of ephemeral signs which may have vanished before the "expert pathologist" can be obtained, all point to the value of the *earliest* medical witness on the scene being the one most likely to supply the needful information. And, curiously enough, in another column of THE LANCET this very week is to be found an instance bearing strongly on the point,[1] in which you say: "And it would have been concluded that he met his death by drowning had not the fact been noted by the medical witness that the eyelids were closed when the body was discovered." If the "medical witness" had been Mr. Troutbeck's "skilled pathologist" (who would have had no opportunity of seeing the deceased till he received Mr. Troutbeck's order to make a post-mortem examination) this fact could scarcely have been "noted by the medical witness"—but it is needless to multiply examples. Mr. Troutbeck's view of the coroner's "excuse" is doing yeoman service for the medical profession, and the constantly increasing numbers of medical practitioners who are selected by the county councils (in preference to legal gentlemen) to fill the important office of coroner bear witness to the growing recognition among the educated classes that nine times out of ten "the medical cause of death," which it is the first duty of the coroner to investigate, is best performed by one who can interpret the technical terms, often *necessarily* made use of by the medical witness, to a jury of average British laymen.

I am, Sirs, yours faithfully,

Sept. 19th, 1905. MEDICAL CORONER.

PS.—I hope you will not think it presumptuous if I add (after reading your article on two cases of recent inquests, the one in South Staffordshire and the other in Lancaster, under the heading "Medicine and the Law") that in my opinion neither would the view that "it was idle to suggest insanity" have been expressed in the one, nor an order for exhumation been publicly called for in the other, had the respective coroners been members of our profession. For in the one the letter laid before the jury would have contained ample evidence to the medical mind of the insane condition of the writer at the time; and in the other I fancy that if the Home Secretary had been (privately) made acquainted with the facts it does not seem unreasonable to think he might not have insisted on exhumation, provided no criminal responsibility had been suggested at the preliminary investigation before the deceased coroner and his jury. Is it conceivable, for example, to take another instance, that the Home Secretary in these days of preventive medicine would censure a coroner who should excuse his jury (of 12 or 15 fathers of a family may be) from viewing the body of a child whose violent death (during tracheotomy, for instance) had revealed the fact that death was due to disease of an eminently infectious character, such as diphtheria? The consequence of an inquest having been held without "the view"—when ample evidence of "identity" is forthcoming—is that the inquest may be "quashed." What reasonable ground for "quashing" can be discovered if no criminal liability had been brought home to anyone by the verdict of the jury?

ANGINA PECTORIS AND ALLIED CONDITIONS.

To the Editors of THE LANCET.

SIRS,—On two points arising out of Dr. T. Oliver's lecture on this obscure subject, published in THE LANCET of Sept. 16th, I desire, with your permission, very briefly to remark. One of these concerns the now well-recognised work of another. I am aware that in a lecture it is not possible to deal with matters historical at any length, but the omission of some names is as inexcusable as the production of *Hamlet* with the melancholy prince left out. After reference to the fact that arterial thrombosis may be attended with pain, Dr. Oliver quotes Charcot's interpretation of these phenomena and his introduction of the term "intermittent claudication," as possibly explaining angina pectoris in some cases. That this is a feasible hypothesis, possibly correct in some cases of an affection which may be symptomatic of many conditions, I am not at present concerned either to deny or affirm. The point I would refer to is that this interpretation was advanced as a brilliant suggestion nearly a century ago by Allan Burns of Glasgow, who must in justice be allowed to have anticipated later writers in advancing the occlusional theory of angina pectoris and whose name one would have expected to transpire in this connexion. In his little book, still so valuable because filled with observations culled at first hand from nature, he deals sufficiently fully with the subject.[1] The explanation was then hypothetical and itis no more now, but to Burns belongs whatever credit there may be in having advanced it.

The other point I refer to less willingly, because it is distasteful to call attention to one's own work. Dr. Oliver, like most writers on this subject since Edward Jenner's day, discusses the question of coronary calcification in relation to angina pectoris. The occasional association of these states is, of course, incontestable. No less incontestable is their frequent dissociation. Quite as incontestable to my mind is the fact that the subject has not been investigated in such a manner as to lead to any definite conclusion on the point. The minute microscopical examination of such vessels in these cases has rarely been undertaken, judging from the reports of necropsies, and they occur quite sufficiently often, both in private and in hospital practice, to have afforded opportunity for such examination. In one of a series of lectures on Cardiac Pain, which I delivered from the same rostrum as that from which Dr. Oliver gave the lecture under discussion, I related some particulars of a case of this kind which occurred in my clinic at the Great Northern Central Hospital and in which I was able to determine what was, to my mind, a significant fact whatever its value. My lectures were fully published, with illustrations, in THE LANCET of Nov. 1st, 8th, 15th, and 29th, 1902. The patient had frequent attacks of angina and died in one of them. The necropsy revealed calcareous arteries, a portion of which I decalcified and numerous sections of which I examined microscopically. Some of these, fortunately, cut across a small intravascular aneurysm in the neighbourhood of which there was a nerve ganglion and neuritic fibres which appeared to me to be an explanation of the very frequent attacks of angina which the patient exhibited both when recumbent and when erect. I carefully refrained from being dogmatic in asserting a necessary connexion between these states but confess that no other condition seemed to me to explain the situation so well and I took it as a type of a condition which has admittedly more than one basis. Whether I was correct or in error in doing so can only be decided by a similar examination of calcified coronaries in cases both associated with, and free from, angina pectoris. So far as I know, my case at present stands alone.

