gains nearly its normal standard. Sometimes this improvement is permanent, but often the anæmia returns at some later date, under the influence of some exciting cause. There is an anæmic tendency in such individuals which might be explained on the anatomical defects described by Virchow (in which case the disease would be practically incurable), but which seems more probably dependent on a defective evolution of the blood-forming organs.

I have ventured to point out these matters because of the difficulty, which I feel sure is a real one, of deciding in any given case whether it is a chlorosis or not. It is not a mere question of nomenclature; if that were all it would matter no whit by what name we signified these conditions; but it is the difficulty which has arisen in consequence of the word "chlorosis" being held to convey more than the word anæmia, and being also arbitrarily considered inapplicable to idiopathic anæmia arising in later life. It may be most desirable, as Immermann (in his article on chlorosis in Ziemssen's *Cyclopædia*) rejoices to think it is the case, that in chlorosis we have a distinct malady rescued from being merged in the "great ocean of anæmia"; but, unless its distinctive features can be more sharply defined than they are at present, the difficulty of separating it from other forms of idiopathic anæmia will constantly be felt.

I desire now to speak shortly of two of the more constant and striking effects produced by the anæmic condition—the signs furnished by the heart and blood-vessels, and the influence it has in determining fatty degeneration. Upon the former of these points, which has been the object of much controversy and speculation, I hardly feel qualified to speak with authority, not having studied the matter so deeply as many. It seems to me, however, that, as regards the production of so-called hæmic murmurs, we have to consider, not only the factor of the blood-impovertyment, and especially its diminution in quantity, but also the factor of the conditions of the walls of the heart and blood-vessels induced by that impoverished blood. Indeed, the idea that the murmurs are solely due to the first of these factors has, I believe, been almost abandoned; it did not afford an adequate explanation, and seemed to be contrary to physical laws. As regards the purely cardiac murmur, there are at least two conceivable explanations for it in which the condition of the muscular walls (dependent on the anæmia) is primarily concerned. One of these is that the heart is dilated, and the murmurs are produced by its cavities being incompletely filled, and the other that there is actual regurgitation through the tricuspid and mitral orifices owing to the imperfect action of the enfeebled papillary muscles deranging the mechanism of the valves. Besides the cardiac bruits, there are murmurs produced over the pulmonary artery and aorta. All of these murmurs are systolic in time, and vary much in intensity and situation; the most constant seat being at the base of the heart, the next at or within the left apex. Dr. Pearson Irvine, whose loss we have had so previously to deplore, studied this subject for a long time, and embodied his conclusions in a paper read before the Royal Medical and Chirurgical Society in 1877. He started from the ground that dilatation, especially of the left ventricle, is the chief cardiac change in chlorosis—an opinion promulgated by Stark,—and endeavoured to explain the murmurs, not obviously vascular, on the fact of this dilatation. One may admit the fact that in many chlorotics there are signs of this dilatation. The matter, however, is very difficult of clinical proof, but it is supported by the *post mortem* evidence of hypertrophy by Virchow and Lewinski—a hypertrophy which is as conceivably produced secondarily to a dilatation, as to a narrowed aorta. The known relation between anæmia and fatty degeneration of the heart supplies us with ground for believing that in slighter cases the heart muscle is enfeebled; and probably also its innervation is affected by the impoverished blood. Dr. Irvine distinguished four cardiac murmurs in chlorosis, having their points of maximum intensity over the left auricle and its appendix, at the left apex, over the fourth left costal cartilage, limited there or transmitted downwards to the right or left, and lastly, in the fifth left space, below it or over the sternum adjacent to it. He was inclined to attribute the production of these murmurs to the dilatation of one or both ventricles, and did not favour the view that any of them were due to regurgitation. The regurgitant hypothesis, which explains the auricular murmur as well as those met with about the region of the apex, is supported on the left side by the almost constant accentuation of the second pulmonary sound, and on the right side by the occurrence of jugular pulsation. The mechanism of this regurgitation may be either a slight inadequacy in the contraction of the papillary muscles, or it may be dependent on dilatation. Although the basic systolic murmur may be in many instances due to this regurgitant eddy into the auricular appendix, there can be no question that bruits are heard strictly over the aortic and pulmonary areas. Of the mode of the production of the former (which are not common) no adequate explanation has been given; but of the latter, the true pulmonary bruit, we have Quincke's view that, in the cases

where it is marked, the lung is retracted and the pulmonary artery slightly compressed at every systole; and we know how pressure by the stethoscope will increase the intensity of these bruits.

