

absence of an apex beat, because the dilated right ventricle, lying in front of the left one, acts as a water cushion or buffer in preventing the apex from reaching the thoracic wall; while the absence of distinct pulsation in the scrobulus cordis, and at the lowest part of the sternum, proves that the heart is feeble, and that in the right side dilatation is the prevalent lesion.

Congenital constriction of the pulmonary artery may occur at various periods of intra-uterine life, and the mechanical results on the cardiac development and consequent symptoms must vary accordingly. When it occurs just previous to the completion of the ventricular septum, and to such an extent as to prevent the complete formation of that septum, we have as a natural result the phenomenon of a complete double circulation with free intercommunication between the two ventricles, one of which is connected with an obstructed and the other with a patent arterial conduit. The natural result of this is, that when both ventricles contract with nearly equal force upon nearly equal contents, that one whose natural outflow is obstructed forces a portion of its contents through the abnormally patent channel into the other ventricle whose outlet is unimpeded; and the consequence of this is, forcible dilatation of the unimpeded outlet—in this case the aorta,—and consequently a systolic murmur of tension accompanying the blood wave and followed by a loud accentuated aortic second, the natural result of the forcible closure of the aortic semilunar valves by an unusually heavy column of blood, precisely the usual condition in this case. When, however, from any cause—such as catarrhal congestion—the pulmonary circulation is more than usually obstructed, the result is the same as in the normal condition: the blood-recoil upon the pulmonary valves is greatly increased, and we have an apparent and temporary transference of the accentuation from the aortic to the pulmonic second sound—a phenomenon which repeatedly occurred in this case while the patient remained under observation.

As to the systolic murmur in the mitral area, the absence of distinct propagation round the left side to the back is opposed to the idea of any regurgitation through the mitral valve. The conditions necessary for the production of this regurgitation were, moreover, entirely absent: because free communication between the ventricles prevents the possibility of over-dilatation of the left ventricle and regurgitation from this cause; while congenital mitral constriction, which might give rise to it, would not only be a very unusual complication in such a case, but is also conclusively disproved by the entire absence of any evidence of dilatation of the left auricle, which in all such cases is sure to be revealed by distinct pulsation to the left of the pulmonary area and in the same plane, evincing dilatation and hypertrophy of the left auricular appendix. The systolic murmur over the left apex is therefore due to propagation from the aortic and pulmonary regions, and possibly also to propagation from the opening in the upper part of the ventricular septum, which I have shown to exist.

In this most interesting case, therefore, there exists contraction of the pulmonary artery above the valves, with deficiency of the interventricular septum at its upper part, dilatation of the right ventricle with destruction of the venous valves at the root of the neck, and dilatation of the ascending aorta; and these conditions may be accepted as facts conclusively proved by the phenomena present. But there is one other malformation the presence of which is rendered probable by what we know of the sequential development of the heart, though it is not, and, so far as I know, cannot be, revealed by any diagnostic phenomena—and that is, more or less imperfect occlusion of the foramen ovale. Not only, therefore, has it been shown that in this case the symptoms depend upon congenital malformation—an opinion which even a cursory inspection of the patient would lead almost everyone to adopt,—but it has also been shown that the physical signs present confirm this view, and that these are precisely such as our knowledge of the sequential development of the heart and of the mode in which these physical signs are produced would lead us to expect to be present in a case of pulmonary constriction arising at a certain period of intra-uterine gestation. The ease with which we can now interpret the symptoms and signs in a case so complicated as the present, compared with the difficulties which formerly beset the diagnosis of the simplest valvular lesion, exhibits in a most striking

manner the very great advance in the diagnosis of cardiac disease which has taken place during the last thirty years.

I have only to add that, under appropriate treatment, the patient left the infirmary improved in every respect, and that she still remains under observation.

Edinburgh.

ON THE COMPARATIVE ACTION OF ALCOHOL AND ABSINTHE.

By DR. MAGNAN,

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PATIENTS affected with acute alcoholism may offer the following peculiarities:—

1. Some of them present convulsive phenomena, which nothing in the previous symptoms allowed us to foresee. The patient, in fact, all at once loses consciousness, and suddenly falls; the face becomes pale, the limbs stiffen, and the muscles of the trunk and neck contract, so as to turn the head to one side. To this stage of tonic spasm soon succeed clonic spasms, twitchings in the arms and legs, with grimaces of the face, and convulsive movements of the eyes; frothy saliva, occasionally tinged with red, covers the mouth, and the tongue is bitten; then the lips grow blue, the face livid, and the breathing stertorous, whilst the urine and fæces escape involuntarily; a more or less prolonged state of stupor following the fit.

