in cases of acute oedema of the lungs is aortic incompetence, a condition which involves the risk of sudden heart failure. The mechanical obstruction resulting within the circulation, provided that the right heart still keeps on beating, leads to engorgement and dilatation of the pulmonary capillaries with an outpouring of serum into the alveoli. The anaemia usually found associated with aortic regurgitation would increase the permeability of the pulmonary capillaries by damaging the nutrition of their walls.

Hypertension of the greater (or systemic) circulation causes congestion in the lesser (or pulmonary) circulation, for, as will be remembered, there are no vasoconstrictors in the pulmonary vessels. As Langdon Brown says: "Changes in the pulmonary circulation are passive and are controlled by the systemic circulation." The congestion in the pulmonary circulation would cause dilatation of the capillaries.

In chronic Bright's disease, the hypertension tends to cause congestion in the pulmonary circulation, and the toxins retained as a result of the renal incompetence may act directly as vasodilators and also damage the vascular endothelium.

Aortic disease could cause a reflex pulmonary vasodilatation, and this would conform with Huchard's hypothesis. If there be concomitant Bright's disease, the hypertension and toxæmia would increase the oedema as explained in the preceding paragraph.

Nervous stimuli, of central or peripheral origin, may cause dilatation of the pulmonary vessels, by direct or reflex action. Many examples have been mentioned in the etiology.

The dilatation of the pulmonary capillaries may be brought about through the action of irritating substances

(e.g., chlorine, phosgene), and in these cases the oedema may be increased through the destructive effect of these substances on the tissues.

A morbid blood-state or intoxication may cause both damage to the nutrition of the vascular walls and derangement of the nervous control. The localisation in the lungs would be determined as follows: the intoxication causes pulmonary hyperaemia through elimination of the poison, and by weakening the heart causes pulmonary stasis. Besides, the resistance of the lung-tissues may have been lessened by previous disease.

Adrenalin, incriminated by Josué as the cause of acute pulmonary oedema in certain cases, produces hypertension by constricting the peripheral blood-vessels. "The blood which is being squeezed out of the rest of the circulation will be forced into the pulmonary vessels, which are unable to protect themselves by adequate vaso-constriction," (Langdon Brown), and pulmonary engorgement with dilatation of the capillaries results. The engorgement may be very marked and the distension so great that rupture of badly supported capillaries may take place.

In infective conditions the toxins may act as vasodilators, but, undoubtedly, damage to the vascular walls by these toxins also plays a very important part. The localisation in the lungs will be determined by the cardiac weakness

and pulmonary hyperaemia resulting from the infection, and perhaps by present or past disease of the lungs themselves.

In pleuro-pulmonary affections like pleurisy, pneumonia, bronchitis, or phthisis, irritation of the pneumogastric may cause reflexly a pulmonary vasodilatation; also the vascular walls may be damaged by the infection, thus increasing their permeability.

Cases of acute pulmonary oedema following thoracentesis are correctly explained by the original theory of Pinault, the sudden decompression allowing a rapid afflux of blood into the pulmonary vessels with dilatation of the compressed capillaries. The permeability of the capillary walls is increased by their nutrition having been damaged by the long inaction of the capillary circulation owing to the compression, and also by a concomitant or previous affection.

In short, it may be said that in most cases it is evident that acute oedema of the lungs results from the sudden dilatation of the pulmonary capillaries, their walls being in a damaged state.

MORBID ANATOMY.

Renaut and his pupil Honnorat have admirably described the morbid anatomy of the lungs in acute pulmonary oedema, and the following account will be largely based on their description.

When the thorax is opened in an individual who has succumbed to an attack of acute oedema of the lungs, the latter are found to be voluminous and turgid, and are often marked on their surface by transverse depressions due to the ribs. The visceral pleura is moist and smooth, and is occasionally separated from the lung by a voluminous collection of yellowish oedematous fluid. The anterior borders of the lungs are distended by emphysema, crepitating softly when pressed between the fingers, and they cover the anterior surface of the pericardium to a more considerable extent than in the normal state. At the bases there is frequently a marked congestion; there the parenchyma is hard and spleen-like.

If the lung is incised there escapes from the cut surface a large quantity of serous fluid, often blood-stained, quite similar to that expectorated. A piece of this lung thrown into water does not sink to the bottom, but remains at the surface or floats "between two waters."

In the midst of the oedematous lung, the pulmonary parenchyma is found to have assumed a jelly-like consist-

ence, of a light violet colour, and traversed by a network of whitish tracts corresponding to pulmonary parenchyma which has been rendered bloodless by counter-pressure. The tissue is entirely deprived of air; all its fragments if thrown into water fall to the bottom. Pressed together between the fingers this lung-tissue expresses a violet-tinted rosy liquid, without any admixture of air, albuminous and not containing any fibrinogen, since it is not spontaneously coagulable. This lesion diminishes progressively towards its edge, in such a way as to pass gradually from the state of "compact oedema" into that of ordinary pulmonary oedema. Under the pleura the lobules are separated from each other by broad tracts of gelatinous appearance.

This compact oedema completely obstructs the alveoli. Developed suddenly like a lesion of nervous origin, it may be the starting-point of the generalisation of the oedematous process throughout the lung. We have here an "acute congestive pulmonary oedema," a morbid form sufficiently established and distinct from other forms of pulmonary oedema.

If a section made perpendicular to the surface of the pleura, be examined microscopically, the lesion is seen to consist throughout its extent of an enormous serous inundation of the pulmonary alveoli. They are filled and distended under pressure by an albuminous liquid deprived of fibrin,

like the fluid of an anasarca, and contain innumerable white corpuscles, practically without any admixture of red cells. At many points the inter-alveolar walls have given way. Thus are seen large spaces, filled with exudate, at the centre of which converge points corresponding to the remains of the ruptured alveolar walls.

The endothelium of the capillaries has everywhere disappeared. It has been carried away by the sudden irruption of the fluid and probably expelled by way of the bronchioles, for no traces of it are to be found on the walls. Here and there, however, are seen among the leucocytes, pressed against one another, and which inject so-to-speak the alveolar cavities, large undifferentiated cells, spherical and granular, for the most part containing grains of black pigment. They correspond to some endothelial cells of the respiratory surface, detached and reverted to an undifferentiated state. Within the limits of the lesion not a bubble of air is to be seen in the exudate: a phenomenon which shows well the sudden impermeability that supervenes following the inundation of the pulmonary parenchyma by the oedematous fluid.

The capillaries of the alveolar walls do not remain permeable; all are flattened by counter-pressure and absolutely devoid of blood. This exsanguineous state is

pushed to such a degree that where the alveolar walls are ruptured there is no evidence of the sanguineous leakage into the exudate. On the other hand, the large pulmonary and bronchial veins are engorged with red blood-corpuscles. Here and there a pulmonary venule has burst, distending with pure blood an adjacent alveolus at the periphery of the lobule.

Finally, under the pleura and between the sub-pleural lobules, the lymphatic spaces, entirely effaced in the normal state, have become developed and have reappeared very nearly as in the new-born. These are the broad tracts of gelatinous appearance, narrowed here and there in their course, and in the midst of which are distinguished the perilobular pulmonary veins, injected by red corpuscles. These bands are filled by an oedematous exudate absolutely similar to that which distends the alveoli, and containing so many white corpuscles that they touch one another.

Thus, this œdema, brought about through active congestion, consists of an enormous irruption of transudation and diapedesis. Nothing resists this movement: the alveolar endothelium is carried away; the functional circulation is sharply arrested by counter-pressure (as happens in the case of the skin in the anaemic centre of an urticarial wheal); the air-cells are invaded to such an extent by

the elements of lymph mixed with serum as to rupture at a multitude of points. At the same time the lymphatic channels, even the most effaced, are widely opened without being able to suffice for the elimination of the exuded liquid which injects and solidifies the lung more efficiently than even an interstitial injection of gelatine could do.

Such is acute congestive oedema. It is evident that this active oedema determines in the lung traumatic lesions of considerable mechanical importance. It is easy to understand that such lesions may be irreparable, and death may follow quickly when the process involves a considerable portion of the pulmonary parenchyma. But the generalised form of this congestive oedema is happily exceptional.

Other post-mortem findings are not constant. The brain is sometimes oedematous, and effusion into the arachnoid has been observed; but, on the other hand, it may be quite normal in appearance. Anaemia of the brain has not been reported.

The heart is frequently hypertrophied, especially the left ventricle. Occasionally signs of pericarditis have been noted. The aortic valve is often sclerosed and incompetent, and this is generally associated with mitral disease. The ascending aorta is commonly the seat of atheromatous changes; it is sometimes dilated.

The coronary arteries may also show patches of endarteritis. The arteries of the body generally may be hypertrophied and degenerated.

The kidneys most often are small in size, with a granular surface and adherent capsules; their cortex is narrowed and may contain cysts. Occasionally the kidneys are enlarged with changes most marked in the tubules. The suprarenal capsules have been reported by some observers as showing histological changes indicative of hyperactivity, but, on the other hand, these are more frequently absent.

The liver may be enlarged and, on section, present a "nutmeg" appearance. The spleen also may be enlarged and congested; occasionally it has been noted to be very soft in consistence.

The pancreas and other endocrine glands usually appear normal. Occasionally no other lesion besides that of the lungs is observed at autopsy, all the other organs appearing quite healthy.

S Y M P T O M A T O L O G Y.

Acute pulmonary oedema may be fulminating, hyperacute or acute with characteristic expectoration, or bronchoplegic without expectoration. In the really fulminating type, the onset is very sudden and the termination extremely rapid, in a few minutes, as in the following case related by de la Harpe: a patient awoke with a start during the night; he jumped out of bed, crossed the room, sat down on a chair breathing with difficulty; a few moments later he died, a quantity of white froth coming abundantly from his mouth and nostrils.

In the hyperacute or acute cases which are much more frequently observed, the following phenomena are noted: suddenly, most often without any appreciable premonitory symptoms, an individual apparently in satisfactory health, is seized by a distressing sensation of thoracic oppression and great difficulty of respiration. The attack occurs most often by night awakening the patient from his sleep, and the acute dyspnoea obliges him to sit up in bed as in an asthmatic paroxysm. Along with the intense dyspnoea there supervenes an incessant, jerky cough, giving no rest, and the patient brings up with very little effort a copious watery (serous) and frothy expectoration of albuminous nature. The thin fluid sometimes seems to pour out of the mouth and

nose. Two or three pints may be expelled in a few hours. It is often rose-coloured, indicating the addition of a congestive element to the oedematous exudation. Innumerable fine crepitant râles are heard over the lungs. They are fine, distinct, moist râles accompanying inspiration, bursting with a sharply defined or metallic noise at the same time conserving their humid character. By their abundance they give the impression of a violent agitation in the lungs. When the fluid reaches the bronchi, the râles become coarser and more bubbling.

With this abundance of râles which rapidly invade the entire chest, one would expect to find on percussion dulness or, at least, a diminution of resonance. On the contrary, there is increased resonance on percussing the chest. This phenomenon of "paradoxical percussion" is due, as well as the obscurity of the respiratory murmur which is often noted, to the production of an acute (compensatory) emphysema. The percussion note may become dull later.

During this violent disturbance, the temperature is not raised, often it is even lowered, although Bouveret has seen it rise to 102° and even 104°F. The extremities become cold, the face pale and anxious-looking, the lips and nails bluish, and the patient becomes bathed in a cold sweat. The sphincters may become relaxed, and an involuntary evacuation of the bladder and less frequently of the rectum

may occur. The patient may die rapidly from suffocation. Sometimes he becomes unconscious and dies in a few hours. But death is not the constant termination of acute or even hyperacute pulmonary oedema. More often, after lasting a few hours the attack subsides slowly. The patient may recover from several attacks recurring at variable intervals; he will do that especially if an active and prompt therapy is carried out.

The pulse is accelerated. Its tension at the beginning of the attack does not fall, those who have been able to be present from the very commencement of the accident affirming that the pulse remains strong and sustained. These characters are indicative of the existence of arterial hypertension. "All of a sudden, most often in the course or towards the end of the crisis, there is noted a considerable fall of the arterial tension which is explained by the acute cardiac failure, which supervenes sharply." (Huchard). The appearance of this arterial hypotension may be accompanied by a rapid oedema of the lower limbs.

According to Huchard, two periods can be assigned to the course of acute oedema: the first characterised by dyspnoea, abundant expectoration, and maintenance of arterial hypertension; the second by acute cardiac failure, scanty or absent expectoration from bronchoplegia, and decrease of arterial tension. In some cases (bronchoplegic

form) the first period seems to be absent, the attack from the very beginning assuming the features of the second stage.

Blood-pressure readings (Amblard, 1911 and 1920) indicate that before the onset of the paroxysm there occurs an increase of the already high arterial tension, both the systolic and diastolic pressure showing a rise of several degrees. Then suddenly the blood-pressure drops markedly. After the attack it rises again, but does not return to its former figure.

The albuminous expectoration so peculiar to this accident is a rosy or pinkish watery fluid accompanied by a large quantity of froth. It forms in the containing vessel three successive layers: on the surface a quantity of pinkish froth, persistent and often very abundant; then a layer of liquid which may have different appearances, sometimes pinkish or yellowish, and transparent when it is abundant, at other times turbid from its admixture with mucus. The last layer forms a deposit at the bottom of the vessel, and chiefly consists of epithelial cells, white corpuscles, and a small number of red cells.

This liquid treated with nitric acid gives an abundant precipitate of albumin. The quantity of fluid expelled is very variable. None in the bronchoplegic form, it varies between a few drachms to a pint, and may even be up to two or three pints or more when the attack is prolonged. The

characteristic thing about this expectoration is the presence of albumin. Indeed, in simple bronchorrhœa, the liquid expectorated is sometimes very abundant but does not give a precipitate with nitric acid. Moreover, it is interesting to point out a notable difference between this liquid of acute pulmonary oedema, and, for example, that of oedema of the limbs. In the latter case the oedematous fluid contains only trivial quantities of albumin, whilst the expectoration of oedema of the lungs is principally distinguished by the large amount of albumin which it contains. This fact is easily explained if the peculiar structure of the pulmonary tissue is considered. The inter-alveolar capillaries are separated from the cavities of the air-cells only by a thin endothelial layer, which forms the walls of the capillaries and the lining of the alveoli. In pulmonary oedema these vessels, distended to their utmost, may rupture at many points, and let transude into the alveoli the sanguineous serum whose richness in albumin accounts for the large quantity of albumin contained in the expectoration of acute oedema of the lungs.

Furthermore, the red blood-cells which escape from the pulmonary capillaries into the alveoli account for the reddish colour of the expectoration. This colour is most conspicuous in very acute cases, where the frothy fluid is often expectorated in extraordinary quantities.

In the bronchoplegic form expectoration cannot be brought up, and the patient dies from respiratory obstruction by the intra-alveolar and bronchial exudate.