The murmur heard in the carotids and other arteries is only an exaggerated degree of what can be heard in health, and is probably due to the impaired tones of the vessel allowing it to be more readily influenced by slight pressure.

The hum in the jugular vein—to which more significance has been attached than it merits, as shown by Dr. Herbert Davies—is produced by pressure, and increased in intensity whenever the flow of blood is accelerated. This is illustrated in the influence of posture—its increase in the standing position, in the rise and fall of the otherwise continuous blowing sound with the respiratory movement, the increased respiration into the chest with every deep inspiration being accompanied by increase in the intensity of the murmur, which can be entirely arrested by forced expiration. It is noticed also that the auricular and ventricular diastole influence slightly the intensity of this bruit, so that in it we have a measure of the variations in celerity of the venous blood-flow.

I ought not to leave this part of the subject without alluding to the hypothesis advanced by Dr. Goodhart (*Lancet*, 1880, vol. i, p. 479), that anæmia may be at the foundation of some of these valvular diseases, especially of the mitral valve, the etiology of which is still obscure. He thinks the evidence conclusive that in anæmia there is ventricular dilatation, just as there is *post mortem* evidence of muscular degeneration, and suggests that the continuance of regurgitation may submit the valves to so constant and irregular a strain as to lead to alterations in their intimate structure, thickenings and puckering, productive of permanent regurgitation, and possibly some stenosis.

DEFECTIVE NERVE-POWER AS A PRIMARY CAUSE OF DISEASE, WITH ITS SPECIAL RELATION TO DIPSO MANIA.*

By STEPHEN S. ALFORD, F.R.C.S.

MUCH of my time of late has been devoted to efforts for promoting the recovery of dipsomaniacs. Those lapsing into dipsomania are generally of a highly nervous, sensitive, lively temperament. This condition often has its commencement in mistaken efforts to sustain exhausted nerve-power, arising from continued overstrain and irregular work at high pressure; or from hereditary inability to endure the ordinary stress and troubles of life; or from a system exhausted by indulgences and late and irregular hours; or even by indolence and ennui; each and all being modified by circumstances of society, and by atmospheric and climatic conditions. In studying these cases, and their etiology, my attention has been directed to the harmful effect produced by the above causes acting on defective nerve-power.

As an old practitioner, I do not pretend to enter into the new physiological or pathological researches as to molecular disarrangements, but trust the few practical observations I can make may lead to a discussion on this subject. Surely, the effect of low nerve-power is not sufficiently recognised as a primary cause of disease. If the nervous system, including the cerebral, spinal, and sympathetic centres, with the sensorial and motor nerves distributed over the body, form its working, regulating, and protecting power, it is evident that their defect or disarrangement must more or less interfere with the functions depending upon them. We know for instance that, even when the nerve-power is only temporarily defective, the controlling influence of the vaso-motor nerves may so fail, that the smaller blood-vessels lose their elasticity, and, becoming distended with blood, produce the appearance seen in blushing. We further see this in the face of the drunkard as the effect of repeated stimulation; the vessels, from being frequently dilated, become at length permanently enlarged. Does not deficient nerve-power, as exhibited in the form of insufficient control over the vaso-motor nerves, cause the first dilatation of the smaller blood-vessels, which, by being intensified or frequently repeated, may pass into inflammation? Or further, by producing a modified increased flow of the vivifying stream, may not this defective nerve-power so interfere with the nutritive processes as to cause perverted deposits and dangerous structural changes, as exemplified in tubercle, cancer, and other destructive formations which occur in an asthenic condition of the system? Thus, what is ordinarily a healthy reparative process, becomes in our system, by defective controlling nerve-action, a destructive power which may end in death. The effect of defective or irregular nerve-power is also evi-

* Read before the North London District of the Metropolitan Counties Branch of the British Medical Association.

dent in glandular secretions, on which depend the functions of important organs, the natural secretion being either increased, diminished, or perverted. This is exemplified by the action of the nervous system in producing a deficiency or superabundance of bile, or in the formation of an excess of sugar, as in diabetes. Similar disarrangements of healthy action may occur in any of the secreting organs when the exciting, controlling, nerve-power is out of gear. Most of the ailments from which we suffer seem to depend, in the first place, on defective nerve-action.

