of cornea pellucida. He was admitted as an in-door patient to the institution on November 11th, when I broke up some of the adhesions with a needle, and next day I made a large iridectomy and extracted the lens. On November 14th, he had good sight; and on the 18th, he was dismissed cured. The left eye is still improving; but, owing to its extensive opacity, he will depend almost exclusively upon his right eye, which was considered hopeless.

List of Cases.

No.	Name and age.	Occupation.	Complications.	Sight.
1	R. W., 58	Labourer.	Anterior adhesion.	Good.
2	J. M., 5	School.	Anterior adhesion.	Good.
3	J. D., 10	School.		Good.
4	W. D., 39	Hammerman.	Posterior adhesion.	Good.
5	R. S., 19	Engineer.	Laceration of cornea.	Medium.
6	F. R., 27	Cabinet-maker.	Iron in lens.	Good.
7	J. B., 19	Smith.		Good. Subsequent atrophy.
8	A. F., 12	Message-boy.		Good.
9	R. M'L., 18	Rivetter.	Posterior synechia.	Good.
10	B. M., 21	Caulker.	Steel in lens.	Good.
11	G. H., 41	Boiler-maker.	Posterior adhesion.	Good.
12	S. O., 61	Chandelier polisher.	Corneal opacity. Iron in lens.	Good.
13	T. H., 34	Engine-fitter.	Lacerated iris.	Good.
14	G. C., 110	Engineer.		Good.
15	A. N., 30	Engine-keeper.	Dislocation of lens, etc.	Good.
16	R. M'A., 18	Boiler-maker.	Lacerated iris and ciliary process.	None.
17	J. J., 31	Granite polisher.	Quartz in lens.	Good.
18	T. H.	Rivetter.	Steel in lens.	Good.
19	J. C., 34	Miller.		Good.
20	D. S., 21	Boiler-maker.	Laceration of iris.	Good.
21	W. B., 19	Cloak-maker.	Lacerated cornea & iris.	None.
22	W. S., 14	Iron-dresser.		Good.
23	A. M'C., 30	Bolt-maker.		None.
24	J. M., 38	Rivetter.		Good.
25	N. M'G., 14	School.		Medium.
26	W. G., 17		Dislocation of lens.	Medium.
27	D. M'T., 34	Boiler-maker.		Good.
28	G. P., 27	Rivetter.		None.
29	J. S., 21	Boiler-maker.		Good.
30	J. C., 28	Mechanic.		Good.
31	J. B., 42	Engine-fitter.	Posterior synechia.	Medium.
32	F. H., 9	School.		Good.
33	J. C., 9	School.		Good.
34	T. U., 9	Engineer.		Medium.
35	F. L., 14	Sewing-girl.	Posterior adhesion.	Good.
36	M. S., 19	Iron-dresser.		Medium.
37	P. M., 22	Labourer.		Good.
38	E. L., 20	Boiler-maker.	Foreign body in lens. Hypopion, etc.	Good.
39	J. K., 25	Engine-fitter.		Good.
40	J. S., 19	Labourer.		Good.
41	J. S., 34	Boiler-maker.	Contortion of eye.	Medium.
42	A. Y., 25	Mechanic.		Result not ascertained.
43	R. K., 27	Engine-fitter.		Good.
44	T. M., 13	Message-boy.		Good.
45	R. C., 15	Rivetter.	Strabismus.	Good.
46	T. M., 21	Servant.		Good.
47	G. B., 8	School.		Good.
48	M. J., 17	Sewing-girl.	Anterior adhesion.	Good.
49	T. M., 19	Rivetter.	Laceration of iris.	Good.
50	N. M'G., 18	Labourer.	Laceration of iris.	Good.
51	A. S., 8	Message-boy.	Lacerated lens by thorn.	Good.
52	R. W., 22	Boiler-maker.	Steel in lens.	Good.
53	T. W., 36	Engineer.	Laceration of cornea and iris.	Medium.
54	D. M'F., 35	Boiler-maker.		None.
55	D. W., 52	Granary-keeper.	Posterior adhesion.	Good.
56	M. S., 19	Iron-dresser.		Good.
57	J. M'K., 29	Rivetter.	Lacerated iris.	Good.
58	N. C., 21	Rivetter.	Dislocation into vitreous.	Good.
59	P. G., 38	Miner.	Posterior adhesion.	Good.
60	E. C., 28	Caulker.		Good.

CLINICAL MEMORANDA.