The duration of an attack is a few minutes in the fulminating œdemas, a few hours, the night, or, in the less rapid forms, two, three, or, even four days.

Hyperacute œdema of the lungs in Bright's disease has been masterfully studied by Professor Dieulafoy, and it is worth while to include here his description of the symptoms. He writes: "The first point of importance is the suddenness of this œdema. Reference to the cases which I have collected shows that hyperacute œdema does not, as a rule, result from bronchitis or some pulmonary condition which has gradually become more severe. The onset is nearly always sudden and unexpected; the unforeseen accident comes on by day or by night...."

However, "in some cases hyperacute œdema is preceded by such prodromata as cough, dyspnoea, or râles. In such a case it seems that the soil is prepared..... It is, however, an exception. Hyperacute œdema of the lung in Bright's disease is hardly ever the result of pre-existing broncho-pulmonary lesions. It bursts out suddenly, like an attack of asthma, although some hours previously no suspicion has been entertained."

"A second point is that hyperacute œdema of the lung

is very often an isolated result of Bright's disease, for, paradoxical as this may appear, it is hardly ever associated with the severe manifestations of uraemia, or with the marked œdema of Bright's disease. Without knowledge on this point, it would appear that hyperacute œdema ought to supervene in the patient who is suffering from anasarca. This may be so, especially in acute nephritis.... This, however, is a rare occurrence. Nearly all the cases which I have collected show that the mischief appeared as an isolated accident in the course of latent nephritis."

It cannot be said, however, that this œdema attacks persons in perfect health. Close examination will show that they are more or less tainted with Brightism. If the 'minor complications of Brightism' be carefully looked for, the evolution of an insidious nephritis may be constructed. A group of symptoms, such as the sensation of dead fingers, cryæsthesia, auditory troubles, cramps in the calves, pollakiuria, itching, electric shocks, and epistaxis will be found in their past history. We shall see that they were not exempt from such a trace of œdema as puffiness of the eyelids and of the malleoli. We shall find that their arterial tension is high, their temporal arteries are tortuous, the second sound accentuated, and a gallop rhythm is present. We shall find that these patients were subject to headache, which they styled

migraine; to suffocation, which they took for asthma; and to colds, which they regarded as due to ordinary bronchitis. If their urine is analysed, albumin will be found; on testing the toxicity, it will be low. We shall find, in short, by an attentive and searching examination of this condition - which for a long time I have named Brightism - that the patient whose health appears to have been fairly good has in reality been suffering from mischief in his kidneys; the urinary depuration has been affected, and he has been exposed to the risk of complications which gave more or less warning."

"After this digression as to the onset, let us resume our clinical analysis of the symptoms. The attack commences with a tickling in the larynx, jerky cough, and distress, which reaches its limit in some minutes, or perhaps some hours. These symptoms are due to sudden blocking of the pulmonary alveoli by sero-albuminous fluid. As a rule, the inundation begins in the bases, and may affect the whole of the lungs more or less rapidly. As the blood-serum transudes into the alveoli and the bronchioles under pressure, the patient is at once seized with fits of coughing, and then brings up the characteristic fluid, which is frothy and rose-coloured. Some patients may bring up as much as two or three pints in a few hours, or even more. It may be that the bronchi have not the power of expelling the

fluid which has thus accumulated, and the asphyxia varies in indirect ratio to the amount of expectoration."

"As soon as dyspnoea appears, innumerable fine subcrepitant and sibilant râles can be heard over both sides of the chest, testifying to the inundation of the alveoli and of the bronchioles. According to the rapidity and the extension of this inundation, the râles invade the whole, or nearly the whole, of the lungs."

"In proportion as the inundation increases, and if the expectoration is ever so slightly insufficient, dyspnoea increases rapidly; the patient is pale, alarmed, and conscious of his extreme danger; the pulse is small and quick, the lips are bluish, the nails livid, the limbs cold; and the struggle may in a few moments end in death (fulminating form), in a day (rapid form), or in three or four days (slow form)."

"In some patients the condition is not absolutely perilous, so long as the sufferer can empty the inundated lungs. Paresis of the expulsive muscles may supervene, expectoration may be quite absent, and death from asphyxia occurs at short notice. In favourable cases the fluid is coughed up as soon as it forms, the inundation is arrested in time, and after a duration which varies from some hours to some days, the dyspnoea improves, the râles diminish, and the patient wins the struggle. We must,

however, not be too sanguine, for the danger, though averted for a moment, may some hours or some days later recur and prove fatal. Sometimes convalescence requires several days; at other times recovery comes on suddenly, and the patient is able to resume work on the next day."

"Hyperacute œdema of the lung in Bright's disease is made worse by the fact that not only may be the patient succumb to an attack in a few hours, but that, although his lungs have recovered, he remains liable to fresh attacks. Several patients have had two or three repetitions of hyperacute œdema, months or years apart."

Summing up, the usual type of case of acute pulmonary œdema manifests itself by a very definite and characteristic syndrome: sudden onset of intense dyspnoea; sense of great oppression in the chest; cyanosis or pallor; incessant, jerky cough; abundant expectoration of frothy, watery, pink fluid of albuminous nature; moist râles all over the lungs, with increased resonance and diminished respiratory murmur; pulse accelerated and, at first, forcible, later feeble; absence of fever. These features combine to give this accident such a characteristic aspect, that it is difficult to confuse it with any other cardiac or pulmonary affection in spite of the analogy of certain symptoms.

D I A G N O S I S.

An accurate diagnosis is very important as a favourable issue depends so much on correct and prompt therapy. The diagnosis of acute pulmonary œdema may be established on the severe and increasing dyspnœa, the characteristic expectoration, the numerous crepitant râles on auscultation with increased resonance on percussion, and the absence of, or only slight, pyrexia. The strongly positive albumin-reaction of the expectoration is considered by Bezançon and de Jong as a reliable sign, very easy to verify. It may be utilised after having eliminated the other conditions where it occurs, as tuberculosis and inflammatory affections of the lungs. Acute œdema of the lungs has to be differentiated from various affections where dyspnœa is a prominent feature.

Pulmonary embolism may by its sudden onset, violent dyspnœa, and often fatal termination give rise to confusion. However, its physical signs are quite different from those of acute pulmonary œdema. At the beginning in embolism the signs are nil: no râles in the chest, normal and never exaggerated resonance. Later, however, traces of congestion and œdema may be found; but this œdema is localised to a circumscribed area of the lung in the region of the blocked artery. There is no abundant albuminous expectoration.

Moreover, a thrombosed focus is present, e.g., phlegmasia alba dolens, varicose phlebitis. However, one must guard against error. An attack of severe dyspnoea may appear in a case of rheumatic or typhoid fever presenting a phlegmasia alba dolens. Before diagnosing definitely pulmonary embolism, the possibility of acute oedema should be considered, for the latter may also occur in those fevers.

Bronchial asthma may be confused with acute pulmonary oedema. The latter often surprises the patient at night, and, moreover, like asthma appears suddenly without preceding phenomena. The term "asthmatic" is frequently loosely applied to those who have attacks of nocturnal dyspnoea. However, the error is easy to avoid. In an attack of asthma the frequency of respiration is not increased; expiration is prolonged and wheezing. Auscultation reveals the presence of sibilant and sonorous rhonchi, indicative of a concomitant chronic bronchitis. Finally, the expectoration, which occurs at the end of the attack, consists of a viscid, pearly mucus, containing Curschmann's spirals and Charcot-Leyden crystals. There is nothing like that in acute oedema of the lungs where the respiration is short and quick, the chest full of fine crepitant râles, and from the beginning of the attack an abundant expectoration of a very frothy, watery, albuminous

liquid.

In acute phthisis, which sometimes occurs in the form of repeated bronchitic attacks accompanied by severe dyspnoea, the expectoration is neither so abundant nor frothy as in acute pulmonary oedema; the auscultatory signs are different, and bacteriological examination of the sputum may serve to remove any doubts.

Oedema of the lungs of insidious onset, results from conditions which give rise to passive congestion, as failure of compensation in (e.g.) mitral disease, or great cardiac weakness as in typhus, enteric, and other prolonged and severe debilitating conditions; and in addition, it is promoted by morbid blood-states which tend to be associated with dropsy elsewhere - e.g. Bright's disease and anaemia. It is bilateral as is acute pulmonary oedema, but it involves, as a rule, the dependent parts of the lungs, and is therefore most commonly localised at the bases. It rarely occurs as an isolated phenomenon. Usually there is also present oedema of the lower limbs, hepatic congestion, occasionally ascites or pleural effusion. Even if it occurs alone, the differential diagnosis is easy. It takes a considerable time to invade a large extent of the lungs; there is no albuminous expectoration; the râles are coarser, and the percussion-note is not exaggerated because there is no acute emphysema. Pulmonary oedema of insidious onset is usually

terminal and indicates that the patient is dying, whilst acute œdema of the lungs may suddenly appear in a person in apparently good health and actually be the cause of death.

In hydrothorax, percussion yields dulness the limits of which vary with the posture the patient assumes. Dropsy is likely to be present elsewhere.

In broncho-pneumonia the dyspnoea is severe and the patient is frequently in a state of collapse. But the condition is often secondary, the temperature is raised, and there are fine moist râles and patches of dulness with bronchial breathing. Expectoration, when present, is thick and purulent.

Oedema of the glottis is nearly always preceded by dysphagia. The dyspnoea differs from that in acute pulmonary œdema. The breathing is noisy and laboured, inspiration requiring much effort and being accompanied by stridor. At the same time sucking-in or depression of the suprasternal and epigastric hollows occurs. On auscultation, only the echo of the laryngeal whistling is audible. The diagnosis may be confirmed by the laryngoscope, or the greatly swollen epiglottis may be felt by the finger.

Laryngeal diphtheria is an affection of childhood, whilst acute pulmonary œdema occurs rather in adults or the aged. In the former the temperature is raised, but is not,

as a rule, high. "The voice is hoarse, and is eventually lost, and breathing is both difficult and noisy. In inspiration, if the obstruction is severe, the larynx is drawn downwards towards the sternum; and the epigastrium, the suprasternal and supraclavicular regions, and the lower ribs are drawn in. The cough acquires the peculiar harsh, ringing quality described as 'croupy', and dreaded by mothers." (Monro). Auscultation of the chest, when there is no pulmonary complication, reveals nothing but the echo of the laryngeal whistling.

Acute pulmonary oedema may be confused with syncope, especially when the latter is of the fulminating type with white asphyxia. It may also be confounded with an attack of angina pectoris. These would be serious errors, for if one mistook the attack of acute oedema for one of syncope or of angina pectoris, one would not carry out immediately a copious venesection which may save a patient from death. The condition is not syncope since the heart continues to beat strongly. It is not angina pectoris, the patient being chiefly dyspnoeic without the intense pain characteristic of angina pectoris. However, patients with aortic disease may have at the same time angina pectoris through coronary arteritis, or toxi-alimentary dyspnoea through renal insufficiency, and attacks of acute

oedema of the lungs.

Mitral stenosis, which is observed much more commonly in females than in males, is often associated with hysteria. Individuals affected with aortic disease are frequently also neurasthenic. In these two types of cases attacks of suffocation ("cardiac asthma") may occur quite independent of pulmonary manifestations.

Acute pulmonary oedema has also to be differentiated from the various types of uraemic dyspnoea. With the purely dyspnoeic form and with the Cheyne-Stokes respiratory type, no confusion is possible. In the first form there are no stethoscopic signs; it is an essentially toxic dyspnoea. In the second type the respiration is characteristic; it is not increased in rate; it is simply modified in rhythm. In a third variety, the asthmatic type, the dyspnoea occurs in paroxysms, surprising the patient in his sleep. Like "cardiac asthma," the accident is marked by distress or a sense of oppression in the chest and rapid breathing. During the attack one finds, not usually at the beginning but rather later, sibilant and sonorous rhonchi. The expectoration consists of mucous, muco-purulent or frothy sputum, which gives a negative or very feebly positive albumin-reaction.

In those cases where the diagnosis is occasionally difficult and where doubt exists, time must not be lost,

and a venesection should be done. As Houdé writes:

"Every time when hesitation occurs, we believe that one must behave as if it were a congestion, because the means employed against the latter would not be harmful if one were wrong....., whilst the reverse would not be true."

A prompt decision and energetic action are imperative.

A large vein must be immediately opened without fear of syncope in spite of the pale aspect of the patient, for it is not syncope which threatens him but asphyxia. (Huchard).

P R O G N O S I S.

This is grave. It varies directly with the rapidity of the phenomena. In the fulminating form death supervenes from asphyxia before the serous inundation of the lungs can be expectorated. This fulminating type is, according to Brouardel, a frequent cause of sudden death. The hyper-acute form is frequently also fatal, since the intensity of the symptoms does not allow sufficient time for applying the appropriate therapy. The prognosis is serious in the acute cases, where the life of the patient is in the hands of the physician. In these forms where the progress is less rapid, death may not supervene for a few hours or even three or four days. Favourable cases, where the patient triumphs over the accident, also occur, in some instances the resolution of the œdematous irruption taking place spontaneously.

After the attack, recovery may supervene quickly. The patient, who the preceding night was moribund, may be able to resume his work the next day, the only remains of the accident being a dreadful horror of its recurrence. However, convalescence sometimes requires several days.

The ultimate prognosis depends on the nature of the affection which the attack of acute pulmonary œdema has

complicated. The accident may recur at short or prolonged intervals. The happy termination of a first attack therefore ought not to inspire too much confidence, because each return of the accident places again the life of the patient in danger.

During the paroxysm the situation in certain patients is not altogether perilous whilst the expectoration is sufficiently abundant to empty the inundated lungs. Should this fail (bronchoplegic form), death from asphyxia would follow rapidly. A falling blood-pressure and a pulse becoming quicker and smaller are of bad omen.

After the attack one may draw certain conclusions for the future from the value of the blood-pressure. The outlook is favourable when the arterial tension rises rapidly to the neighbourhood of its previous level; it is serious if the pressure undergoes a very considerable fall, when a recurrence of the accident with an unfavourable issue has to be feared; a less marked decrease, but the tension being maintained below its original level, results from a dilatation of the left ventricle which succeeds the pulmonary oedema and is capable of leading to death in a few weeks.

The attack sometimes remains solitary, e.g., following thoracentesis and in infectious diseases. On the other hand, acute pulmonary oedema in Bright's disease usually reappears after a variable interval - in a few hours, the

following night, or after some days. In more favourable cases there are some months or several years of respite. After a few attacks acute oedema of the lungs may be replaced by another kind of toxic or neurotic manifestation, such as pericarditis, angina pectoris, or cerebral uraemia.

As already indicated, the gravity of acute pulmonary oedema depends greatly on the cause which has provoked it. An active therapeutic intervention, however, may appreciably reduce the seriousness of the prognosis.