As preservers of health, it behoves us to endeavour to arrest the evil at its source; we must not treat symptoms alone; much time and suffering may be saved by tracing a disease back to its primary cause, so as to have reliable principles of action. By thus recognising the primary disturbing cause, and preventing the first run down of nerve-power, much may be done to arrest disease. This neurasthenia or low nerve-condition is first recognised by lassitude, as seen in a more marked form in the utter prostration which precedes acute attacks of disease. Whenever we feel tired, and overdone nature plainly tells us she is exhausted, and will not be neglected with impunity, to force her by stimulants to continue work is a dangerous experiment, which, if often repeated, will sooner or later end in a complete breakdown. But, while yielding to this needed repose, other functional disarrangements may need attention. Insomnia, with drowsiness and gaping, is a distressing condition, arising from cerebral exhaustion; it does not so much produce special local affections, as a miserable misanthropic state. The true restorer from this condition is rest, with a carefully regulated diet, avoidance of stimulants, and general attention to the secretions. These may be beneficially supplemented by mild nerve-tonics. Sick-headache is an early symptom of nerve-exhaustion, and the rest which it enforces upon us often prevents other dangerous attacks. Sick-headaches chiefly prevail during the more active and anxious period of life, between the ages of fifteen and fifty. Hay-fever is another nervous idiosyncrasy, attacking those whose nerve-powers are on the stretch; barristers and other close mental workers being especially liable to it.

There are numerous morbid fears arising from deficient nerve-power, such as fear of places, fear of society, dread of being alone, apprehensions of various kinds; causeless, but real and distressing, fear of everything and everybody. Any function may be more or less disturbed, stimulating real disease, and in this respect resembling hysteria, although hysteria generally arises from some special local cause. Spinal exhaustion is manifested by restlessness and physical excitement. Dr. Beard of New York has devoted special attention to this subject; for its fuller treatment, I refer you to his numerous publications. He calls this nervous exhaustion neurasthenia. The past neglect of this negative nerve-condition has resulted in much erroneous treatment, and often the aggravation, rather than the alleviation, of disease.

Exhausted nerve-power may arise either from excessive, late, or irregular mental work, unhealthy employments, contaminated air, intemperance in any form, indolence or *ennui*, climatic influences, or the exactions of society, and other apparently unavoidable artificial conditions of life. It frequently arises from hereditary causes.

Unless brought under early and careful treatment, neurasthenia may lead to melancholia, dipsomania, epilepsy, insanity, and other distressing conditions.

The treatment must be chiefly directed to general regimen, aided by mild nerve-tonics and electricity.

Dipsomania or inebriety is defined by Dr. Beard as "a fundamental disease of the nervous system, primarily of a functional character". Dipsomania, as well as insanity, paralysis, chorea, neuralgia, and other diseases of the nervous system, must be considered scientifically as belonging to the departments of neurology and psychology. It is associated with periodic mental depression, insomnia, general nervousness, tremors, mental irritability, hallucinations, delusions, moral decline, and in some cases trance, any or all of which conditions may precede, accompany, or follow an attack. These symptoms, however, are not found in all cases, nor in the same case at all times; but each paroxysm is invariably connected with an irresistible craving for stimulants, opium, or chloral.

Dipsomania and mere drunkenness are two distinct conditions. The former is irresistible, independent of the ordinary efforts of the will, often quite unconnected with temptation, and arises from an individual condition; whereas drunkenness depends more on outside allurements. Like neuralgia, hay-fever, and insanity, dipsomania is periodic; whilst the vice drunkenness is constant, or only modified by external circumstances. Dipsomania may be either hereditary, or the result of an inherited nervous diathesis, and transmitted like other family diseases. Drunkenness, it is true, may arise from an inherited vicious temperament, but can be distinguished by the character of the individual.