We have here, it is evident, an attack of epilepsy. Such a fit may occur equally at the periods of onset, of full establishment, or of the decline of the attack of delirium tremens—that is to say, in stages of the attack when the trembling is very violent, or, on the other hand, very slight. After the attack the trembling follows its usual course: it diminishes if the attack of delirium tremens is on the decline; it increases, on the contrary, if the fit has come on at the earlier part of the attack.

The fit, then, is not the highest expression of the motor disturbance, nor is it the most advanced degree of that general tremulousness which accompanies delirium tremens; it is an accident of a different order, superadded to the other disturbances of motility, and due to a distinct cause, which from the antecedent history is usually absinthe, more rarely bitters, vermouth, or *vin blanc*, the adulterated white wine which is sold retail in Paris by most of the wine merchants, and is very different from that drunk in the neighbourhood of Vignobles.

An acquaintance with this fact is not unimportant either from a diagnostic or therapeutic point of view; and perhaps bleeding would have been used more moderately for patients affected with epilepsy wrongly called “alcoholic,” if practitioners had been thoroughly impressed with the idea that the attack was due to the direct action of a poison which possesses the special property of stimulating the excitomotor functions of the medulla and spinal cord, without the necessary intervention on the part of the brain of that determination of blood to which certain writers have ascribed the principal rôle in the production of convulsive seizures.

To sum up, the epileptic attack is an exceptional phenomenon due to a special cause, and independent of the degree of trembling.

2. In certain rarer cases, in patients suffering from acute alcoholism, we observe the occurrence of an attack of delirium which presents all the general characters of alcoholism—that is to say, hallucinations of very changeable character, of painful, disagreeable, and aggressive nature, recalling, it may be, either the profession of the individual or the dominant preoccupations of the time of attack. But with this characteristic delirium the trembling in this class of patients is next to nothing; it may even be entirely wanting, so that the attack of acute alcoholism may be found reduced simply to intellectual disturbances. Cases of this kind have been on record for some years past. M. Motet relates some observations of the kind in his thesis of 1859 “On Alcoholism, and the Poisonous Effects produced in Man by the Liqueur Absinthe.” In his cases the patients were especially drinkers of absinthe.

How are we to explain this rapid evolution of delirium in the absence of motor disturbances? It is that absinthe acts in the same way as belladonna, henbane, datura, and haschisch (*cannabis Indica*), and does not require, like alcohol, to prepare its way, for, as is shown by physiological experiment, it can rapidly give rise to hallucinations and delirium before the alcohol contained in the liqueur of absinthe has had time to produce trembling in man. We have here, then, a second fact—viz., the premature appearance of hallucinatory disturbance without trembling—a fact which, from clinical observation alone, we might attribute to the absinthe, and of which physiological experiment gives us the confirmation.

3. In the greater number of cases of patients affected with acute alcoholism, the trembling and intellectual disturbances progress simultaneously, or present only slight differences in their successive evolution. These patients, according to what we can ascertain about them, have usually wine or brandy only to excess.

The present communication has for its object to furnish experimental proof of the facts taught us by clinical observation.