T R E A T M E N T.

Quick and decisive action is essential. The embarrassed lungs must be disencumbered, and after the crisis measures ought to be taken to prevent a recurrence.

To combat the attack "the urgent indication is bleeding. In spite of the coldness of the patient and the threatening collapse, which would at first appear as contra-indications, there must be no delay, and, without losing an instant, 10 to 15 ounces of blood must be withdrawn. The marvellous results of bleeding must have been seen to make its importance clear. I do not exaggerate in saying that it produces in the patient a visible change." (Dieulafoy). The venesection does not empty the alveoli filled by the exudate, nor does it reopen at once the capillary network which has been compressed and rendered bloodless. It prevents the progressive and very often rapid invasion of healthy portions of the pulmonary tissues, for, left to itself, the œdema ascends implacably, one might say actually under the ear of the observer, from the base to the apex. Blood-letting operates by counteraction; it suddenly substitutes a new circulatory régime for that commanded by the pathogenic process. Furthermore, under its influence the heart, relieved, recovers its energy, and with the evacuated blood a notable quantity of toxins is eliminated.

The major indication for venesection is the dyspnoea, and the quantity of blood withdrawn ought to be proportional not to the supposed resistance of the patient, but to the intensity of the dyspnoea, to the gravity of the threatening asphyxia. 10, 20, or even 30 ounces can be withdrawn. For the phlebotomy, the largest superficial vein at the bend of the elbow should be used. Some prefer the external jugular vein.

As adjuvants to blood-letting, or alone in cases where it is not feasible (e.g., opposition of relatives) and in less urgent attacks, wet cups may be applied over the chest, hepatic region and kidneys, or, better still, two dozen leeches. Dry cups and sinapisms over the trunk and lower limbs (inner surface of thighs and calves) are also useful. They are to be specially recommended over the seat of oppression in the chest. A hot-water bottle should be applied to the feet, or they may be placed in a hot mustard bath.

Applying a blister to the chest must be avoided, for the patient often has Bright's disease, and the action of cantharides would only aggravate the situation.

The cold bath, an excellent decongestive agent, may be employed in certain cases, especially in acute oedema in febrile conditions with hyperthermia (Fouineau).

According to Grossmann atropine, whose action is

antagonistic to that of muscarine which produces in animals acute pulmonary oedema, ought to be employed. The therapeutic utility of this physiological antagonism is denied by the French observers. However, atropine should be given, as it may be useful in lessening the pulmonary secretion and in stimulating the respiratory centre. Grain $\frac{1}{50}$ should be administered hypodermically. A good plan is to give the atropine at once and follow it immediately with venesection to about a pint.

A paralytic state of the bronchi and diaphragm may supervene in the later period of the oedematous attack, and is one of the most powerful causes of a fatal termination. To combat this, recourse may be had to a hypodermic injection of strychnine, gr. $\frac{1}{30}$.

Rapid, energetic, cardiac stimulants are often indicated. The action of digitalis or of sparteine is too slow. It is better to give subcutaneous injections of caffeine sodium salicylate, gr. i, camphor (gr. ii) in oil, or ether, m. 15 to 30. These may be repeated every half hour or hour. It should not be forgotten, however, that the kidneys are often inactive, and therefore caffeine is to be used with caution. That is why it is recommended to begin with a dose of 1 grain, and this may be repeated several times in the hours which follow. If the patient can swallow, he may be given by mouth a drachm of spiritus aetheris,

Hoffmann's anodyne, or aromatic spirit of ammonia in water.

Adrenalin is contra-indicated for reasons already mentioned. By causing a peripheral vasoconstriction, it would increase the vascular engorgement in the lungs owing to the absence of vasoconstrictors in the pulmonary vessels. The same objection cannot be made to pituitrin, as it lowers the pressure in the pulmonary circulation. A subcutaneous or intramuscular injection of 1 c.c. may be given. It has been highly recommended.

For the acute respiratory distress one thinks of morphia; but caution is necessary. French opinion is altogether against its employment, except in œdema following thoracentesis where it should be given at once. Though it is theoretically contraindicated, practically it is very valuable. It may be of great service especially when the attack is subsiding. Grain $\frac{1}{4}$ may then be given hypodermically to calm the panic-stricken patient. Along with it should be administered gr. $\frac{1}{100}$ atropine. I think that morphine may safely be given when the pulse is strong and the patient is expectorating freely, but it is dangerous if there is evidence of bronchoplegia. The following advice in Osler's Principles and Practice of Medicine (Mc Rae) is sound. "Patients who have repeated attacks should be warned against over-exertion and with

the first symptoms of an attack should be given ammonia, and morphia and atropine hypodermically." Inhalations of chloroform have also proved beneficial in the paroxysm, but its use is not without danger. Oxygen in large doses may also render some service in allaying the dyspnœa. Carbonic acid, administered per rectum, has given Teissier a remarkable success. It stimulates the respiratory centre, increasing the respiratory effort.

The pathogenic idea of a spasm of the left ventricle made one hope of overcoming this contraction and re-establishing the equilibrium in the pulmonary circulation by producing an intense peripheral vasodilatation by means of amyl nitrite. But the latter is itself capable of provoking acute pulmonary oedema, and here "the medication becomes the accomplice of the disease" (Huchard). To overcome this disadvantage, Winkler proposed the oxycarbonated amyl nitrite, which does not determine oedema of the lungs. In addition to its peripheral vasodilating action, it increases the contractile power of the heart. Professor Teissier hoped that excellent results would be obtained from its use. Huchard advises the use of trinitrin (nitroglycerin), whose vasodilating action is well known. If hypertension is present nitroglycerin, gr. $\frac{1}{100}$ (a hypodermic tablet), is to be placed under the tongue and repeated until an effect is produced. (McRae).

Ergot and apomorphine have been employed, but are to be avoided. The former by causing a general vasoconstriction would increase the pulmonary tension, like adrenalin does, whilst emetics may be dangerous on account of the collapse which is apt to come on after them.

Strophanthus has been found useful. When the condition is associated with auricular fibrillation, an intravenous injection of strophanthin, $\frac{1}{4}$ mg., would be indicated. It should only be given if the patient has not been having recently any digitalis, and should follow the venesection.

Ipecacuanha has been advised in cases where the oedema supervenes in individuals affected with diffuse bronchitis.

With a view to arresting the supposed spasmodic contraction of the left heart, galvanisation of the vagus has been recommended.

Heroic measures have also been employed in desperate cases. Sahli has practised tracheotomy, and introduced a soft rubber tube through the tracheal opening into the encumbered bronchi, and aspirated by this means the accumulated fluid obstructing the respiratory area. Another procedure which has been done is tapping of the right auricle. This has been carried out with the aid of a fine needle introduced through the fourth right intercostal space, with the object of rapidly reducing the congestion of the extremely distended right heart. According to Chemery, this method

has been successfully accomplished on several occasions.

The occurrence of acute pulmonary œdema in a pregnant woman requires special consideration. After having combatted the urgent symptoms by the means already indicated, it will be necessary to provoke evacuation of the uterus by the most rapid means (e.g., Champetier de Ribes' bag). Intervention must be quick, because œdema of the lungs is a more formidable complication than eclampsia. When the pregnancy has reached the seventh month, the possibility of saving the child's life has also to be considered. Its chance of salvation will be the greater the less long it will have respired an insufficiently aërated blood and the less it will have participated in the maternal asphyxia (Vinay).

The acute symptoms over, the patient may be allowed diluted milk, alkaline waters, weak tea, and imperial drink. At the same time he should have 10 to 15 grains of theobromine sodium salicylate (diuretin) three times a day. After that one recommends the dietary and medication suitable to the affection which has been the cause of the œdematous attack, e.g., digitalis in cardiac cases, sodium salicylate in rheumatics.

Preventive measures have now to be considered. Dieulafoy writes: "Danger once averted, do not lose sight of the patient. Absolute milk diet must be prescribed,

chloride of soda, which favours œdema (Widal) avoided, and the urinary secretion closely watched. In short he must be treated as a case of Bright's disease, and be advised to avoid with the greatest care every cause of overwork and chill." The absolute milk regimen may after a time be replaced by a lacto-vegetarian diet. If the urinary output is insufficient, diuretin ought to be prescribed, 10 to 15 grains three times a day.

Subjects attacked by acute pulmonary œdema are often arteriosclerotic. One must be careful in prescribing for them potassium iodide, for Huchard has seen a fatal attack following the administration of 30 grains of this drug. One will have to be content to treat such cases menaced by this complication by attention to the dietary and mode of life, and by regulation of the bowels.

In the numerous cases where there exists aortitis or peri-aortitis, counterirritation should be applied to the sternocostal region in the shape of frequent painting with iodine, and even application of the actual cautery. Cupping of this region has also been recommended.

Acute pulmonary œdema following thoracentesis, according to Dieulafoy often results from the too rapid withdrawal of a too large quantity of fluid. Hence the prophylactic requirements are obvious. An aspirating needle of narrow bore should be employed, and the quantity of liquid with-

drawn at a time should not exceed one litre ($1\frac{3}{4}$ pints).

To recapitulate, the treatment of choice consists in an immediate hypodermic injection of atropine, gr. $\frac{1}{50}$, followed at once by venesection to about 20 ounces. Other valuable therapeutic agents are pituitrin, 1 c.c., administered subcutaneously or intramuscularly, and strychnine, gr. $\frac{1}{30}$, injected hypodermically. If required, rapid cardiac stimulants like caffeine sodium salicylate, gr. i, camphor (gr. ii) in oil, or ether, m. 15 to 30, may be given every half hour or hour by subcutaneous injection. When the paroxysm shows signs of abating morphine, gr. $\frac{1}{4}$, with atropine, gr. $\frac{1}{100}$, should be administered hypodermically.

Of subsidiary utility are wet or dry cups, especially over the entire posterior pulmonary area, a large mustard-plaster applied to the seat of oppression in the chest, and a hot-water bottle to the feet. Oxygen may also be helpful.

To prevent a recurrence, the chief requirements are milk diet, avoidance of sodium chloride, and careful watching of the urinary output.

C O N C L U S I O N S.

1. Acute suffocative pulmonary oedema is a well-defined morbid syndrome constituted by a sudden transudation of serous fluid from the lung-capillaries into the pulmonary interstitial tissue, alveoli and bronchioles.
2. The condition is not uncommon, but has been inadequately recognised in this country.
3. It occurs most commonly in chronic cardiovascular and renal affections, associated with hypertension. It has also been observed in nervous disorders, intoxications and morbid blood-states, infectious diseases, pulmonary affections, following thoracentesis, and occasionally it has occurred idiopathically.
4. The attack is ultimately due to an increased permeability of the walls of the pulmonary capillaries as a result of their dilatation, which is produced through mechanical, toxic, or nervous factors. The increased permeability is usually intensified by the damaged state of the vascular endothelium.
5. The morbid anatomy confirms the seriousness of the condition, both the pulmonary circulation and aëration being so gravely embarrassed that death from asphyxia may rapidly follow.
6. The symptomatology is very characteristic, and enables the condition to be recognised as a definite clinical entity.

7. The diagnosis is based on the sudden onset of intense dyspnoea, the abundant, frothy, serous, often blood-stained expectoration of albuminous nature, the crepitant râles all over the chest with increased resonance on percussion ("paradoxical percussion"), and the absence of pyrexia.

8. The prognosis is serious. Even when the patient recovers from an attack, he is still liable to recurrences which again place his life in danger.

9. The treatment must be quick and energetic, the most useful procedure being a liberal venesection. This should be preceded by a hypodermic injection of atropine.

- C A S E S . -

- I. Aortic incompetence, arteriosclerosis, hypertension.
- II. Aortic and mitral incompetence.
- III. Aortic incompetence.
- IV. Aneurysm of the heart.
- V. Aortitis. Sudden death.
- VI. Aortic and coronary atheroma. Sudden obstruction of the left coronary artery.
- VII. Generalised arteriosclerosis and interstitial myocarditis.
- VIII. Aortic and mitral disease, subacute tubular nephritis.
- IX. Aortic disease and probably chronic nephritis.
- X. Pericarditis, interstitial nephritis.
- XI. Chronic Bright's disease, hypertension, cardiac hypertrophy.
- XII. Early interstitial nephritis, hypertension, slight cardiac hypertrophy.
- XIII. Early Bright's disease.
- XIV. Saturnine nephritis, cardiac hypertrophy.
- XV. Chronic Bright's disease.
- XVI. Chronic Bright's disease.
- XVII. Chronic interstitial nephritis with cardiac hypertrophy, influenza, acute tubular nephritis.
- XVIII. Acute nephritis due to scarlet fever.
- XIX. Acute nephritis.
- XX. Hysterical stigmata.
- XXI. Peritonitis, compression of the vagus.
- XXII. Ether anaesthesia.
- XXIII. Pregnancy associated with cardiac disease.
- XXIV. Articular rheumatism.
- XXV. Typhoid fever.
- XXVI. Pneumonia.
- XXVII. Chronic bronchitis and emphysema.
- XXVIII. Thoracentesis.
- XXIX. Idiopathic. Sudden death.
- XXX. Idiopathic. Recurrent attacks.

CASE 1.

Aortic incompetence, arteriosclerosis, hypertension.

Huchard: *Revue internationale de médecine et de chirurgie*, Paris, November, 1895.

A few weeks ago I saw in town a patient, aged 44, who had suffered for about fifteen years from aortic incompetence of rheumatic origin. But it is useful to add that, for several years also, his arterial system had commenced to show sclero-atheromatous degeneration. The temporal arteries had become more and more pronounced, the radial arteries hard and resistant to the touch, the vascular tension much augmented, the subclavian arteries raised up. For some months, the long phase of quiescence of his aortic affection, had been succeeded by a period disturbed by various phenomena, at first unimportant in appearance: dyspnoea on exertion and nocturnal paroxysms, some vague præcordial pains, caused chiefly by walking, and from time to time a very small quantity of albumin in the urine, and a little oedema of the legs.

One day, suddenly, the dyspnoea was different from what it had been before; the patient's face was pale with some dark patches of cyanosis on the cheeks, an incessant cough set in, followed by continual spitting, then spumous expectoration, slightly pink, and so abundant that in two or three hours several spittoons were almost filled. Then I noticed for the first time, in the middle of the chest and to the left, a focus of very fine crepitant râles which rapidly reached a portion of the pulmonary apex. All this disappeared within a few days.

But this disturbance was a serious warning; it was repeated on several occasions; soon the râles invaded both lungs, from their central part or from the base to the apex, and the patient succumbed in a few weeks in the midst of asphyxial symptoms.

What was the matter with him? A very important complication, more frequent than is believed and which often passes unnoticed: an acute oedema of the lung.

CASE II.

Aortic and mitral incompetence.