Dipsomania or inebriety may be sudden in its attacks, following the

action of physical injury, direct or indirect, such as blows on the head, concussion of the brain or spinal cord, sunstroke and its effects, any traumatic injury that disturbs the harmony of the organism; diseases of both a local and constitutional character which affect the system also react in this way; hæmorrhage, typhoid fever, rheumatism, and nearly all disorders of the stomach and liver, which break up and pervert healthy nerve-power, are liable to react in inebriety. After severe injury or disease, the natural nerve-vigour is lowered, and departure from health comes on quickly. Neurasthenia or asthenic nerve-power is the common indication in these cases of the departure from health. Neurasthenia is to the nervous system what anæmia is to the blood.

Inebriety also results from psychological causes, as depressing disappointments, hereditary nervous susceptibility, malformation of organs, or defective powers, either in function or structure, which may be diverted into inebriety from the slightest exciting cause, or the desire to relieve exhaustion. Inebriety may be the result of long continued painful emotions, either of fear or of joy; bad company, exhausting indulgences, or the mysterious effects of mind over body; all forms of suspension, alteration, and change in action of mental functions.

Whenever the strain in the nerves and brain is beyond a certain stage, exhaustion follows, for which stimulants are taken. Inebriety is a craving to satisfy this want, and the greatest effort of will is powerless to resist the infatuation for stimulants. It is important to diagnose between dipsomania and drunkenness, since the efforts for the treatment and recovery of dipsomania do not apply to the mere drunkard, and may be even of little avail for him. The man who drinks recklessly, from companionship and sociability, is very different from one who, in spite of most earnest striving to avoid drinking, is irresistibly impelled to do so. We can no more draw distinctly the line of demarcation between drunkenness and dipsomania, than we can determine precisely where day ends and night begins. Inebriates generally have a strong desire to overcome their failing, although, when unaided, they are powerless to do so. As in other nervous diseases, the mind can be made to act on the body; so that, when stimulants are withdrawn, it then forms an important element in assisting the recovery. An inebriate during the intervals has no desire for alcohol. This is not the case with the drunkard, who is always ready for his beverage. The inheritance of inebriety follows the same laws as other nervous diseases. Drunkenness and inebriety are allied to each other much as eccentricity is allied to insanity, the former often passing into the latter. One of the chief predisposing causes of inebriety is civilisation, entailing, as it does in its modern development, so great an expenditure of nerve-force; and its action in this way is seen more amongst the higher and middle classes, and those who live by brain-work and indoor occupations.

Inebriety has arisen amongst us and progressed *pari passu* with other nervous diseases, to which class it belongs. The exciting causes of inebriety are stimulants, opiates, and chloral, which, by acting in a secondary manner upon low nerve-powers, produce the diseased condition known as dipsomania.

In America, an apparent paradox exists, for, while there is less excessive drinking and more total abstinence than in any country of the world, still there is more inebriety. The explanation of this is found in the fact that, from climatic and other surrounding circumstances, stimulants can scarcely be taken by a nervous temperament, even in moderation, without inducing inebriety. There is universally an absence of stimulants at all meals in the United States.

Atmospheric influence is another undoubted exciting cause of this affection, as sometimes indicated by the production of a paroxysm from exposure to sea-air, easterly winds, and other similar changes.

The treatment of inebriety must be carried out on the same general principles as for all other nervous diseases; firstly, by the removal of the exciting causes, and secondly, by fortifying the system with sedatives and tonics. The exciting cause can only be effectually removed by confinement in a retreat, thus separating the patient from alcohol, opium, or chloral. The necessary period of restraint varies from a few months in mild cases, to even years in the more confirmed cases. When the care and treatment of inebriates are more generally recognised, and effectual opportunities exist, so that cases can be treated in early stages, greater success will be obtained. In America, it is estimated that one-third of inebriates under treatment recover, another third are restored for a time, and the remainder are unaffected by treatment. This is as large a proportion of recoveries as we find in any other class of disease; in insanity, only one-tenth of those under treatment are restored to health.