VACCINATION BY VESICATION.

THE following method will be found more generally applicable than any I have seen described, as it requires but one interview with the patient; and while it is almost equally as expeditious as the use of the lancet, is much more certain. Fill the hollow cylinder of an ordinary sized key nearly, but not quite, full of dry cotton-wool well pressed in; dip a small piece of wool into strong liquor ammoniæ, and place it on the dry wool, so as just to fill the cylinder; then hold it firmly on the part to be vaccinated, so that the wool dipped in the liquor ammoniæ is kept in contact with the skin. In from one to two minutes, the cuticle will be found pale and wrinkled, and can be easily rubbed off by friction with a towel. The vaccine matter is then applied to the abraded surface.

FRANK BUSZARD, M.D., Northampton.

HYPERTROPHY AND DILATATION OF THE HEART.

By J. MILNER FOTHERGILL, M.D., Vienna.

THOUGH hypertrophy of the heart has been recently allowed to be, in almost every case, a compensatory change and not a disease *per se*, we are not yet acquainted with the steps by which this change is inaugurated. That it is a result of some impression on the cardiac ganglia, and a trophic process set on foot, seems somewhat more than probable. Still, in other organs, we find involuntary muscular fibre with a power of self-increase in bulk, but we do not find the same accumulation of nerves as in the heart. Under these circumstances, then, we must feel that the mode by which cardiac hypertrophy is inaugurated is somewhat obscure. As some name must be used to cover what we do not know, it may be called a trophic action of the cardiac ganglia. When we come to the questions of under what circumstances this hypertrophy is produced, the ground is much clearer. We find hypertrophy of the heart-walls under three different sets of circumstances: (1) when an obstruction is offered to the flow of blood; (2) when the heart is subjected to distension under unusual pressure; (3) when its action is impeded, as in displacement, pericardial adhesion, etc.

1. With the first we are as yet most familiar: and it is under these circumstances that we find hypertrophy of the purest kind—that is, without dilatation. Thus in aortic obstruction, when free from regurgitation, we find pure and simple hypertrophy. A gradual obstruction is opposed to the complete and perfect contraction of the left ventricle, and hypertrophy grows up and meets and overcomes this resistance. So, too, in Bright's disease, where we have, according to Dr. George Johnson, hypertrophy of the muscular tunic of the arterioles, we have an obstruction offered to the flow of blood, and changes, usually hypertrophy, inaugurated in the left ventricle. But when the obstruction offered is of a more rapid nature, we find dilatation occurring, to a greater or less extent, in accordance with the rapidity of the formation of the obstruction, the extent to which the obstruction attains, and the heart's power to meet the obstruction; this last depending very greatly upon the general nutrition. But this dilatation is soon accompanied by hypertrophy; except in cases of intense general debility, or defective coronary circulation. This brings us to the second division, where there is also distension of the heart under unusual pressure.

2. Though hypertrophy of the heart is associated usually with the idea of obstruction in the blood-stream, still we find it in its maximum intensity in aortic regurgitation, and that, too, when most free from admixture of obstruction. For the same obstruction would also offer an impediment to the backward rush of blood from the aorta. We find hypertrophy combined with dilatation in the left ventricle in mitral regurgitation. We find too, according to Niemeyer, hypertrophy growing around dilatation when the heart-wall has yielded, owing to its softened condition, after pericarditis. In all the conditions we have hypertrophy produced without any obstruction to the blood-stream—*i.e.*, the condition of hypertrophy is found under these circumstances, whether any obstruction be found combined with them or not. These conditions themselves cause hypertrophy. Now, on what condition does this hypertrophy essentially depend? There is one condition common to all these instances, and the hypertrophy is found to its greatest extent where this cause is in strongest action. The condition is that of distension under increased pressure, and is found most marked in aortic regurgitation, where the ventricle is filled by blood coming in from the aortic recoil, as well as from the pulmonic veins through the mitral orifice. Here the muscular chamber in diastole is distended by the elastic recoil of the greater as well as the lesser circulation. Under this extreme distension, then, we find extreme hypertrophy. We also find hypertrophy and dilatation when the left ventricle is filled under unwonted pressure from a gorged pulmonic circulation alone—*i.e.*, in mitral regurgitation. Here so much blood is driven backwards through the mitral valve at every systole. The pulmonic vessels are already full of blood forced into them by an hypertrophied right ventricle; to this is added the amount of blood passing backwards through the insufficient mitral valve. Thus the pulmonic system is gorged and forces blood, in diastole, through the mitral valve under increased pressure. This is increased by the energy with which the dilated and hypertrophied auricle completes the ventricular distension by its contraction. Under these circumstances, we find that we have hypertrophy and dilatation of the left ventricle; first, dilatation inevitably the result of distension under increased pressure; and hypertrophy setting in to resist the over-distension. This is all clear enough, and presents no great difficulties. The chamber is filled under undue pressure in both the above cases, and hypertrophy sets in until the heart-wall is again equal to the increased