I am, Sirs, yours faithfully,

ALEXANDER MORISON, M.D., F.R.C.P. Edin. & Lond.

Upper Berkeley-street, W., Sept. 17th, 1905.

To the Editors of THE LANCET.

SIRS,—In the course of his interesting and instructive remarks in THE LANCET of Sept 16th on the treatment of angina vera Dr. T. Oliver states that of all drugs that give relief there is none that can compare for immediate action and efficacy with nitrite of amyl, while in some cases of angina vera nothing short of the administration of morphine or opium will give relief. There is one drug which compares favourably with nitrite of amyl in the treatment of angina pectoris and that is chloroform. The inhalation of chloroform removes the strain from the heart by vasomotor dilatation of the arterioles much more safely and effectually than does nitrite of amyl. Moreover, chloroform has none of the disadvantages of nitrite of amyl and as it abolishes pain almost immediately neither morphine nor opium is necessary. The inhalation of chloroform need not be pushed beyond the stage of unconsciousness and, unless the heart is actually failing, it always gives complete relief.

I am, Sirs, yours faithfully,

Harley-street, W., Sept. 19th, 1905. EDWARD LAWRIE.

THE CONDITION OF THE BLOOD VESSELS DURING SHOCK.

To the Editors of THE LANCET.

SIRS,—I agree with Mr. J. P. Lockhart Mummery that a full discussion of our divergent views on this subject would take up more of your space than we can hope for, but I should like to be permitted to reply to his letter in your

[1] THE LANCET, Sept. 16th, 1905, p. 842, The Eyelids after Death.

[1] Observations on Diseases of the Heart, &c, Edinburgh, 1809, p. 138.

last issue[1] and to endeavour to convince him and your readers that the theory I have put forward explains the phenomena under consideration better than any other.

Mr. Mummery's first point is to explain the absence of suffusion of the skin with blood as a consequence of dilatation of the vessels in the state of shock. He says that this occurs only "*when the general blood pressure is still high.*" But a combination of a high blood pressure in the central areas with dilatation of the peripheral vessels seems to me very unlikely to occur under any circumstances. It is the tone of the distal vessels that keeps the blood pressure up, and the large vessels are so elastic that if a gradual relaxation of the arterioles takes place from any cause and ultimately becomes complete there must, I think, be a stage during which an obvious suffusion of the surface would be expected. Mr. Mummery wrote[2] "that the exhaustion of the vaso-motor centre is not a sudden process but takes place slowly, the vessels in the splanchnic area first becoming dilated. And in the earlier stages of shock contraction of the peripheral vessels occurs to compensate if possible for the fall in blood pressure brought about by the dilatation of the splanchnic blood vessels." But he also wrote[3] that "the first effect produced upon the blood pressure by injury to any part of the body richly supplied with nerve endings ..... is a rise. If the injury is continued or is very severe this initial rise is either replaced by a fall in blood pressure or is followed by a fall." From this I conclude that, in the first instance, the contraction over-compensates the relaxation of the arteries and this initial rise of blood pressure makes it the more difficult to understand the absence of a stage of suffusion of the surface if the arteries gradually dilate.

As regards the second paragraph of Mr. Mummery's letter I regret very much if I misrepresented his and Dr. G. W. Crile's views in discussing the change from contraction to relaxation of the vessels when shock occurs. Dr. Crile wrote that the cause of the fall of blood pressure, which he invariably found during severe shock, "must be an exhaustion of the cardiac muscle, of the cardiac centres, of the blood vessels or of the vaso-motor centres,"[4] and, *by a process of exclusion*, he selected the last as the true cause. Mr. Mummery wrote[5] that "the essential factor in the production of the condition which we know as shock is a steady fall in general blood pressure. This fall in blood pressure results from exhaustion or fatigue of the vaso-motor centres." I argue that an *exhaustion* of the vaso-motor centre cannot arise and induce a relaxation of the vessels until the tendency to contraction of the vessels has been very powerfully excited. Therefore an intense contraction of the vessels should first occur. If at some stage of the process relaxation takes place suddenly there should, I think, be clinical signs of such a violent change. The change, if it is due to *exhaustion* of the vaso-motor centre, ought to take place suddenly, but if, on the other hand, we assume that it occurs gradually, then the vessels should at first return to their normal size. So long, however, as the degree of shock is deepening there is no evidence of a sudden change from contraction to relaxation, or of a gradual return to the normal. If the vessels gradually dilate without any previous contraction, the condition can hardly be attributed to *exhaustion* or fatigue of the vaso-motor centres.