As to special treatment, we find that, whilst the patients are under the alcoholic influence, in large doses of one to two drachms freely given, are of decided use. Diluents of barley-water with lemon-juice, butter-milk, or similar drinks, form an important feature of treat-

ment in the early stages, acting as diaphoretics and diuretics, and thus clearing the system. Vapour-baths have a useful and soothing effect, and divert and occupy the attention. Cannabis indica is useful to promote sleep and soothe the patient. As soon as the restless craving has abated, we must order exercise and light occupation, together with a mild diet, including a free use of fruit, but avoiding butchers' meat. The red cinchona bark is certainly useful in restoring nerve-power, while caffeine and coca can be taken when special nerve-exhaustion arises. The various nerve-tonics, as quinine, arsenic, and strychnine, can be given with benefit. Iron is not so useful, unless in the presence of anæmia. Total and constant abstinence from stimulants is essential. After recovery, to prevent a relapse, as well as in the course of treatment, religion certainly has a fortifying influence in aiding the resistance to the craving, which will return from time to time. It is also important to avoid old associations and habits. My observations in America have convinced me that, under judicious management, a large proportion of dipsomaniacs may be recovered. At present, no sufficient opportunity exists in this country for their treatment. Efforts are being made to establish a pattern inebriate home in this country; and a meeting is to be held at the Mansion House on May 17th, at 3 P.M., to start such an institution, to be called the Dalrymple Retreat for Inebriates.

/ GUNSHOT WOUND OF THE BRAIN: DEATH AFTER SEVEN WEEKS.

By ALEXANDER NEWINGTON, M.B.Cantab.,
Wheatley, Oxon.

ON the afternoon of December 4th, 1880, I was summoned to see a farmer who had attempted suicide by shooting himself under the chin with a small revolver. The patient had already been seen by my friend Mr. Briscoe, surgeon to the Radcliffe Infirmary, Oxford, who had dressed the wound, and requested the friends to hand the case over to me.

The man had been in a desponding state for some time; but, on the evening before the attempt, he had friends visiting him, and they did not observe anything strange either in his conversation or manner, and he seemed to be in his usual spirits. On the morning of the 4th, he rose earlier than usual, and went out to his farm, although it was dark. He had evidently roamed about for some time, and, about 8 A.M., when a mile and a half from his home, he fired a small revolver in a perpendicular direction under his chin; he appeared then to have fallen across a sheep-trough, and bled profusely, as quite a pint of blood was found in the trough besides what was on the ground. About two hours after this, he returned home, walking erect, with his greatcoat wrapped round his neck. On the road home, he had to get over three hedges, one of which was a difficult one, with a nasty ditch on the far side. When he reached home, it was noticed he could not express himself except in monosyllables. He passed through the house, and locked himself in a privy in the garden. After some time, he was induced to open the door, when he was led into the house and taken up to bed.

At 3.30 P.M., I arrived, and found J. B., a well-made man of forty-six years, in bed. He was very restless, requiring three men to hold him in bed. There was congealed blood about the neck, mouth, and nose. The left eye was slightly protruding, and the eyelids much swollen and ecchymosed, especially the upper lid. The face was rather drawn to the left side. He was unable to speak. I found a small wound under the chin, about two inches behind the symphysis of the lower jaw. The right side of the body was paralysed. There was no ptosis of the right eye. Pulse 60; temperature normal. I was told that, soon after being put to bed, he became comatose, and remained so for some time. At 9 P.M., he was comatose; pulse 60.

December 5th, 11 A.M. He was comatose. The left pupil was rather contracted; both pupils acted to the stimulus of light. Pulse 60; temperature normal. At 9 P.M., he was still in the same condition. I passed a catheter, and drew off a pint and a half of clear urine. Pulse 60.

December 6th, 11.15 A.M. He had just got into bed, having suddenly got up and walked four times round the room, and looked out of the window for some time. I was told he had gradually come to himself. He could say "yes" and "no", and evidently understood when spoken to. The paralysis of the limbs was quite gone, but that of the face remained. Pulse 64. At 4 P.M., he was constantly rubbing his face with a handkerchief, as if washing. He opened both eyes. I passed the catheter. Pulse 100.