The following case was reported by me in the British Medical Journal, March 20th, 1926, p. 528.

The man is a printer and is 33 years of age. He has valvular disease of the heart, the aortic and mitral valves being incompetent. The heart is enlarged, and there is conspicuous pulsation of the carotid arteries. His countenance is strikingly pale, and the pulse is of the collapsing type. The lungs, however, are clear, and there is no œdema of the ankles; but the urine contains some albumin. The systolic blood pressure is 150, and the diastolic 80 mm. of mercury. The cardiac affection dates from an attack of rheumatic fever at the age of 16, and does not interfere with his following his occupation, which does not involve much exertion.

During the past year and a half he has had four attacks of acute, or rather hyperacute, œdema of the lungs. They have all occurred in the middle of the night and presented the same features. On arrival I find him sitting propped up in bed, anxious-looking and cyanosed, and constantly expectorating a frothy serous fluid which comes up without any effort. Coarse moist râles are heard everywhere over the chest. The pulse is rapid and forcible. He is fully conscious and repeatedly asks for something to be done quickly to relieve him of his distress, which appears to be a feeling of impending suffocation, for he is literally drowning in his own secretion. His extremities are cold. The application of a hot-water bottle to his feet, a hypodermic injection of atropine gr. $\frac{1}{50}$, followed later by morphine gr. $\frac{1}{4}$, render him more comfortable. The following morning he is quite well.

The attack comes on without any warning, waking him from his sleep, and usually lasts several hours, during which time he brings up a couple of pints of the watery, pinkish-tinged, frothy fluid, the coloration being most obvious in the froth.

Since recording the above, I have learned that the patient had another two attacks, in the latter of which he succumbed with symptoms of asphyxia.

CASE III.

Aortic incompetence.

M. Rockfelt: British Medical Journal, June 19, 1926, p. 1033.

The patient is a woman, aged 52 years. She has aortic incompetence, signs of which were first observed three years ago. Between her attacks she is very pale; the heart is moderately enlarged, and the systolic blood pressure in the arm is 160 mm. Visible pulsation in the carotid arteries and water-hammer pulse are well marked. There is œdema of the feet, but no signs in the lungs. She is irritable and difficult to manage.

During the past year she has had nine attacks of acute œdema of the lungs, all of a similar character. The interval between the first two was about three months, but they have gradually become more frequent, only a fortnight intervening between the last two attacks. They all occurred (with one exception) between 10 and 12 p.m., and nearly all on Sundays - perhaps brought on by excitement and overeating on these days. The onset is sudden and without any previous warning. On arrival, she is found making efforts to sit up in bed; she is anxious and terrified and appears to be aware of the danger of the attacks. The breathing is rapid and difficult. The face is pale and covered with a cold sweat. The lips and nose are livid. The fingers are cold and clammy. The pulse is feeble, small, and rapid. The temperature is subnormal. There is a profuse pink frothy expectoration and foaming at the mouth. After a short time consciousness is lost. Over the entire chest are heard numerous coarse bubbling râles. The attack lasts about two hours. On one occasion the patient had two attacks in one night, between which she recovered consciousness and lost her distress. On the day following an attack she feels quite well, and has often been found out of bed.

The treatment adopted in each attack consisted of a hypodermic injection of morphine 1/6 grain and atropine 1/100 grain, aromatic spirit of ammonia (1 drachm) by the mouth, and a hot-water bottle to the feet. If there was not much improvement in fifteen minutes the atropine was repeated.

CASE IV. (Résumé).

Aneurysm of the heart.

Parisot and Spillman: Gazette hebdomadaire de médecine et de chirurgie, Paris, July 15th, 1897.

A woman, aged 69, without any pathological history, was admitted into the Saint Julian Hospice on January 11, 1897.

For three months she had been suffering from respiratory difficulty and feelings of suffocation. She was of medium height and obese. Face swollen, oedema of lower limbs, apyrexia.

Examination of heart revealed nothing abnormal. The sounds were regular and well marked. Pulse feeble, but regular and equal. Patient complained of a somewhat sharp pain in the præcordial region. Urine scanty (600 to 700 grams in 24 hours), clear, and without albumin.

On auscultation of lungs: expiration prolonged at apices and subcrepitant râles at bases. Increased resonance in front, and dulness at bases.

In the evening of the 11th, the patient was suddenly seized with an attack of intense dyspnoea. The hands were cold, cyanosed; face swollen, oedematous; lips violet-coloured. Oedema of the legs was marked, and respiratory difficulty intense.

In the lungs, fine subcrepitant râles from apex to base. Nothing in the heart. Pulse small and feeble.

This attack ceased at the end of three-quarters of an hour, after injections of ether and cupping. It finished by the rejection of a very abundant, watery, aërated and frothy expectoration.

The following days the patient had three attacks, which occurred towards seven in the evening.

Treatment: Twelve drops of Tincture of Strophanthus (5%).

22nd.- Pulse more regular, more sustained; dyspnoea less severe; oedema of legs almost disappeared.

On January 24th and February 20th, fresh attacks with frothy expectoration.

March 29th.- At 8 o'clock in the evening, after having had her food as usual and feeling well, with the exception of a little oedema of the legs, the patient was seized with oppression. Numerous râles were heard in the chest, the respiration became more and more difficult, and

she died in ten minutes.

Autopsy.- Lungs congested at the bases, and emphysematous at the apices and anteriorly. On cutting, abundant frothy serum escaped.

Heart slightly increased in size and laden with fat. Valves normal. On opening the heart, at the apex was discovered an aneurysm, whose cavity was filled with a large stratified clot. The ventricular wall, at this point, was reduced to some muscular fibres surrounded by sclerous tissue.

The coronaries were markedly atheromatous; likewise the aorta.

The kidneys were small and granular; the cortex atrophied; some old infarcts were present in the parenchyma.

The liver was slightly cirrhotic.

The other organs appeared normal.

CASE V. .

Aortitis. Sudden death.

Lesage: Thèse, Paris, 1896.

On July 22nd, 1896, John P....., aged 40, was on a visit to "rue Dragon." He had not been feeling well since the previous day, complaining of great fatigue and general lassitude. His indisposition becoming worse, he went into a chemist's, bought a draught, and returned to his friends. A few minutes later he fell down without warning and expired.

He was not known to be suffering from any disease.

Medico-legal autopsy.- External examination reveals no signs of violence or ecchymosis.

On opening the thorax, the lungs appear prominent, very voluminous, as if inflated, and, on cutting them, an enormous quantity of froth escapes; the lungs constitute a veritable sponge of froth. The latter appears at the mouth. There exists a sanguineous exudate of about 250 grams (8 ounces) in each pleural cavity, but the lungs are entirely free.

The ascending aorta is the seat of internal lesions, a mixture of old atheroma and recent changes characterised by gelatinous-looking patches. This aortitis is very localised; it does not extend beyond the ascending aorta and stops before the sigmoid valves which are free.

All the other organs are healthy.

Conclusion.- John P.... has succumbed to an acute pulmonary oedema determined by aortitis of which the autopsy discloses unequivocal signs.

CASE VI. (Résumé).

Aortic and coronary atheroma. Sudden obstruction of the left coronary artery.

Fouineau : Thèse, Paris, 1898, p. 116.

R....., aged 67, labourer, is a mild alcoholic, and the only infectious diseases he has had are measles and scarlatina at the ages of 9 and 14 years respectively.

At 45 years of age, attacks of oppression began to appear, and were treated as asthmatic attacks by potassium iodide and inhalations of pyridine.

As R.... got older, these asthmatic attacks increased in frequency and duration; and, since the age of 50, R.... has suffered as it were from constant dyspnoea, directly he exerts himself.

He has been treated for emphysema with chronic bronchitis, but no one has ever spoken to him about cardiac trouble.

He has had neither palpitation nor præcordial pain radiating into the left arm.

He went into hospital for this dyspnoea on exertion, with its augmentation during the night, but at the same time without any attacks of oppression.

Physical examination.— R.... is very muscular, and without any oedema of the legs. Deformity of thorax due to this emphysema.

In the lungs is noted a diminution in the intensity of the vesicular murmur, diminution of vocal resonance, slow and prolonged expiration, with sonorous sibilant râles.

As the lungs cover the heart, auscultation of the latter is rendered difficult.

The aorta is dilated, and its beats can be felt behind the suprasternal notch. In the second right intercostal space is heard a loud systolic murmur. No diastolic murmur, no arrhythmia. Pulse 80 and sustained; the arterial tension is 210 mm. Hg.

The liver appears to be large, is regular and not painful; but it is simply displaced downwards, for the hepatic dulness begins two finger-breadths below the nipple.

R..... has chronic constipation and hæmorrhoids; his urine is abundant ($1\frac{3}{4}$ to 2 litres per day), and he has slight nocturnal polyuria. Neither albumin nor sugar is present.

One afternoon R.... went into the garden; suddenly, while sitting on a bench, talking, he was seized with dyspnoea and died at the end of an hour and a half.

During the phase of asphyxia his face was swollen,

of a livid colour, and his limbs were cold and violet-coloured.

The chest was filled with fine, moist râles. Auscultation of the heart revealed a tachycardia, with extreme weakness of the cardiac beats.

Autopsy.- Lungs voluminous, oedematous and congested posteriorly; emphysematous anteriorly. On section, a pink, frothy serum flowed out in great abundance. The sub-pleural space had become evident. Neither foci of hepatisation nor of haemorrhage. All parts of the lung floated.

The heart was hypertrophied. The left coronary artery was sinuous, dilated and indurated. No valvular lesions nor any of the endocardium. The aorta was atheromatous. The left coronary orifice was punctiform, obstructed by a calcareous plaque. All the proximal part of this artery was obstructed by a fibrinous blood-clot.

The liver was large and congested: nutmeg liver.

The spleen was slightly enlarged.

The kidneys showed a certain degree of interstitial nephritis.

The suprarenal capsules and pancreas were normal.

CASE VII. (Résumé).

Generalised arteriosclerosis and interstitial
myocarditis.

Fouineau: Thèse, Paris, 1898, p. 128.

B....., aged 53, broker, has a somewhat voluminous previous history: measles at 8 years of age, scarlatina at 12; following the latter, palpitation for a month, and since then difficulty in running or going up stairs.

At 20, syphilis, under treatment for six months only; several attacks of gonorrhoea, which resulted in a urethral stricture. Habitual alcoholic.

Then he had dysentery on five occasions. In 1870, he had scurvy for two months.

In 1881, when 39 years of age, he had fairly good health despite his alcoholic habit and the respiratory difficulty that he had since the age of 12.

For about ten years he has had some varicose veins, as well as polyuria and pollakiuria.

In April last, he began to cough as the result of a chill. The respiratory difficulty increased; B..... noticed that his legs were much swollen in the evening, and in the morning he had slight oedema of the eyelids. Frequent attacks of migraine, and loss of appetite.

Five days ago he went for a long walk which tired him greatly; this was followed two days later by very severe dyspnoea for which he was admitted into hospital.

B.... is a very muscular individual; he is half-sitting up in bed. Fine râles are heard in both lungs from top to bottom. No diminution of percussion note. The vocal resonance is normal.

B.... coughs frequently and brings up a very abundant mucous expectoration.

His heart beats in the sixth intercostal space.

The beats are strong and raise the thoracic wall.

No murmur but bruit de galop. The pulse is small and compressible. The arteries are hard, the temporal tortuous.

The liver is large, the spleen appreciable.

No oedema of the legs. The urine voided (400 grams in 24 hours) contains no albumin.

B.... has very pronounced myosis, the pupils are pin-point, the patellar reflex is diminished.

Milk diet, caffeine, oxygen, dry and wet cups, improve the patient's condition. On the 25th the dyspnoea is greatly diminished, the râles are less numerous; 750 grams

urine secreted.

31st.- B.... retains his bruit de galop, and an arterial pressure of 190 mm. Hg., but he has passed $2\frac{1}{2}$ litres of urine within the twenty-four hours and the pulmonary symptoms have disappeared.

B.... left hospital on June 9th completely recovered.

CASE VIII.

Aortic and mitral disease, subacute tubular nephritis.

J. Lindsay Steven: Lancet, Jan. 11, 1902, pp. 74-75.

The patient, a man, aged 40 years, was admitted into the infirmary for the first time on Dec. 27th, 1899, on account of swelling of the legs of a month's duration, and shortness of breath on exertion, cough, and expectoration of a fortnight's duration. It is unnecessary to go into the clinical history in detail. It is sufficient to say that the man was found to be the subject of aortic obstruction and regurgitation and of mitral regurgitation. In addition the presence of albumin, blood, and granular tube casts in the urine indicated a subacute nephritis, probably of tubular character. These parts of the case were frequently made the subject of clinical demonstration in the wards during both his first and his second residence, the latter beginning on Nov. 23rd, 1900, and ending with his death on Jan. 27th, 1901. At present we are interested in these elements of the case in so far only as they were complicated with attacks of acute suffocative pulmonary oedema, and I shall from the ward journal read to you the accounts written at the time of the first and the second last seizures.....

"Dec. 30th, 1899.- To-night, at 7.25 p.m., the patient was suddenly seized with great dyspnoea. He was terribly distressed for breath and plucked at the clothes on his chest. His face was cyanosed and the veins of the neck were distended. The pulse during the attack numbered between 150 and 160 and was full and bounding. There was a good deal of rattle in the trachea and he spat up copiously a white frothy material which later became pinkish. Amyl nitrite and inhalations of oxygen were administered with some relief, after which the pulse numbered between 120 and 130. The heart was beating very forcibly and the chest was full of râles. A hypodermic injection of strychnine $\frac{1}{60}$ grain) was given. The patient still continued very breathless. His body was cold and covered with a profuse clammy sweat. The pulse continued about the same rate but varied greatly in force. Additional inhalations of oxygen seemed to give but little relief and stimulants were refused. The sputum became very profuse and was more distinctly red. About 9.15 p.m. strychnine ($\frac{1}{60}$ grain) was again given

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hypodermically. Dr. Lindsay Steven was telephoned for and saw the patient at 9.30 p.m. when the condition was much the same. Since then he has had brandy both hypodermically and by the mouth. At 11.30 p.m. the patient was still very ill and breathless. The pulse was fairly good and numbered about 140. His sputum was rather less profuse but distinctly red. The body surface was warmer and the sweat much less.