pressure under which it is distended, and a balance again attained. On the attainment of this new equilibrium, the dilatation is arrested, and the dilating process ceases. But in considering the last division of dilatation, namely, where dilatation occurs without increase in the dilating power, but is associated with inefficiency in the muscular walls of the heart, a somewhat complicated problem is before us. That the heart-walls should yield before even the ordinary venous pressure when they are themselves weakened by inflammatory action, is no more than we might expect; and that, on recovering to some extent their structural integrity, hypertrophy grows around that dilated ventricle, and enables it once more to fulfil its function in a moderately efficient manner. It is even consistent with clinical experience, if that condition of distension, or even dilatation, be removed, and no evidence of dilatation remain.

When the ventricle yields under no temporary condition of its walls, we find usually three different conditions, forming subdivisions; (1) some obstruction offered to the flow; (2) a condition of subparalysis of the heart, or rather of the cardiac ganglia, due to general exhaustion of the sympathetic; and (3) defective nutrition. In all these conditions we again find one condition common to them all: and that is imperfect contraction of the ventricle. Here we have the ventricle already partially filled on diastole; so that either the pulmonic veins must remain partially gorged, or the chamber must dilate until the venous distension is relieved. It thus becomes a question of venous elasticity *versus* the dilatability of heart muscular fibre. A question now naturally arises as to how this does not invariably proceed, but becomes arrested. The muscular fibre, more stretched in each diastole, would distend to the point of over-distension and paralysis; and so in the right ventricle, it often does in the attacks of cardiac asthma. The arrest of this dilatation is brought about by the elasticity of muscular fibre itself. Carpenter shows that, when a muscle is hung up and a small weight attached to it, it soon stretches to its full normal length; this he calls the primary elongation of a muscle. But a greater power is needed to stretch it further, though it is capable of being stretched further. But the secondary elongation, as Carpenter terms it, involves the elasticity of the muscular fibre. It is, then, this secondary elongation of muscular fibre which is induced in cardiac dilatation. When the muscular fibre is distended under increased pressure, this secondary elongation is produced, until hypertrophy, or rather hyperplasia, renders the muscular wall again equal to the distending power. But in the last form of cardiac distension, where there is no increased pressure in the distending power, but the heart becomes dilated from inefficiency in its power to resist the normal distending pressure, we find that dilatation is arrested by the elasticity of the muscular fibre. The process of dilatation or distension of the muscular chamber goes on until the resistance offered by the muscular elasticity is equal to the distending power of the venous elasticity; then it stops. As soon as a balance is again attained between the resistance offered by the elasticity of the muscular fibres of the heart, and the distending force exercised by the elastic recoil of the distended veins and pulmonic artery, *plus* the other causes of blood-circulation in the pulmonic system—as respiration, etc.—the dilatation ceases. The sensation of over-distension seems pretty clearly to be the cause of that trophic action which we call hypertrophy. The walls that yielded once, no longer give way, but become supported by a spontaneous thickening, which enables them to resist distension further. Even when the exciting cause of the dilatation is obstruction offered to the flow forward of the blood, the hypertrophy not only enables the heart to overcome the obstacle, but enables it to resist further dilatation. It may be put thus even: that, where the heart is unequal to immediate hypertrophy to meet obstruction, it can do so secondarily when the sensation of over-distension is added to that of the original obstruction. We do, in fact, regard hypertrophy with dilatation as an inferior form of compensation to pure hypertrophy; and Traube calls it the second form of compensation, or imperfect compensation. So when no hypertrophy is produced by dilatation, we regard the case as much graver than where there is hypertrophy. In fact, by the amount of hypertrophy in comparison to the amount of dilatation, we compute the patient's chances of a prolonged existence. Where there is no hypertrophy, and a case remains with simple dilatation, a condition of great debility is inevitable, and the feeble condition of the left ventricle is no longer over-balanced in consequence of the feeble venous current coming over from a weakened right heart.