December 7th, 11.30 A.M. He was able to speak better, and answered

rationally. The bowels had been moved after an aperient. Pulse 80. At 3 P.M., I got him out of bed, and induced him to pass urine.

December 8th. He could put more words together. He hesitated much, and spoke very slowly. He was restless at times. Pulse 116; temperature normal; respirations 19.

December 9th. There was further improvement in speech. He said his head "felt funny", and that the roof of his mouth felt sore. He opened his mouth wide when asked to do so. I could see where the bullet had passed through the posterior third of the left hard palate, but could discover no mark of injury to the tongue. He was cheerful, and had a good appetite; he swallowed easily. His memory was very defective.

December 10th. The improvement in speech was maintained. He looked distressed, and complained much of headache. He protruded his tongue easily; it turned to the right at the middle line. He said there was a little pain at the back part of the tongue. Pulse 96; temperature 100.

January 9th, 1881. He had made steady improvement up to this date.

January 10th, 11 A.M. I found him very dull, and weak on his legs. His right hand was weaker than his left. I was told he had a restless night, and wandered a good deal. At 7 P.M., I was sent for in a hurry. He had been vomiting since 5 P.M. He had complete paralysis of the right side, but no ptosis. Pulse 60; temperature 102.6; respirations 19.

January 21st. He was very talkative; he repeated everything said to him. He could tell the time by the clock over the mantel-shelf in front of his bed. He could get his right hand up to his face. Pulse 120, very feeble.

January 22nd. He vomited after food.

January 24th. He was still able to move the right hand to the face. He was very dull. The catheter had been used daily since the 14th instant. Pulse 130, very feeble. At 11.30 P.M., he died very quietly.

Post Mortem Examination.—Sixteen hours after death, I made an examination of the contents of the skull, Mr. Briscoe assisting me. Rigor mortis was well marked. On removing the calvarium, the general appearance of the dura mater was healthy, with the exception of a spot about the size of a shilling over the top of the left frontal lobe, which was congested. There was no mark of injury on the inner surface of the calvarium. On removing the dura mater, it was found adherent at the spot where it appeared congested; in fact, a portion of the brain-substance adhered to it when peeled off. Three small plates of bone were found in the depression of the frontal lobe caused by removing the dura mater; one was about a fourth of an inch square. The base of the brain was normal, except a small depressed scar in the posterior frontal convolution. A director with slight force could be passed into this depression, and out at the lacerated spot in the superior frontal convolution. On dividing that portion of the brain to the inner side of the director, a canal was seen extending along the course of the probe, and this evidently had become partially obliterated through the process of reparation. We had expected to find an abscess on opening this canal, and were surprised to find it nearly healed, and no trace of the bullet. We next proceeded to cut through the outer surface of the left frontal lobe, when about an ounce of pus escaped, and the lobe appeared to collapse. In the cavity, we found the bullet, which was a small conical one, weighing one drachm. Just in front of the articulation of the lesser wing of the sphenoid with the orbital plate, and about three-fourths of an inch to the left of the crista galli, was a jagged hole half an inch square, with two or three pieces of lead on the edges of the opening. A probe, passed into this, went to the back of the eyeball and to the outer side of the optic nerve. It could not be passed throughout the course the bullet had taken, as repair had taken place.

REMARKS.—It will be seen, from the clinical history of this case and the *post mortem* examination, that the bullet, having entered two inches behind the symphysis of the lower jaw in the middle line, passed through the back of the tongue, the left hard palate, the outer and back portion of the left nasal cavity, to the inner side of the left optic nerve, and passing through the orbital plate and left frontal lobe, must have struck the calvarium and rebounded into the left frontal lobe. The case is interesting in many ways: the distance the man was able to walk after so severe an injury; his inability to articulate; the gradually increasing condition of coma up to 2 P.M., he then gradually becoming restless, and exhibiting a great deal of strength, although one side was paralysed; the return to coma lasting a day and a half. The fact of regaining his strength again so rapidly is also interesting, and the gradual improvement up to January 10th, five weeks after the injury. No symptoms of acute inflammation of the