"Dec. 31st.- About 2 a.m. the patient began to recover from the seizure described in the foregoing note. Dr. Lindsay Steven from his own observations confirms the accuracy of the account, and taking all the phenomena into consideration is on the whole disposed to think that the seizure has probably been caused by embolism of one of the large pulmonary branches. The blood-stained and highly frothy sputum with the excessive struggling for breath were indicative of an acute pulmonary oedema, and the presence of the renal disease raised the question as to whether the whole seizure might not be uræmic in origin. The discovery this morning of distinct dulness at the right base below the scapula and of numerous fine, moist, crackling râles, chiefly inspiratory, and of a distinct bronchial respiratory murmur, is a strong indication in favour of embolism. At 3 p.m. the patient complained of cold but had no actual rigor. His pulse at 4 p.m. was 124 per minute and his respirations were 50. His temperature was 103.60. He was not expectorating so much but the sputum was very bloody. He continued much the same and at eight o'clock his pulse was 120 and his respirations were 60 per minute. When seen at nine o'clock he was lying on his right side. The body surface was very warm and the skin was covered with sweat. The pulse was 132 and the respirations were 60 per minute; the breathing, however, was not very distressed. There was still a good deal of rattle in the trachea.

"Jan. 11th, 1901.- Last night about 7.25 p.m. the patient passed into a dyspnoeic attack in all respects similar to that described in note of Dec. 30th, 1899. The seizure lasted until 2 a.m. and was accompanied by a frothy bloody expectoration to the extent of 26 ounces. During the seizure the respirations numbered 56 and the pulse 160; the chest was full of snoring and wheezing rhonchi; and the patient had to sit up, being unable to remain in bed. When the violence of the paroxysm had passed he was able to return to bed, but not to lie down, and in the sitting posture obtained short snatches of sleep. The expectoration examined this morning consisted of three parts - (1) a large amount of pink and white froth which

floats on (2) a thin watery tea-coloured fluid, and (3) of a very small amount of blood-clot at the bottom of the spittoon. The physical characters of the expectoration can only indicate acute pulmonary hyperaemia and oedema. This morning the apex beat is found in the fifth interspace five and a half inches to the left, and is abrupt and slapping in character. The cardiac area is greatly increased, particularly to the right. Right border two and a half inches to right of mid-line; left border five and a half inches to the left at level of fifth interspace. Upper border is at level of upper margin of fourth cartilage. A loud superficial ventricular systolic murmur is heard at the apex and faint ventricular systolic and ventricular diastolic murmurs in the aortic area. At the right base posteriorly, from the level of the sixth dorsal downwards absolute dulness on percussion is obtained, and in the area numerous fine crackling râles with inspiration are heard. Similar crackles are also obtained at the left base. Throughout the present residence the temperature has been normal or subnormal and the average daily quantity of urine rather above than below the normal amount. The amount of albumin has averaged about 2 per cent. (Esbach) and a trace of blood has always been present."

The patient died in a similar attack on Jan. 27th, 1901; and I now read to you Dr. C. Workman's report of the post mortem examination.

"Post-mortem summary.- Subacute tubular nephritis; chronic changes in aortic valve; stenosis of mitral valve; and congestion of other organs.

"Post-mortem report.- A well-developed and well-nourished body, rigor mortis still present. There is well-marked oedema of the lower extremities. The heart is enlarged. This is chiefly in left ventricle. The pericardium contains a considerable quantity of serous fluid. The pulmonary valve is competent; the aortic is incompetent. The pulmonary curtains are healthy. The aortic curtains are considerably thickened, especially along the margins. The right curtain and the adjacent portion of the posterior are infiltrated with lime salts. There is a large thrombus pyramidal in shape adhering to the margin of the posterior curtain. The endocardium over the septum immediately below the right aortic curtain is thickened over an area of about one inch square. The left ventricle is dilated and the wall is considerably hypertrophied. There is stenosis of the mitral valve and great thickening along the margin of the curtains. There are several small thrombi on the auricular surface of the valve. The left auricle is dilated.

The right ventricle shows some hypertrophy and the right auricle is also dilated. Coronary arteries present one or two small patches of atheroma. Cardiac muscle is of good consistence and colour. Both pleural cavities contain a quantity of serous fluid. Both lungs present a number of adhesions to the chest wall. Both are deeply congested and oedematous and there is no evidence of pulmonary embolism or thrombosis. The liver is enlarged and presents a nutmeg appearance. The spleen is enlarged and congested. Both kidneys are enlarged, the capsules being non-adherent. The surface presents a mottled appearance, the yellow of the mottling being due to a fatty degeneration of the tubules. The left kidney presents slight hydronephrosis. No obstruction is found in the right ureter. The aorta presents a perfectly healthy appearance. The brain presents nothing unusual. The stomach and intestines are healthy. The weight of the organs is as follows: heart 19 ounces, right lung $39\frac{1}{2}$ ounces, left lung 40 ounces, liver 80 ounces, spleen $13\frac{3}{4}$ ounces, and the right kidney and left kidney together $20\frac{1}{2}$ ounces."

CASE IX. (Résumé).

Aortic disease and probably chronic nephritis.

Fabre: Thèse, Toulouse, 1905, p. 50.

A Custom House servant, aged 45, complains of being subject to sudden attacks, during which he can hardly breathe, and which last from an hour and a half to two hours, then disappearing spontaneously.

In certain of these attacks he has had an abundant and whitish expectoration.

In 1898, when 40 years of age, he had anasarca following the ingestion of cantharides powder.

In 1904, he experienced attacks for which he was admitted into hospital: he is a fairly corpulent man; his face is pale; it seems to be somewhat swollen; there is no malleolar oedema.

He is a nervous man, very interested in his disease; he is also an alcoholic. He has had syphilis; further he has aortic disease: examination of his heart revealed that the second sound at the aortic orifice was greatly accentuated, "ringing," and that there was a marked dilatation of the aorta; he is also probably a nephritic: he has albumin in the urine in fairly large quantities; he shows certain minor symptoms of Bright's disease.

From September 29th to December 9th he had five attacks, in three of which his life was in danger.

Suddenly he experienced a sensation of intra-thoracic constriction and respiratory difficulty; then some fits of coughing, which caused a very slight amount of spumous expectoration; the respiratory distress increased more and more; the dyspnoea became intense: 40 respirations per minute. The patient was on his knees on a chair, his head bent forward, his brow bathed in sweat, he was anxious-looking and pale, his lips of violet colour.

The pulse was strong and tense, but rapid (120 to the minute). His heart beat energetically.

On auscultation, coarse sibilant râles were heard over the entire chest, and fine crepitant râles at both bases.

Ten wet cups at the time of this attack rapidly relieved the patient and in an hour respiration had again become easy.

The next day auscultation of the lungs revealed nothing abnormal.

Examination of the urine after the attack revealed traces of albumin.

At the time of an analogous crisis it was noted that it was preceded by an appreciable diminution in the emission of urine.

Under the influence of milk diet and theobromine, the patient passed about two and a half litres urine during the twenty-four hours; the day before the attack he passed only one and a half litres. The preceding nights his sleep had been restless; he suddenly awoke with a jump. For some days he experienced a feeling of intra-thoracic constriction, as if he had a weight on his chest. Finally, he confessed to us that he had been secretly eating food that had been brought to him from outside.

CASE X.

Pericarditis, interstitial nephritis.

Huchard: Séance de l'Académie de médecine, Paris,
April 27, 1897.

A man, aged 52, was admitted into the Necker Hospital on August 14th, 1896, owing to respiratory disturbances of ten days' duration (incessant cough, constant oppression, abundant expectoration). His face was pale, eyelids slightly swollen, urine abundant, containing a trace of albumin; there was no bruit de galop.

At the right apex was detected a focus of very fine crepitant râles, suggesting the existence of concurrent tuberculosis; but examination of the sputum was negative. Further, at the end of eight days, was observed the disappearance of the oedematous focus, the crepitant râles and the expectoration, with an increase of albuminuria.

At the end of September, without known cause for this aggravation, the albumin increased to 5 grams per litre, and on October 21st, after some uræmic disturbances (vomiting and diarrhoea, semi-comatose condition), considerable dyspnoea suddenly appeared, coinciding with the presence of a veritable shower of very numerous crepitant râles, extending almost throughout the lungs, without any expectoration; the heart was very accelerated with embryocardia, the stomach considerably distended, and we noted a very strong double pericardiac friction sound at the base of the præcordial region. The orthopnoea became extreme, the face was of a cadaverous pallor, and the patient died from asphyxia during the night with a temperature of 35.6°C.

At autopsy, small, granular kidneys weighing 170 grams the two; about 100 grams of a sero-fibrinous liquid in the pericardiac cavity, recent lesions of pericarditis at the base; some pleural adhesions on the right side without effusion; the lungs without infarct, but infiltrated with such abundant serum that there flowed from the parenchyma, on simple compression with the fingers, more than a litre of a sero-sanguineous fluid of salmon-like appearance.

CASE XI. (Résumé).

Chronic Bright's disease, hypertension, cardiac hypertrophy.

Bouveret: Revue de médecine, Paris, March 1890, No. 3.

G.L., a navvy, aged 62, began ten months ago to have temporary attacks of oppression and palpitation as the result of fatigue or prolonged work.

April 21st, 1889.— He had an apoplectic seizure. The aphasia and right hemiplegia which followed, disappeared in about ten days.

The present condition commenced two days ago, May 17th, 1889. The patient was suddenly seized with a fit of coughing and very intense oppression, followed by the abundant expectoration of a pinkish fluid.

The dyspnœa is extreme to-day. The patient is sitting up in bed; his face is pale, with cyanotic patches; his limbs are cyanosed. Every moment he has an attack of coughing, followed directly by abundant expectoration. During the last twelve hours he has expectorated about two-thirds of a litre of a frothy, pink fluid, precipitated by nitric acid. The chest is full of sibilant and subcrepitant râles. There is also a little weakness in the motility of the right side. A little delirium. Headache.

The pulse is very strong and sustained; the finger is lifted with great energy. There is a considerable increase of the arterial tension. The pulse of all the arteries accessible shows the same strength and the same exaggerated fulness; its rate is 104.

The feet are slightly œdematous. The liver extends several finger-breadths beyond the ribs. The hypertrophy of the heart appears considerable. At the base and along the left margin of the sternum, a murmur is heard with the second sound, comparable to the murmur of aortic insufficiency. Since the beginning of this attack of orthopnœa, the patient has passed very little urine, dark yellow, containing much albumin.

May 20th, 1927.— The dyspnœa is still severe. Râles fill the chest and obscure the cardiac sounds. During the night, the patient has filled several spittoons with this pink albuminous fluid. Temperature 39.90C.

Bled 400 grams; dry cups; German brandy; injection of caffeine and of ether.

The dyspnœa had not diminished by the evening, and the patient died the evening of the following day.

Autopsy.- Both lungs are the seat of an enormous œdematous congestion, which extends from the base to the apex. On section, liquid flows. The bronchi are full. No hæmorrhagic foci, nor any patches of bronchopneumonia.

The heart is enormous, 920 grams. The hypertrophy affects the left side of the heart especially. The myocardium seems healthy. The right papillary muscle alone contains a small patch of sclerosis. Yellow plaques of endarteritis on the large coronary trunks. The valves are healthy. The ascending aorta is dilated, but not atheromatous. The visceral pericardium is covered with large milky plaques.

The liver is large and has the nutmeg appearance of the cardiac liver.

Kidneys.- The capsule is very adherent, and the surface very granular. Some small cysts. The cortical substance is diminished in thickness, and the striation is less clear than in the normal condition. The pyramids and medullary substance are the seat of intense venous congestion.

CASE XII. (Résumé).

Early interstitial nephritis, hypertension, slight cardiac hypertrophy.

Bouveret: Revue de médecine, Paris, March 1890, No. 3.

M.B., aged 45, is suffering from interstitial nephritis in its first stage. When 23, he went to Indo-China, and remained there nineteen years. He had dysentery several times, and severe variola. He abused both the pleasures of the table and tobacco. In 1879 and 1886, he had renal colic and passed some gravel.

After returning to France, he continued to smoke a great deal and to take much meat and wine. He suffered from moderate oppression after the slightest effort.

November 30th, 1888.- He returned home in the morning after walking 3 kilometres. He was suddenly seized by a distressing sensation of oppression in the chest with an irritation in the throat and a dry cough, the attack becoming more and more intense. The oppression increased to orthopnoea. A doctor, summoned hurriedly, was struck by the pallor of the face and found that the chest was full of râles. After several minutes of this respiratory distress, there was abundant expectoration of a watery fluid, mixed with air bubbles and slightly tinted with pink. From this moment the dyspnoea diminished and the patient experienced considerable relief. The fluid expectorated exceeded a litre during this attack, which lasted from seven to eight hours. Expectoration continued for two days, more or less copiously; it was fifteen days before the patient could resume his occupation.

M.B. continued to take much wine and alcohol.

March 4th, 1889.- He came to consult me for the first time. He complains of oppression directly he walks quickly or goes upstairs, and of an unusual sensation of lassitude. He looks like a man in good health, but has the symptoms of commencing interstitial nephritis. He has a rapid pulse, 90 per minute; the radial artery is hard, tense, difficult to compress; the arterial tension is evidently markedly exaggerated. The præcordial impulse is very energetic. The left ventricle is very slightly hypertrophied. The cardiac sounds are strong, loud, especially the second aortic sound. Neither murmur nor bruit de galop is present. On auscultation of the lungs, some very fine subcrepitant râles are heard right at the base. The urine is slightly

albuminous, and, from the patient's statement, slight polyuria is probable.

Prescription: Moderate milk diet; 1 gram of sodium iodide daily, at meal-times; dry cups over lumbar region.

March 7th, 1889.- Second attack of orthopnea, but much less intense than the first.

May 1st.- General condition good. Sensations of oppression and lassitude a little less pronounced. Urine still albuminous.

June 19th.- Without any appreciable cause, a third very violent attack took place, just when the patient was going to bed. Sensation of tickling in the throat, fits of coughing; the oppression increases; the face and extremities become livid. Expectoration appears, very abundant and pinkish. This time the expectoration was earlier, and the sensation of suffocation was less distressing and of shorter duration. The attack lasted four hours and two litres of fluid were ejected. The chest was full of râles, and the patient experienced a kind of sensation of internal bubbling. The lassitude following the attack was less pronounced than previously; and the following day the patient was able to resume his occupation.

October 15th, 1889. The condition of M.B. seems satisfactory. The oppression and usual lassitude have much decreased owing to the almost complete milk regimen followed for several months. The pulse remains frequent (80 to 90). The arterial tension is still increased, 220 mm. Hg. The urine, somewhat pale, contains a little albumin; the amount passed reaches at least two and a half litres. The cardiac hypertrophy is hardly noticeable. The paroxysmal œdema of the lung has not occurred for the last three and a half months, but it is to be feared that the patient has not lost it, for the nephritis persists.

CASE XIII. (Résumé).

Early Bright's disease.

Dieulafoy: Bulletin médical, Paris, November 3, 1897.

October 15th.- This man, aged 45, compositor, was attacked by an oppression so rapidly distressing, that there was only time to take him to the Hôtel-Dieu, where, on arrival, he gave the impression of imminent asphyxia and approaching death. His face was pale, covered with cold sweat, his eye dull, lips livid, fingers and nails blue, the respiration distressed and hurried, the pulse miserable and accelerated; such was the condition of the moribund.