Before showing the action of essence of absinthe on the nervous centres, it is needful to recall in a few words the mode of action of alcohol. Let us observe, for example, what takes place in a dog submitted to the poisonous action of this agent. Some minutes after the administration of the alcohol the animal leaps, yelps, and runs in every direction, then staggers and becomes stupefied; the paws, especially the hinder ones, cross each other and give way under him. The paralysis at first of the hind quarters soon reaches the fore part of the body; the animal falls into a state of complete relaxation and comatose sleep; when raised he sinks like a lifeless mass, all energy and elasticity being lost. The temperature is lowered, sensibility is abolished and cannot be aroused, in cases of complete intoxication, by the most active excitants. These symptoms are reproduced in the same animal every time it is submitted to the action of alcohol, and during ten or twelve days we do not see any new symptoms supervene; there are no alterations in the general behaviour, no illusions or hallucinations, and no epileptic or epileptiform attacks. But if one continues to administer a daily dose of alcohol, sufficient to bring on intoxication, one remarks in the dog from about the fifteenth day a nervous excitability of quite peculiar character. The animal is melancholy and uneasy; he listens, the least noise makes him start; when the door is opened, seized with fright, he runs and crouches in the darkest corner of the room; he no longer responds when patted, he runs away and tries to bite when one attempts to take hold of him, and utters sharp cries at the mere threat of blows. This irritable and timid condition increases each day, and from the end of the first month, illusions and hallucinations becoming added to it, it is transformed into a veritable delirium. In the middle of the night he utters plaintive moans, or even whilst all is quiet he begins to bark, the cries becoming louder and more frequent, as if an enemy were approaching; speaking or calling does not reassure him—one must interfere with a light. At last, during the day he growls without cause; then thinking that he is pursued, he cries out, runs scared hither and thither, with his head turned back, and snapping in the air.

In some cases the hallucinations and delirium are the causes of fatal accidents, the animal in its flight may fall out of a window or down stairs exactly like a drunken man. With these intellectual troubles one sees, from the second month, a trembling appear, which is at first limited to the paws, but gradually becomes general, and reaches the muscles of the trunk and head. This trembling sometimes persists for twelve hours in the day, and follows each fresh dose of alcohol; it presents a rhythmic type, with short and somewhat rapid oscillations, is of variable intensity, stopping for a few moments, and is complicated, moreover, from time to time by tremors in small isolated groups of muscles. But never, in the experiments with alcohol, is an epileptic attack produced, whilst this, as we are about to make evident, is the principal manifestation of poisoning by absinthe.

In a weak dose, essence of absinthe induces a more or less marked muscular tremor, little abrupt jerking shocks, like electric discharges, repeated one or several times, in the muscles of the neck, and giving rise to rapid and very

limited movements of the head, which is carried upwards and backwards; the contractions reach in succession the muscles of the shoulders and the back, and then provoke stronger abrupt contractions, which raise, by jerks, the anterior part of the body. This action of absinthe, more especially directed to the head and neck—that is to say, its influence upon the bulbo-cervical region of the cord, is the more remarkable because alcohol acts in the reverse order. The latter, in fact, as we have seen, produces at the onset paraplegia, before paralyzing the anterior parts of the body; absinthe, on the contrary, provokes spasmodic contractions in the fore-quarters before producing generalised convulsions. And this is not all: sometimes one sees a very interesting phenomenon come on in the dog; the animal stops all of a sudden, stays motionless as if stupefied, with the head low, a dull look, and the tail hanging down. It keeps this attitude for thirty seconds to two minutes, and then spontaneously regains its habitual appearance. This is a vertiginous condition, which is not without analogy to the *petit mal* or “absence” of the epileptic.

The action of the essence of absinthe in a large dose is different, or, rather, it is a higher degree of intensity of the phenomena.

After prodromata analogous to the occurrences of which we have just spoken, or even abruptly and more or less rapidly, according to the mode of introduction of the poison, epileptic attacks come on: the animal loses consciousness, falls, and stiffens in the tonic convulsions which form the first stage of the fit. Most frequently the extensor muscles of the neck and back contract energetically so as to produce opisthotonos; but with this one almost always remarks a slight pleurosthotonos either to the right or to the left. Sometimes, in place of an extension of the head, it is flexion which is in excess, and which may by a forcible emprosthotonos make the animal turn over itself. To the tonic convulsions succeed, after the lapse of a few seconds, clonic convulsions, with snapping of the jaws, foam (sometimes bloody) on the lips, and biting of the tongue; and one sees the evacuation of urine and fæcal matters, and even of semen, occur in some cases. The attack over, the animal continues in a state of slight stupefaction for a brief interval. The attacks of epilepsy show themselves sometimes with this *ensemble* of symptoms, and leave intervals between them of ten or twenty minutes, or even longer.