3. When the heart is displaced by pleuritic effusions, or by growth within the thorax; when pericardial adhesions impede its action, or even more still, when a general pericardial adhesion surrounds it; whenever the heart is placed, by surroundings, so as to labour at a disadvantage, we find that this trophic action is inaugurated; only, as in the last division, it is wanting from defective nutrition. The hypertrophy, in this last form, is neither to overcome obstruction offered to the blood-

stream, nor yet to withstand distension from increased distending power, but is connected with debility from impeded action.

Finally, is there any one condition common to these various conditions? I am inclined to believe that there is. This condition is tendency to over-distension. In obstruction gradually offered, the over-distension from imperfect contraction is met at an early stage under the most favourable circumstances at a point of visible dilatation in the less favourable cases; and not arrested at all in the least favourable. The same holds good of over-distension from increased distending power, with this difference, that the increase in the distending force invariably produces visible dilatation, and is only arrested by massive hypertrophy, proportioned to the increase in the distending force. So, in the simple cases of distension from impaired heart-wall resistance, sooner or later hypertrophy comes in, and is only totally absent in the most unfavourable cases. In the third division, when the heart is placed at a disadvantage by surroundings, hypertrophy sets in when distension of the heart-walls is becoming increased. It seems, indeed, somewhat more than probable, that hypertrophy of the heart is a trophic action by which it resists dilatation. A certain amount of evidence in favour of this is furnished above. In addition, there are the contrivances to obviate distension of the right ventricle in the diving mammals, consisting of a massive right heart, supported by trabeculae, and relieved by enormous plexuses of bloodvessels. And finally, a last piece of evidence is the recommencement of dilatation, or yielding of the heart-wall, when degeneration cuts down that increase in the muscular structure. The instalment of degeneration of the increased muscular fibre is the signal for the reinauguration of that dilatation which this hyperplasm had arrested. There seems strong grounds for the suspicion, to put it not too strongly, that hypertrophy is a trophic action of the cardiac ganglia, the result of the impression of distension by these cardiac ganglia. Dr. Rutherford has recently told us, that the centres of vaso-motor regulation are not confined to the contents of the encephalon, but that "possibly there are vaso-motor nerve-centres in the submaxillary ganglion, and in the ganglia found in the penis". In a little further time, perhaps, he will be able to tell us of their presence in the ganglia of the heart, so variously constructed. It would be quite in accordance with the above *resumé* of the various conditions under which hypertrophy is found to originate, if it should be ultimately demonstrable that hypertrophy of the heart is the consequence of an increased blood-supply to the heart-walls, by a dilatation of the vessels of the coronary circulation, and thus the formation of a "broad and rapid stream" of blood to them; and that this dilatation has taken its rise in an impression communicated through the vaso-inhibitory nerves, that impression itself originating in a primary impression of distension received by the cardiac ganglia. This would enable us readily to see how we find hypertrophy and dilatation so variously combined, and connected with the general conditions of health, and to comprehend the intrinsic truthfulness of Traube's clinical divisions of (1) hypertrophy, or complete compensation; (2) hypertrophy combined with dilatation, or imperfect compensation; and (3) simple dilatation, or total absence of compensation. We may then venture to consider that hypertrophy of the heart is the result of secondary elongation of the heart-fibres, and that the trophic action is induced when distension is involving the elasticity of the fibres, in accordance with the history given above.

ON THE ASPECTS OF LAW AND MEDICINE IN RELATION TO OUR CRIMINAL JURISPRU- DENCE, WITH SPECIAL REFERENCE TO CASES OF INSANITY.

By GEORGE MAY, Esq., President of the Reading Pathological Society.

PUBLIC attention has been drawn to this important subject by the late trials of the Rev. John Selby Watson and Christiana Edmunds, the results of which I cannot but regard as a reproach to our legislation, too intolerable to be longer sustained. The only plea of insanity accepted by the judges in criminal cases, is the inability of the prisoner to distinguish right from wrong at the time when the crime was committed. The opinions of many learned psychologists are in direct antagonism to this legal dictum; and they declare it to be erroneous and inconsistent with our knowledge of the manifestations of unsound mind. This conflict of opinion has been so painfully developed in the trial, conviction, and subsequent relieve, of Miss Edmunds, that public feeling has been shocked, and our laws have been brought into general disrespect if not contempt. It is believed that many persons of unsound mind retain the abstract knowledge of right and wrong, although the judgment and the power of controlling the will may be diminished or