Auscultation of the heart was impossible; auscultation of the chest revealed throughout both lungs some sibilant râles and a veritable shower of fine subcrepitant râles. The patient coughed and spat, and his abundant expectoration had a frothy, pink appearance in the spittoon. Further, the eyelids were swollen, the legs slightly œdematous; the urine was strongly albuminous. Temperature 35.2° C.

These various symptoms evidenced the existence of nephritis; and the frothy, albuminous, pink sputum joined to the shower of fine râles which encumbered the chest all enabled one to diagnose hyperacute œdema of the lung of Bright's disease.

Wet cups were at once applied, and a venesection of 300 grams was done. A change of appearance took place in the patient. His respiration became freer, expectoration scantier. In less than an hour, the shower of fine râles disappeared from the upper parts of the lung and death was conquered. Absolute milk diet was prescribed, lactosed drinks, and a daily dose of 20 grams of Trousseau's diuretic wine.

The following day the patient was transformed, he was breathing easily, the asphyxial tint had disappeared, and the temperature was normal. Auscultation of the heart, which had become possible, revealed a slight bruit de galop; on auscultation of the lungs, râles could only be heard at both bases.

The urine, almost nil the previous day, but strongly albuminous, had now increased to 200 grams. The battle was won.

The following day the patient was able to tell us that for several months he had experienced some of the minor disorders of Brightism: Pollakuria, cramps in the calves, sensation of dead fingers, œdema of the eyelids and of the malleoli; and it was during the course of this Brightism

that the attack of pulmonary oedema had occurred.

To-day, the puffiness of the face and oedema of the legs have completely disappeared. The quantity of urine has reached 150 grams; however, albumin persists to the amount of 1.5 gms. daily, and urinary depuration is still inadequate, for the toxicity of the urine, experimentally tested, is far from having attained its normal level.

CASE XIV.

Saturnine nephritis, cardiac hypertrophy.

Giraudeau: Bulletin médical, Paris, November 3, 1897.

A house-painter, aged 45, suffering from saturnine disorders, was admitted into the Hôtel-Dieu in 1895 for anasarca following saturnine nephritis. Four days after the patient's admission into hospital, Dr. Giraudeau found him one morning in the grip of the most intense dyspnoea, which had commenced the previous evening. The oppression had been so violent that several times the patient thought he was dying. During the night three spittoons had been filled with a spumous, pink foam; the rosy tint was very pronounced in the first spittoon, it was less so in the second, and still less in the third. Auscultation of the chest revealed a veritable shower of very fine subcrepitant râles.

A venesection of 500 grams was done, and the following morning, though the dyspnoea had not completely disappeared, the patient was much better, the râles were less numerous, not so fine, and the expectoration was much less abundant. The following days the expectoration was accentuated without, however, the oppression disappearing altogether. Anasarca was very pronounced, the urine was strongly albuminous, and much less toxic than in the normal condition. Finally, the patient tolerated the milk diet very badly.

Two months later this patient was seized with toxic dyspnoea, uræmic, with Cheyne-Stokes respiration. The uræmic symptoms became aggravated and the patient fell into a constant drowsiness, which terminated in uræmic coma and death. At the autopsy, the two kidneys were found to be small, indurated, pale and cystic. The left ventricle was very hypertrophied.

CASE XV.

Chronic Bright's disease.

Dieulafoy: Bulletin médical, Paris, Nov. 3, 1897.

In September 1892, I admitted into my wards at the Necker hospital a patient suffering from uræmic disorders with oliguria. This patient with Bright's disease complained of oppression and violent headache. On auscultation, fine râles were noted in both lungs, especially posteriorly on the right side. These dyspnoëic symptoms, as well as the headaches, began about two months ago. The urine contained a slight proportion of albumin. His legs were markedly oedematous. As treatment, I prescribed an absolute milk diet and lactose drink. After several days of this regimen, the patient felt much better, the dyspnoëa had almost disappeared, only a few scattered râles were audible, and the oedema of the legs had markedly diminished. Nevertheless, the quantity of urine remained far below normal, which was not a good sign.

Suddenly, in the evening of September 3rd, the dyspnoëic disturbances re-appeared in an intense form. To the oppression, which continued to increase, abundant spumous, frothy, pink expectoration was added, as the consequence of Bright's hyperacute oedema of the lung, which persisted a part of the night. The following day the dyspnoëa still continued and, on auscultation, fine râles of all kinds were detected. I applied a number of wet cups to the chest; and the day after that, September 5th, I prescribed a blood-letting of 250 grams. Marked improvement followed as a result of the bleeding, the râles diminished and respiration became almost free.

CASE XVI. (Résumé).

Chronic Bright's disease.

Huchard: Revue de médecine et de chirurgie, Paris,
November 1897.

Patient, aged 45, has suffered from interstitial nephritis for several years. Attacked suddenly during the night by extreme dyspnoea. A doctor called in diagnosed uræmic dyspnoea, owing to the slightly albuminous urine having suddenly become very scanty. But one symptom was difficult to explain: a very abundant frothy expectoration. Further, very fine and numerous subcrepitant râles were detected at the bases of the lungs from the beginning of the attack.

The following day, owing to a concurrent epidemic of influenza, and having regard to the fine râles which had invaded the two lower thirds of the lungs, influenza of suffocative catarrhal form was suggested. But there was no fever, despite acceleration of the pulse, and every other influenzal symptom was absent in this patient. I saw the patient at 4 p.m.: intense orthopnoea, face pale and almost livid, pulse 140 with hypothermia 36.2°C., 76 respirations per minute, slight oedema of legs which only supervened a few hours before, abundant albumin when there were only a few centigrams the previous night, a small and compressible radial pulse with arterial hypotension when a few days before it was very resistant and cord-like; considerable swelling and pulsation of the jugular veins took the place of the exaggerated beats of the cervical arteries of the preceding days, with signs of a very acute dilatation of the right ventricle following those of left ventricular hypertrophy.

The heart beats were accelerated, with very distinct mid-diastolic bruit de galop. But the most remarkable thing was the presence of fine, compact, numerous râles heard throughout inspiration and during two-thirds of expiration, râles which had promptly invaded the whole chest from base to apex. Another surprising thing was the complete suppression of all bronchial expectoration, when it had been so abundant a few hours before, and had filled several spittoons. The bronchi were suddenly paralysed; and the diaphragm was as if immobile and paralysed in its turn.

I diagnosed hyperacute oedema of the lung with an extremely

grave prognosis. Despite abundant blood-letting, despite the use of purgative enemata, of dry cups, of injections of ether and camphor in oil, this patient succumbed rapidly from asphyxia, less than twenty-four hours after the commencement of the first symptoms.

CASE XVII.

Chronic interstitial nephritis with cardiac hypertrophy, influenza, acute tubular nephritis.

Merklen: Congrès international^{de} médecine, Paris, 1900. Semaine médicale, Paris, 1900, p. 617.

A patient, suffering from chronic interstitial nephritis, latent up to then, was seized with acute tubular nephritis during an attack of influenza. He was admitted into hospital where the condition seemed to improve under the influence of milk diet, but the specific gravity of the urine remained low, at 1008, with two litres of urine; and one day the urea figure fell to 1.56 gms. per litre.

The following day he was suddenly seized with an attack of acute pulmonary oedema, immediately subdued by treatment. During this attack, the pulse was accelerated, but did not weaken. The heart remained strong and its dulness did not appear increased. The arterial tension, which the day before was 22 cm. by Potain's sphygmomanometer, increased to 25 cm. Hg.

In short, there was no sign of cardiac weakness. The following day a focus of bronchopneumonia was detected at the right base. A few days later there was a second attack of pulmonary oedema, which was also stopped by a copious blood-letting; then a third attack which did not respond, and during which the patient died suddenly, just at the moment when my intern was taking his pulse; this enabled him to verify a sudden stoppage without any weakness or preliminary irregularity.

Cryoscopic examination of the urine, made the previous day by Mr. H. Claude, showed a maximum renal impermeability, and a few hours before death I found on the patient's forehead an abundant white powder consisting of crystals of sodium chloride and urea.

At autopsy, the kidneys were atrophied, the left ventricle hypertrophied but not dilated. It had stopped in systole and had the appearance of so-called concentric hypertrophy. The lungs were infiltrated with oedematous serum which flowed out on section. There was also cerebral oedema, and a small focus of bronchopneumonia in the right base, with recent pleuritic adhesions.

Histological examination of the myocardium showed grave changes of the right ventricle as well as of the left: interstitial oedema with numerous leucocytes, fibrinous exudate, granular degeneration and fragmentation of the muscular fibres.

CASE XVIII.

Acute nephritis due to scarlet fever.

Giraudeau: Bulletin médical, Paris, Nov.3, 1897.

In April 1897 was admitted into the Tenon Hospital a young girl, aged 17, complaining of lumbar pains and œdema of the legs of several days' duration. The day of admission generalised anasarca was diagnosed, with puffiness of the face; the urine, very scanty and high-coloured, contained abundant albumin. There was considerable fever, the temperature having risen to 39°C. On auscultation of the heart a slight bruit de galop was detected, without any valvular lesion. On examining the patient more closely, very distinct desquamation was detected on the soles of the feet and in the interdigital spaces, resembling the desquamation of scarlatina. Although questioning the patient did not enable one to reconstruct the entire clinical picture of this eruptive fever, the existence of acute nephritis of scarlatinal origin had to be admitted as the most likely hypothesis. Absolute milk diet was prescribed.

The œdema persisted during the following days, the quantity of urine excreted did not exceed 200 to 250 grams; albuminuria was intense and the temperature oscillated between 38° and 39° C. Lactose drinks, purgatives, wet cups to the lumbar region were all associated with the milk diet. The patient had been more than eleven days in hospital, having so far shown nothing in the case of the broncho-pulmonary apparatus, when she was seized, before the morning visit, with a sudden and severe attack of suffocation accompanied by abundant, frothy, pink expectoration which filled two spittoons in less than half an hour.

A venesection of 300 grams was done. An hour later the dyspnoea had diminished, but the expectoration remained still frothy and pink. In the chest were heard subcrepitant râles extending from the base to the apex. A quantity of dry cups were applied and inhalations of oxygen prescribed.

During the following hours the patient experienced considerable relief, but in the afternoon the dyspnoea reappeared, as intensely as in the morning, accompanied by the spumous, pink expectoration. To combat this

unpleasant return, she was again bled 300 grams. Calm was restored at once and the young girl passed a good night. Two days later there was a fresh and terrible attack of dyspnoea, with frothy, pink expectoration.

An immediate venesection did not overcome the attack, the asphyxia became menacing and the patient, no longer expectorating, succumbed in the night.

CASE XIX.

Acute nephritis.

J. Lindsay Steven: Lancet, Jan. 11, 1902, p. 74.

The following are the clinical notes entered in the ward journal by my resident assistant, Mr E.H.Roberts:-

"A woman, aged 38 years, was admitted into the Glasgow Royal Infirmary in a somewhat collapsed and highly dyspnoëic condition at 2.30 a.m. on Jan. 29th, 1901. She had retired to rest at 11 p.m. in her usual health. Shortly afterwards she felt that she could not get her breath properly, and attempted to improve matters by first lying on her left side, then on her right side, and lastly in the dorsal decubitus, but all to no purpose, the difficulty of breathing becoming rapidly worse. The dyspnoea was increased in a short time by frothy material coming up her throat which was chiefly whitish and at times pinkish in colour. The frothy fluid was expectorated in considerable quantity, the patient in the intervals gasping for breath. At this time severe lancinating pain was experienced across the front of the chest. A loud wheezing in the chest during the respiratory acts was observed by her. The patient was housekeeper to an old bachelor friend of Dr. Lindsay Steven who saw her at midnight and advised her removal to the Glasgow Royal Infirmary, whither she was removed in a cab. During the journey her condition was so critical that at times it was feared she would have succumbed. Upon admission she was gasping for breath. The face was somewhat flushed, the lips were livid, and she was freely expectorating a whitish frothy sputum. There was no cough. The respirations were noisy and wheezing, were greatly embarrassed, and numbered 34 per minute. The pulse was small, rapid, regular, and numbered 105 per minute. The temperature on admission registered 96.40° F., at 8 a.m. 99.80°, at noon 99.60°, at 4 p.m. 99.40°, at 8 p.m. 99.40°, and at midnight 98.60°. On auscultation of the chest, as quickly as possible, it was found to be the seat of fine and medium moist râles. By 6 a.m. she had sufficiently recovered to be bathed in bed, and by 9 a.m. she was able to lie down, her head being supported by two pillows, and henceforth she continued to make rapid and steady progress.

"Jan. 30th.- To-day the patient was almost herself again and it was possible to make a formal clinical report.

"Past history.- The patient states that she had always enjoyed good health until the onset of the present

attack. She had never noticed any swelling of her face, but had noticed some swelling of her feet and ankles at night occasionally which she attributed to her having to be a great deal on her feet. She had never had any urinary trouble, not having noticed anything unusual in the quantity or appearance of her urine. She had been subject to headaches occasionally. There had been no nausea or vomiting, no anaemia, or general weakness. Her appetite had been fair.

"Family history.- Her mother is alive and well. Her father is dead, the cause of his death being unknown. She has two brothers who are alive and well.

"Present condition.- Decubitus is dorsal and quiet. She is well developed and of fair nutrition. The pupils are equal, medium, and react normally. The lips and mucous membranes are of normal colour. The tongue is moist and coated irregularly with a greyish fur. The respirations are quiet and unembarrassed, and number 26. The pulse numbers 76 per minute, is of fair volume, medium tension, and regular. The temperature is 98.6°. There is a highly varicose condition of the veins of the right lower limb, with brownish discolouration of the middle and lower thirds of the leg.

"Respiratory system.- The chest is well-developed and symmetrical, while the respiratory movements are of normal excursion. On examination nothing abnormal with the exception of an occasional moist râle is detected, the respiratory murmur being of normal vesicular quality.

"Circulatory system.- The apex beat is somewhat heaving and diffuse and located in the fifth interspace, three and a half inches to the left of the mid-sternal line. The præcordial dulness has the following limits: upper, upper border of the fourth rib; right, three-quarters of an inch to the left of the middle line; left, three and three-quarter inches to the left of the middle line. The greatest transverse measurement is three inches. There is accentuation of the second sound in the aortic area, while the first sound in the mitral area is somewhat low-toned; otherwise the cardiac sounds are pure.

"Abdomen.- The hepatic dulness cuts the sixth rib in the mid-clavicular line and measures vertically three inches. No splenic enlargement can be detected. On examination no abnormality is detected.