The action of essence of absinthe, however, does not reveal itself only by a stimulation of the excito-motor power of the nerve-centres; it shows itself also by intellectual disturbances; and, acting in an inverse mode to that of alcohol, which requires a certain time to produce delirium, this substance gives rise to hallucinations at the first onset, without previous preparation, in an animal which up to that time was free from every ailment. In fact, what we see in the dog, in some cases, after intravenous, subcutaneous, or stomachal injections of essence of absinthe, is as follows:—In the interval between two epileptic attacks, and sometimes before the convulsive symptoms, or even without convulsions, the animal is seized with an attack of delirium. All of a sudden he erects himself on his paws, the hair bristles, the look becomes wild, the eyes, injected and brilliant, staring at some particular spot where there is nothing apparent to draw his attention; he barks furiously; advances and retires as before an enemy; with open mouth, he throws his head suddenly forwards, and immediately shuts his jaws and shakes them from side to side as if he wished to tear his prey in pieces. This attack of delirium may recur several times; then the effects pass off, and the animal becomes quite calm.

It is not necessary to insist here upon the parts which belong to the spinal cord and to the brain respectively in the production of this group of symptoms. It will be sufficient to call to mind that, after section of the cord below the medulla oblongata, we obtain separately and at different times an attack of bulbar epilepsy (head, eyes, mouth, and face), and an attack of spinal epilepsy (limbs, trunk, and neck). On the other hand, after the removal of the cerebral hemispheres, the convulsions are produced in the same manner. Lastly, we have seen that the delirium may show itself without convulsive seizures. All these are so many proofs demonstrating that the cerebro-spinal axis in its entirety concurs in the production of epilepsy; but that each one of the parts may be influenced separately, and may give rise to a determinate group of symptoms. We will add,

moreover, that the essence of absinthe is a valuable agent for the study of the mechanism of epilepsy, and for the observation of the changes in the cerebral or retinal circulation which accompany the different periods of the attack.*

CASE OF
PRIMARY SCIRRHUS CANCER OF THE
LARYNX; TRACHEOTOMY.

By EDWARD J. COOKE, B.A., M.D. T.C.D.

JAMES B—, aged fifty-eight, came under my care while residing at King's Lynn in July, 1872, and gave me the following history of himself. For several years he had suffered on slight provocation from severe colds and sore-throat, attended with aphonia, which conditions were daily becoming more aggravated. He was very weak, and had some difficulty in swallowing, together with a fixed pain behind the thyroid cartilage; cough and expectoration troublesome, the phlegm often mixed with blood; loss of appetite. No history of syphilis or phthisis, but had in his early years been addicted to habits of intemperance.

I prescribed iodide of potassium in ten-grain doses three times daily with bark, also one tablespoonful of cod-liver oil once a day. Locally I applied a strong solution of iron and gargles of tannic acid with some good effect. Later on I found most benefit from the daily application of a forty-grain solution of the nitrate of silver to the ounce of water. In September he went to London, and became an out-patient at the Victoria-park Hospital, where, in addition to my internal treatment, he was ordered the iodine spray, which he continued for about six weeks, when his condition became so serious that he was advised to return home immediately, which he did on Nov. 14th, but I never heard exactly what his medical adviser thought of him. I saw him on the evening of his return; he was quite exhausted by his journey; the aphonia had increased, and swallowing was almost impossible. I prescribed an antispasmodic mixture, hot sponges to be applied to the throat, and the room to be kept warm by means of steam vapour.

Nov. 15th.—Had a wretched night; symptoms most alarming. Had an immediate consultation with my colleague, Dr. Lowe, and we agreed that tracheotomy was urgently demanded, as affording the only chance for my patient's safety.—2 P.M.: Respiration 64; pulse 140. I proceeded to open the trachea, without chloroform, and was somewhat hindered towards the latter part of the operation by the hurried breathing, which rendered any fixation of the trachea difficult even for a moment. After cutting upwards through three or four rings of the trachea, air began to enter, but was much impeded by quantities of purulent mucus, which almost filled the opening, so much so that it was with much difficulty the tube was first introduced, and had to be extracted immediately, as the patient's breathing was so obstructed for a few minutes by the cause above-mentioned. After a few minutes the tube was again introduced, and retained in position by means of tapes round the neck. The hæmorrhage, which was of a venous character, was slight. I was ably assisted during the operation by Dr. Lowe and Mr. Plowright. The latter kindly remained with the patient for several hours, and was constantly employed in keeping the tube free.—8 P.M.: Pulse 100; respiration 32; expectoration less; slept at intervals.