"Alimentary system.- The tongue is moist and coated irregularly with a greyish fur. The bowels are regular, but the appetite is indifferent. The urine is acid, its specific gravity being 1022; it is clear amber in colour,

and there is a slight flocculent deposit. Microscopically hyaline and granular tube casts, leucocytes, and squamous epithelium are observed. Albumin is fairly abundant. The tests for sugar and blood are negative.

"Feb. 1st, 1901.- Since Jan. 30th there has been marked and steady improvement. The patient is now practically well. The temperature has ranged between 98.6° and 99°. The respirations have varied between 24 and 32, and the pulse between 68 and 78. She sleeps well and her appetite is fair. The bowels are regular. It has been impossible to estimate the amount of urine passed owing to some being lost."

The following note was made by me on Feb. 5th.

"The whole course of this case indicates that the patient has been suffering from acute suffocative pulmonary oedema associated with acute renal disorder, as evidenced by the albuminuria and tube casts at the time of admission. Very great improvement is now to be recorded. Absolutely no trace of albumin is found in the urine this morning, which to the naked eye presents perfectly healthy appearances. The temperature throughout has been normal, and the quantity of urine on the whole somewhat less than usual. There is perhaps very slight accentuation of the second aortic sound and quite distinct of the second pulmonic. The first sound at the apex is slightly muffled in character and the apex beat is very vaguely palpable in the fifth interspace four inches to the left. Posteriorly pulmonary percussion is normal and the respiratory murmur presents healthy characters back and front."

The clinical notes continue: "Feb. 12th.- Since the above record the patient has remained practically well. Her appetite to-day is good, the urine is normal, and the bowels are regular. On examination of the lungs nothing abnormal is observed. The cardiac condition remains much as in the previous note. She is now allowed up daily, a Martin's elastic bandage being worn for the varicose condition of the veins of the right lower limb.

"March 7th.- Since the previous record the patient has remained quite well. The lungs, on examination, are found to be quite normal, as is also the heart. The urine is entirely free from albumin, and microscopically nothing beyond an occasionally squamous cell is to be observed. She leaves for her home in the north, feeling quite well."

The clinical record which I have just read to you

may be taken as a very characteristic history of an almost pure case of acute suffocative pulmonary oedema occurring in the midst of apparent health and associated with renal disorder of a temporary character.

CASE XX. (Résumé).

Hysterical stigmata.

Lévi (Léopold): Archives générales de médecine,
October 1895, p. 473.

C...., aged 21, is the fifth of six children, all living, without neuropathic taint. When three years old, he had his only illness, whooping cough, which lasted eight months.

June 22nd, 1895.- C.... had been for several days out of sorts, complaining of headache and malaise, but he ate, slept, and occupied himself as usual.

On Saturday, after a day's work, he was seized without any cause, about 9 o'clock in the evening, with such a terrible sensation of dyspnoea, that he demanded "a razor to open his chest." He passed the whole night without sleeping, dozing only momentarily when he would start dreaming aloud. The next day he was admitted into St. Antoine, and spat blood.

C.... drinks about one litre of wine daily, and an absinthe now and then. Sometimes on a Saturday he has a drinking bout. As a rule his nights are disturbed by many dreams, in which he falls down precipices.

He is of a nervous temperament. His character is unequal. He experiences joys and sorrows without reason. He is irascible. Under the influence of any annoyance, he has the sensation of a ball ascending the whole length of his oesophagus. He sometimes bursts into laughter for some futile reason.

Examined from the view point of hysteria, he appears to have a slight concentric contraction of the right visual field. The corneal reflex is abolished on that side. Hearing is diminished on the right. Hypoaesthesia of the right side. Sensibility has disappeared over the right scapular region.

On admission on the 23rd. his temperature was $39.8^{\circ}\text{C}.$, 86 respirations per minute.

June 24th.- C.... has a livid face with cyanosis of the lips. Face covered with sweat. No œdema of the limbs.

The patient is restless and in the grip of a suffocating orthopnoea.

Temperature: $38.2^{\circ}\text{C}.$ Respiration: 85.

Pulse small and regular, 126.

The patient has a very abundant expectoration of sanguineous serum.

Auscultation reveals throughout both lungs short and harsh inspiration and expiration. Numerous fine subcrepitant râles, especially on the right side.

Auscultation of the heart is impossible; the cardiac sounds are masked by the respiratory murmur.

Pain on palpation in the right hypochondrium and in the right iliac fossa.

25th.- The condition has moderated a little. The patient has passed, during the twenty-four hours, 900 grams of urine, containing a little albumin.

About every ten respirations, the patient expectorates. The serous expectoration is very abundant and mixed with blood.

26th.- Urine: 1500 grams. Temperature: 37.6° C.

The dyspnoea has still further increased: 100 respirations per minute. The patient is sitting up in bed, his face pale with cyanosis of the lips and nose. Abundant perspiration on the face.

During this visit the patient seems to be choking. A venesection of 300 grams is immediately done. This relieves him and the expectoration decreases.

27th.- General improvement. The dyspnoea has diminished; the albumin has disappeared from the urine.

28th.- Improvement continues. At 2 in the morning, the patient had a profuse epistaxis.

The auscultatory signs and expectoration have disappeared.

The patient left the hospital on July 2nd, and has remained well since then.

CASE XXI. (Résumé).

Peritonsillitis, compression of the vagus.

Amblard: Gazette des hôpitaux, Paris, August 18, 1906,
vol. lxxix, p. 1119.

M.M., admitted June 4th, 1905 into the Vernois Ward, for an angina from which he has suffered for three days.

He is a negro, very robust, and without pathological antecedents. For three days deglutition has been painful; a condition of malaise has appeared, and he had a severe rigor accompanied by chattering of the teeth.

On admission, deglutition is very painful, the left side of the isthmus of the fauces is the seat of very intense pain, and the diagnosis of left phlegmonous angina is confirmed.

The patient speaks with difficulty, for the peritonsillar swelling has increased; and any movement of the tongue or of the temporomandibular articulation is almost impossible.

It is very difficult to get the patient's mouth open and to see the markedly oedematous uvula, pushed towards the right by the prominence formed by the left anterior pillar, the prominence being dull red and very painful.

Externally, the neck is slightly deformed, and an inflamed, swollen and painful gland is felt.

The tongue is white, the patient has almost constant nausea, and is very weak from two sleepless nights. Temperature, 39.8°.

The urine contains a slight trace of albumin.

Two days later, June 6th.— The patient has a somewhat severer dyspnoea, attributed to the swelling of the neck, which has greatly increased. The phlegmon is incised, and the general condition improves considerably. Temperature, 37.5°. The pulse is more regular. The urine is free from albumin. Nothing abnormal in the lungs or heart.

On the 7th, the patient considers himself cured.

The following day we learn with stupefaction, that at 11 o'clock at night, the patient, having got up for a moment, gave a cry of agony and collapsed. The house-surgeon was called; the patient who was visibly asphyxiating was given syrup of ether; then two injections, one of ether, the other of caffeine were

administered. A venesection and artificial respiration had no effect; the patient died.

At the autopsy, the cause of death was apparent directly the thorax was opened. Both lungs were invaded in their entire extent by considerable œdema. The fragments, of hard consistency, floated, but sank at the slightest touch. No other lesion of the lungs.

Nothing in the kidneys, heart, or central nervous system.

No trace of nephritis or degeneration of the hepatic cells could be detected under the microscope.

No lesions of the aorta or of the coronaries. No œdema of the glottis.

Simply, in the neighbourhood of the vasculo-nervous bundle of the neck, on the left side, the lymphatic glands were more voluminous, without, however, there being any œdematous infiltration and adenophlegmon.

In this case, it is easy to conclude that there was an irritation of the pneumogastric by the proximity of the inflamed lymphatic gland which compressed it in the cervical region.

CASE XXII. (Résumé).

Ether anæsthesia.

Poppert: Deutsche medicinische Wochenschrift, 1894, vol. xx, pp. 719-722.

Male; 46 years old; farm-hand; previous good health; no cough; long-standing alcoholic habit, moderate in degree for one and a half years previous to admission. Admitted May 20th; right inguinal hernia in the early stages of irreducibility and inflammation; medicinal treatment until May 31st.

Ether narcosis; radical operation for irreducible inguinal hernia; small abscess in the sac and recent adhesions; no perforations; wound packed; no bowel reduced into the abdomen. Narcosis lasted 30 minutes; 130 c.cs. of ether used; Czerny's, then Juillard's mask employed; anæsthesia slight; subconscious movement; slow, strong pulse; slight cyanosis; regular respiration; one emesis of gastric mucus; mucous râles toward the close of narcosis; prompt recovery and consciousness; no immediate complications.

After more than an hour increasing dyspnoea with loud râles, frequent cough, and much mucous sputum appeared. The pulse was so strong that no stimulants were needed, and the diagnosis was rendered of unusual accumulations of mucus, which the patient, being fully conscious, was expected to cough up for himself. Rapid heart failure with progressive cyanosis, accompanied by large quantities of blood-stained mucus in the nose, mouth, and throat, appeared, and death supervened during manifest œdema of the lungs.

The autopsy was held on the day of death and showed generalised peritonitis with recent adhesions, two intra-abdominal abscesses; hernial canal admitted several fingers; contents of sac adherent. Normal heart; slight aortic sclerosis; lungs slightly emphysematous, very markedly œdematous; the trachea and bronchi were filled with blood-stained, watery mucus. The other organs were normal. The cause of death was assigned as acute œdema of the lungs.

CASE XXIII. (Résumé).

Pregnancy associated with cardiac disease.

Vinay: Lyon médical, November 1, 1896, p. 289.

J.G., aged 32, had, when 18 years of age, an attack of acute articular rheumatism of all the joints, which caused her to have a double mitral lesion: incompetence and stenosis. Despite this, she was able to go successfully through two pregnancies. Both children, born at term, are alive.

The present pregnancy is the third, and the last period was from the 10th to the 15th September, 1895. Shortly after conception, she developed a bronchitis; at the same time she had some expectoration streaked with blood, night-sweats and anorexia.

May 16th, 1896.- Patient has dyspnoea on exertion and some epistaxis; oedema of the legs is fairly pronounced.

A few rhonchi in the lungs. Slight cough and scanty, white, frothy expectoration.

The apex-beat is in the fifth intercostal space in the nipple line; a harsh systolic murmur is perceptible there, conducted to the axilla and the back; the cardiac beats are tumultuous, with some missed beats. The pulse is regular, 80 per minute, with some missed beats.

The urine is pale, fairly copious, giving an abundant precipitate of albumin.

The uterus reaches to three or four finger-breadths above the umbilicus; the child is alive.

Treatment: Digitalis, cupping, laxatives, milk diet, rest in bed.

On the 24th., at 11 o'clock at night, the patient was suddenly seized while in bed with an attack of intense oppression; respiration was accelerated and difficult. Her face was anxious-looking, cyanosed, and at the same time numerous subcrepitant rales appeared in the chest. She was bled 300 grams; then 12 cups were applied to the back; injections of ether and caffeine; inhalations of oxygen.

It was in vain; the dyspnoea increased, the heart became irregular and greatly accelerated, then slowed down.

The patient preserved her intelligence till four o'clock in the morning, then agony supervened and she died

at half past five.

There had been no indication of the onset of labour, and directly death was certain, Caesarean section was performed, but the child had already succumbed, as is usual in such cases.

Autopsy.- Both lungs showed in their upper thirds the characteristic lesion of congestive oedema. The tissue was crepitant on pressure, it did not sink in water. Neither infarcts nor patches of bronchopneumonia. On section, there flowed out abundant, almost colourless, frothy serum. The inner surface of the bronchi was congested, and their lumen was full of frothy and reddish mucus.

Heart: Weighed 400 grams. The pericardium was healthy; the left auricle was enormously dilated; the myocardium appeared to be healthy. Mitral incompetence and stenosis. The valves were thickened.

Liver: 1500 grams; pale, fatty.

Kidneys: The right kidney weighed 140 grams; the left 130 grams; the parenchyma was very pale.

Uterus: 920 grams, showed nothing of special significance.

CASE XXIV.

Articular rheumatism.

B. Ball: Thèse d'agrégation, Paris, 1866.

C...., ward maid, aged 57, admitted on January 25th, 1865 into the Salpêtrière, under Charcot.

This fat, strong and plethoric woman has for some years been subject to fits of apoplexy with transient loss of consciousness.

For fifteen years she has been subject to attacks of articular rheumatism. For the last six days she has had pain the whole length of her legs, but chiefly in the knees, which are somewhat swollen. The hands are swollen, especially at the metacarpo-phalangeal joints. Slight fever, pulse 100 and irregular. A murmur is audible, of greatest intensity at the base of the heart.

From January to April, alternating remissions and exacerbations. Quinine sulphate, 2 grams; then Fowler's solution, 4 minims.

April 21st.- Very marked stiffness of the left knee, which is very painful.

April 25th.- At the morning visit an effort is made to overcome this stiffness, and during this attempt the patient resists violently.

Ten minutes later, a sudden attack of oppression occurs, a slight hæmoptysis takes place, and the patient is seized with all the symptoms of imminent asphyxia. Abundant froth comes from her mouth and nostrils, she becomes pale and cold, and dies at the end of five minutes.

Autopsy.- Voluminous lungs, very congested and showing considerable oedema.

Heart: mitral stenosis, some small vegetations on the auricular aspect of this valve and on the semilunar cusps of the aortic valve.

Brain absolutely healthy. Other viscera without appreciable alteration.

The left knee, which was the worst during life, showed but slight changes; the cartilages were not altered, but there was a severe synovial infection.

The right knee, on the contrary, showed at various places a very marked velvet-like alteration.

CASE XXV. (Résumé).

Typhoid fever.

Fouineau: Thèse, Paris, 1898, p. 199.

A labourer, J...., aged 42, was admitted into the Jenner Ward on July 25th, 1897. He had measles at 7 years of age; acute articular rheumatism, for three weeks, at 13. At 19 and 27, he had fresh attacks of rheumatism. Aortic incompetence has existed since then.

J.... has complained, since that time, of oppression on exertion, vertigo, palpitation. There has never been œdema of the limbs nor hepatic pain. No syncope, nor symptoms of angina of the chest. Existence was possible if all overwork were avoided.

Nine days ago he was compelled to take to bed; he had lost his appetite, and was extremely fatigued; headache, epistaxis; increase of attacks of giddiness and exhaustion. Fever and fairly abundant diarrhœa.

On admission he had a temperature of 39° C., extreme weakness, slight abdominal meteorism, gurgling in the right iliac fossa, some lenticular rose-spots on the abdomen, on the inner side of the thighs, and on the back, and tremor of the arms.

Some discrete râles of bronchitis in the lungs. Diastolic murmur over the aortic area. Pulse 110.

Urine scanty and dark. Golden yellow diarrhœa. Troublesome insomnia.

Treatment: Six cold douches per day, 40 centigrams of bromohydrate of quinine, cordial drink, and milk diet.

To sum up, J.... had typhoid fever of moderate severity, the temperature oscillating between 39° and 39.8° C.

On the morning of the fourteenth day, the situation suddenly changed; the pulse of 110 increased to 170; face pale and covered with sweat; limbs violet coloured. The temperature was now no more than 37.5°. The lungs were invaded by a shower of crackling râles characteristic of œdema and pulmonary congestion. The respiration was very frequent; the tongue dry; carphology. Constant fits of coughing and mucous expectoration tinged with blood, almost filling two spittoons. The urine was slightly albuminous.

Bled 250 grams at 11 o'clock, and subcutaneous injection

of 5 centigrams of sulphate of sparteine.

From 9 a.m. to 6 p.m. the condition became graver: cardiac collapse, the expectoration stopped suddenly and the cough disappeared. Extremities cold and violet coloured, face covered with sweat; temperature 40.5° , and J.... died from asphyxia at 10 p.m.

Autopsy.- Lungs swollen, emphysematous along their anterior margins. No pleural adhesions. The lungs are violet coloured, resistant under the finger. On section, an abundant quantity of reddish serum escapes, as if from an over-full sponge. The pulmonary parenchyma does not crepitate.

On the right side as on the left, only this single lesion is found: pulmonary oedema and congestion pushed to an extreme degree. The trachea and bronchi are full of the reddish froth which the patient expectorated.

The heart is hypertrophied, especially the left ventricle. No pericardial adhesions; myocardium healthy. The aortic valves are sclerosed, incompetent. The coronaries are patent.

The aorta, of normal calibre, shows some patches of fatty degeneration.

Liver normal.

Spleen enlarged, soft and diffluent.

The ileum shows Peyer's patches, lesions which become accentuated as the ileo-caecal valve is approached. These patches are ulcerated, with irregular floors. The edges are irregular and undermined. Follicular hypertrophy with central ulceration, more pronounced in the large intestine. The peritoneum shows no alteration anywhere.

CASE XXVI. (Résumé).

Pneumonia.

Tonnel: Écho médical du nord, Lille, June 27, 1897.

A woman, aged 64, admitted into hospital February 18th, 1895.

Nephritis with oedema of the legs appeared a year ago.

February 2nd.- Oppression appeared during the night. A focus of pulmonary oedema was detected in the right supraspinous fossa.

Urine contained 3.5 grams albumin per litre.

February 18th.- Commencement of a right-sided pneumonia.

February 23rd.- Signs of pneumonia accentuated. Dyspnoea more intense. Albumin persisting.

February 26th.- Suddenly, after a sensation of tickling in the throat, the patient developed a constant cough, and expectorated about three-quarters of a litre of lemon-coloured fluid, mixed with rusty greyish sputum. The patient had intense orthopnoea. No cyanosis. She was pale. The heart was irregular, and beat feebly and rapidly.

The patient, as soon as the attack had passed, after about an hour, fell into a state of complete prostration.

February 27th.- Whilst during the attack the chest was full of sibilant râles and both coarse and fine subcrepitant ones, we now found on auscultation large moist râles localised to the middle lobe of the right lung and some small dry râles in the rest of the lungs.

Urine: 3 grams albumin per litre.

The temperature, 38° C. before the attack, 38.2° during the attack, fell to 36.8° after.

March 27th.- The patient died.

Autopsy: Lungs oedematous, except the middle lobe of the right lung which showed lesions of pneumonia.

Kidneys: Irregular and deformed. Small cysts. Lesions of mixed nephritis, observed under the microscope.

CASE XXVII.

Chronic bronchitis and emphysema.

M.J., a widow, aged 67, who used to attend at my surgery as a sufferer from chronic bronchitis and emphysema, sent for me about one o'clock in the morning of December 10th, 1926 with a message to come immediately as she was dying. On arrival, I found her in the throes of an asphyxial state with the addition that she kept bringing up, without effort, the abundant, frothy, pinkish, watery sputum characteristic of acute pulmonary oedema. She was sitting up in bed, cyanosed and cold. Her pulse was strong and rapid, and there was no pyrexia. In addition to the usual wheezing rhonchi, the chest was full of moist bubbling and crackling râles. Having ordered hot-water bottles to be applied to her lower extremities, I gave her a hypodermic injection of atropine, gr. 1/50, and then carried out a liberal venesection from her left median basilic vein, withdrawing 15 ounces of blood. The result was most gratifying. Relief came very quickly. Before leaving her, I gave her a dose of brandy in water to help her to sleep. I did not wish to give her morphia owing to her old chest trouble. I took back with me some of the expectorated fluid, and slowly added a portion of it to a little pure nitric acid in a test-tube. An opaque white ring formed at the junction of the two fluids, revealing the presence of albumin.

When I called again later in the day, the patient seemed little the worse for her previous night's experience. Her only complaint was of dryness in the throat, no doubt due to the atropine. Though she felt she could get up, I encouraged her to remain in bed for the rest of the day.

A few days later, on December 14th, she called at my surgery. I then noted, as on previous occasions, the presence of sibilant rhonchi and prolonged expiration, due to her old-standing bronchitis and emphysema. The cardiac sounds were faint, but her pulse was firm and regular. Her blood pressure was 165/110. The urine had a specific gravity of 1012, and contained a trace of albumin.

Although this patient had had before attacks of nocturnal dyspnoea, regarded as "asthmatic," she had not previously had such a violent seizure; neither had she before brought up sputum so copiously. She could not account for the attack as she had been feeling in her usual

health during the preceding day, and had not had a heavy supper. Nor was she constipated. The attack supervened quite suddenly, awakening her from her sleep.

CHAPTER XXVIII.

Thoracentesis.

R.T. Binns: Medical Journal of Australia, Aug. 9, 1924, pp. 141-142.

A woman, forty years of age, was admitted into the Adelaide Hospital suffering from acute nephritis. Four weeks before her admission she contracted a sore throat, swollen glands in the neck and a few days later a rash on her arms and thighs. The rash disappeared in four days and peeling of the hands, feet and abdomen began later. The patient had evidently had an attack of scarlet fever. When she first got out of bed after two weeks she noticed that her legs were swollen and that her face and eyelids were oedematous. The amount of urine passed during her illness had been much less than normal. She had not received medical attention until four days before she was admitted. She had not had any serious illness before. She was a mother of three healthy children.

The patient was a rather stout, middle-aged woman. On the day of admission her temperature, pulse rate and respiratory rate were within normal limits. There was very slight oedema around the eyes; the glands behind the angles of the jaw were swollen and tender; the throat was inflamed. The only abnormality discovered on examination of the heart was that the second sound at the base was accentuated.

Physical signs in the chest indicated the presence of free fluid in both pleural cavities, extending upwards as far as the angles of the scapulæ. No free fluid was detected in the abdomen, but there was considerable tenderness in the region of the kidneys. Both feet were oedematous. The urine contained a high percentage of albumin and some blood. By microscopical examination of the urine blood cells, epithelial and blood casts were detected. The systolic blood pressure was 180 mm. and the diastolic 130 mm. Hg. The patient's condition was diagnosed as acute nephritis complicating scarlet fever.

On the fifth day after admission the amount of fluid in the pleural cavities had increased to such an extent as to embarrass breathing. Aspiration of the pleural cavities was indicated. Slightly more than a litre of fluid was drawn off from the left side. This temporarily relieved the dyspnoea. Two hours after the aspiration

the patient suddenly began to cough and a large quantity of frothy serum was expectorated. The breathing became very rapid and shallow and was accompanied by "bubbling" sounds. The face, lips and extremities became very cyanosed and the patient soon lost consciousness. The pulse rate was very rapid. A venesection was immediately performed and 450 c.cs. (15 ounces) of blood allowed to escape. Atropine and cardiac stimulants were given and also continuous inhalations of oxygen. Two hours after the attack the breathing had become less rapid and quieter and the cyanosis had disappeared and the pulse rate become slower. The patient had evidently recovered temporarily from the attack of acute pulmonary oedema. Three days later, however, the temperature rose to 39.40° C. (103°F.), the pulse rate to 150 and the respiratory rate to 50. She complained of thirst and expectorated offensive sputum. The lungs had now become infected and she succumbed on the following day to the complication of pneumonia.

The attack of acute pulmonary oedema occurred in this case two hours after a paracentesis of the pleural cavity. The immediate dangerous symptoms were relieved temporarily by the venesection.

CASE XXIX.

Idiopathic. Sudden death.

I was called shortly after four o'clock one morning to a patient who, on my arrival, I found to be recently dead. A small quantity of froth was present at his mouth and nostrils. I elicited the following information. The man was a tailor, aged 48, who had always been in good health and of temperate habits. He went to bed the night of his death, after an ordinary day's work, without having made any complaint whatsoever. About 4 a.m. he suddenly awakened his wife by shouting he could not breathe; he jumped out of bed, crossed the room, collapsed and died within a few minutes. I carried out a post-mortem examination for medico-legal purposes.

He was a well-nourished individual with a tendency to obesity. The brain appeared normal. On opening the chest, one was immediately struck by the appearance of the lungs. They seemed very bulky and congested, and showed the markings of the ribs on their surface. The pleural cavities contained a quantity of watery, sanguineous effusion. There were no pleural adhesions, the lungs being quite free. The anterior borders of the lungs crepitated softly when pressed between the fingers. On cutting the lungs, a pinkish, serous, frothy fluid flowed out even without pressure. The bronchi and trachea were full of pinkish froth. Cut pieces of the lung thrown into water floated.

The heart was well covered with fat, somewhat in excess of the normal. However, it was not enlarged. Its muscle appeared healthy, and the valves were competent. The aorta showed no evidence of disease, and the coronary arteries were patent. One remarked on the healthy appearance of all the abdominal viscera.

No other cause of death could be found than that of œdema of the lungs. This must have been of such an acute nature (bronchoplegic form) that it killed the man before he could disencumber the engorged organs by expectoration.

CASE XXX.

Idiopathic. Recurrent attacks.

T. Lissaman: Lancet, Feb. 8, 1902, pp. 366-368.

I am fortunate, or unfortunate, as the case may be, to have under my care a patient who has suffered from this affection (acute suffocative pulmonary oedema) for two and a half years. She is a married woman, aged 45 years, with five children. She is stout and well nourished, with no traces, as far as I have observed, of any organic lesion of the heart, kidneys, or lungs, or, indeed, of any organ. She first came under my notice in June, 1899. I found the patient sitting up in bed, leaning forward with her head almost touching her knees, which were raised up, suffering from most terrible dyspnoea. Her face was livid; she was bathed in cold sweat; her breathing came in short, sharp gasps; and with each expiration she ejected, without any apparent effort, a thin pinkish fluid. She was apparently dying. The remedies which I then applied were an injection of one ounce of brandy with gruel into the rectum - she was far too distressed to swallow anything - the application of hot-water bottles to her feet, and the free application of very hot fomentations to the præ cordium. The pulse was small and very rapid, the respirations were from 30 to 40 per minute, and the temperature was normal. Loud moist sounds were heard all over the chest. The amount of expectoration was very great. After four or five hours there was some improvement in her condition and she was able to swallow. I administered at intervals of 15 minutes two drachms of sweet nitre diluted with water. She was apparently very much relieved after three doses of this remedy. On the following day she seemed to be little the worse for the attack with the exception of being languid and tired and disinclined for exertion. I prescribed rest in bed for a day or two and gave her a mixture containing bromide of potassium and camphor. I ceased attendance on the third day as she seemed to be all right. The urine was not examined. Four days afterwards, on July 3rd, I was hurriedly sent for about the same time in the evening, and there was a repetition of the above-described condition, with the same result. After this second attack I made a very thorough examination of the

patient, but could find no organic trouble of any kind. The urine on this occasion was not examined, but I gave directions that a sample should be retained every day, and that if the attack recurred the sample of urine passed immediately before the attack should be sent to me. On July 30th the patient had a third attack, and the urine was examined, but nothing abnormal was detected in it. She had 20 attacks in the next 12 months.....

In March last I made up my mind that I would treat the next attack with chloroform, having already tried nitrite of amyl with unsatisfactory results.

I commenced the administration of chloroform in fear and trembling as to the result. I administered it in the ordinary way on a handkerchief, giving the patient a whiff of chloroform and a breath of air in alternate inspirations. The gasping, short nature of the breathing, and the obvious obstruction due to the frothy material in the air-passages prevented any great amount of chloroform vapour being introduced into the lungs. However, after from 10 to 30 minutes of administration in this way a marvellous change came over the patient. The lividity disappeared, the breathing became easier, the action of the heart became far less laboured, and the respirations slowed down, the patient lying back on the pillows apparently asleep. This condition lasted perhaps for an hour, when I roused her, and she expressed herself as feeling all right. So striking was the relief afforded that my patient begged me to leave the remedy with her in future, so that she could administer it to herself in case of need. I would not consent to this at that time, as I feared that carelessness in giving the vapour in too concentrated a form might end in disaster. During the next three months she had 23 attacks, at each of which I administered chloroform, with the invariable result that the attack was stopped in from 20 to 30 minutes..... I, having lost all fear of any ill-effect from the judicious administration of chloroform in her case, devised the following method for her husband to carry out. I gave him two drachms of chloroform with instructions to put in an ordinary tumbler a few square inches of clean blotting-paper, to pour the chloroform on the blotting-paper, and to hold the tumbler upright under the patient's mouth and nose, but to be very careful that she did not take more than three inspirations from the tumbler without one intervening inspiration with it away..... The attacks now, from beginning to end, with the administration of chloroform at the earliest possible moment, rarely last half an hour, and my patient appears none the worse the day following them. She has had no

medicine for many months. I have notes of 72 attacks in the two and a half years during which she has been under my care and they were all as instantaneous in their onset as an epileptic fit. They varied somewhat in intensity and duration from time to time, but they were all characterized by these constant phenomena: suddenness of onset, intense agonising pain in the chest, frightful dyspnoea, lividity of the face, profuse cold sweat all over the body, laboured action of the heart, with a small rapid pulse, and, lastly, but most important, the ejection (one can hardly call it expectoration, for that implies coughing, which is almost absent) of enormous quantities of thin pink, sero-mucous fluid. I have measured as much as 42 ounces of this fluid in an attack lasting eight hours. I have never detected rise of temperature in the rectum or vagina during an attack. They are almost invariably preceded by a desire to go to stool and the dyspnoea always comes on then during the act of defecation. They very often occur about the period of menstruation, especially if that is delayed a few days, as it often is, though it is normal in other respects.....

She seemed to be drifting into a condition of imbecility, like an epileptic does, until the systematic administration of chloroform stopped the violence of the attacks. She still has attacks - about two a month (instead of three or four a week as in March last) - it is true, but as the duration of them is so short and the remedy inspires such confidence she feels sure eventually she will completely recover.

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