16th.—Slept several hours during the night. Pulse 96; respiration 30. Deglutition less difficult; took quantities of beef-tea, eggs, &c. Wrote on the slate he felt more comfortable than he had done for months. During the morning the tube slipped out three times; eventually I put in one larger, and with a sharper curve, and had no further trouble with it.

21st.—All conditions improved. Pulse 86; respiration 28. Sleeps most of his time.

30th.—Wrote on the slate that he felt much better. Can speak a little when his finger is applied over the opening of the tube. Can swallow freely.

It would be tedious to relate his daily history, and it will be sufficient to state that he continued to improve for about five weeks, when a small and painful abscess appeared in the thyro-hyoid space, which I opened on three occasions. This led me still more to regard the case as one of diseased cartilages. The laryngoscope from the first gave no accurate evidence of the true condition of the parts affected beyond turgescence of the epiglottis and vocal cords, the larynx being so sensitive as to render careful examination impossible. This small abscess now disappeared, and was soon followed by pain about the artificial opening, as if something pressed on the tube and also hindered deglutition. On removal of the tube, I found it was pressed out of its course by what seemed to be large unhealthy granulations. These I reduced by application of nitrate of silver. After this he again took large quantities of nourishment—port wine, &c.—and sat up daily; could eat an apple, and seemed to gain much flesh. About the end of March he felt much pain in the right parotid region, followed by a very tense swelling, which I poulticed; this gave some relief. Perspires very much; tendency to a low form of bronchitis. Towards the end of April I opened this swelling, but got no matter, only serum. The opening healed rapidly. On July 1st saw him for the last time. He was much weaker, but sat in the open air when weather permitted. About the middle of July his wife wrote to inform me he was unable to leave his bed; all fluids passed through the tube. He now suffered very little pain till the night before he died, which was passed in great suffering, and he expired August 15th, exactly nine months from the date of operation.

Notes of the post-mortem appearances were supplied me by the kindness of Mr. Plowright, then house-surgeon to the Lynn Hospital. There existed the remains of an extensive abscess of the right side of the larynx, reaching upwards to the angle of the lower jaw, "which was roughened"; the vocal cords were destroyed, the upper portion of the larynx being grown up by what both Dr. Lowe and Mr. Plowright pronounced to be a "scirrhous mass"; the abscess also extended downwards into the chest, and it was surprising how any one could have lived so long with such a mass of complicated disease in his larynx, which was all but impervious. The thorax was not examined.

Remarks.—The post-mortem examination revealed a state of things which up to my last visit was not suspected, as I looked on the disease as strumous ulceration of the cartilages, and regret that repeated attempts to examine his larynx failed, owing to the extreme sensitiveness of that organ, and later on to his inability to open his jaws wide. The case cannot fail to be of interest to the profession, not only from the prolonged existence of the patient, but also from the fact that cancer is happily a rare primary disease of the larynx, only a few cases having been demonstrated during life by Dr. Walker, of Peterborough, Sir Duncan Gibb, of London, and Dr. Johnson; that it generally exists in the epithelial or medullary form, and, according to the most reliable authorities, chiefly as a secondary disease. As far as the operation is concerned, this case undoubtedly proves, with many others, that, even under the most extreme circumstances, tracheotomy often furnishes most encouraging results, the greatest danger being delay.

Sheffield.

SLIGHT INJURY TO LEG; SUDDEN DEATH;
THROMBI IN RIGHT VENTRICLE
FROM POPLITEAL VEIN.

By A. B. SHEPHERD, M.A., M.B.,
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THE following case presents many points of similarity to those lately published in THE LANCET by Surgeon Thomas Browne, M.D. (June 27th, 1874, p. 901), and Mr. Geo. G. Gascoyen (Aug. 8th, p. 189). For the life-history I am indebted to Dr. Stewart, of Southwick-street; for the post-mortem notes I am entirely responsible.

R. G—, aged forty-nine, a healthy, spare, though largely-made man, of active and temperate habits, slipped, on June 1st, 1874, through the rounds of a ladder, and

* Magnan: Recherches de Physiologie Pathologique avec l'Alcool et l'Essence d'Absinthe. "Epilepsie" (Arch. de Phys. Normale et Pathol., Mars et Mai, 1873), &c.