As regards the obvious condition of the splanchnic vessels I have no experience of experimental work, but I have seen parts of the splanchnic area very frequently when patients have been in a state of profound shock, and I have never noted a dilatation of the small vessels which seemed to me to be caused by shock, whilst I have often observed that there was very little hæmorrhage so long as large vessels were not interfered with. When I have seen these parts suffused with blood by dilatation of the small vessels the cause has generally been some anæsthetic difficulty connected with the respiration. I do not deny—indeed, I asserted in my lecture—that the blood collects in the warmer parts of the body during the condition of shock,[6] but my contention is that "in so far as an unusual amount of blood may be found in the splanchnic area, it is forced rather than drawn into the central parts."[7] This to some extent answers Mr. Mummery's question as to where the blood goes. But if he will read my paper carefully[8] he will see that I cannot agree with him that the blood is incompressible for the purposes under consideration. He pointed out in his lectures that as shock develops the specific gravity of the blood is raised, and this raised specific gravity seems to me quite inconsistent with the view that the vessels are dilated. It indicates rather that the blood is under unusual pressure The change must, I think, be due to a squeezing of the plasma from the vessels into the tissues. The plasma has a lower specific gravity than the blood. There is no doubt in my mind that if life continues and if this raised specific gravity of the blood is not quickly altered by a return of plasma to the blood-vessels it must very soon lead to a diminution in the number of blood corpuscles and thus a certain amount of blood will be destroyed.

Mr. Mummery finally says that the chief argument against the theory I have put forward is found in the action of adrenalin and similar drugs but I have given an explanation of this action which he does not controvert. Adrenalin is the strongest vaso-constrictor known and if the superficial vessels are intensely contracted, as I believe they are in the state of shock, whilst the internal vessels are full, adrenalin can have little effect on the already intensely contracted superficial vessels but may powerfully affect the somewhat dilated internal vessels and thus may raise the blood pressure in the large systemic arteries exactly as pressure on the abdomen does. The effect is very temporary and I think it may give rise to great danger from cardiac overwork. Mr. Mummery does not mention the chief basis of my criticism of the views he advocates. When Dr. Crile arrived at the conclusion that a fall of blood pressure must be due to relaxation of the arteries from exhaustion of the vaso-motor centres he did so by a *process of exclusion*, but he did not take into consideration all the possible causes of lowered blood pressure. At least he did not mention the possibility that contraction of the vessels may also cause a fall of blood pressure. This contraction of the vessels is the explanation of the vascular phenomena of shock which I advocate and I hope that those who are interested in the subject will carefully consider the evidence I have brought forward.

I am, Sirs, yours faithfully,

Portman-street, Sept. 16th, 1905. JOHN D. MALCOLM.

[1] THE LANCET, Sept. 16th, 1905, p. 852.
[2] Ibid., p. 852.
[3] THE LANCET, March 25th, 1905, p. 776.
[4] Blood Pressure in Surgery, p. 401.
[5] THE LANCET, March 18th, 1905, p. 699.
[6] THE LANCET, August 26th, pp. 576 and 579.
[7] Ibid., p. 573.
[8] Ibid., p. 577.

VARICOCELE—WHAT OF IT?

To the Editors of THE LANCET.

SIRS,—I have for long past desired to initiate a discussion in your columns regarding varicocele with the object of eliciting the views of leading surgeons as to how far, if at all, it is a disability or disqualification for the public services. I have the more reason for requesting expressions of opinion in view of the fact that for a period of five years or more I have been president of the army medical boards for the examination of candidates for admission to Woolwich and Sandhurst, and of university men, militia and yeomanry officers and candidates for commissions in the regular forces, and have seen in those five years no small number among these young men and lads rejected on account of varicocele. The subject of varicocele has also presented itself closely to my notice as senior medical officer of recruiting in the London area (headquarters at St. George's Barracks). As to my own views on the subject I cannot do better than give an extract of a short article I wrote in volume I, page 181, of the *Journal of the Royal Army Medical Corps* with the caption (as our American friends term it) Observations on Some Points in the Medical Regulations for Recruiting:—

In respect of variococele the following remarks were made in a report on recruiting by the late Sir Thomas Crawford[1] in the Army Medical Report for 1862: "With a view of determining the extent to which lesions in the genital organs are disqualifications in a soldier, it may be well to premise that it has been generally held by army surgeons that recruits having any disability which can by any possibility interfere with the free motion of the body or extremities or which may give colour to the alleged existence of pain should be rejected. This well-grounded opinion rests on the accumulated experience of the department and is supported by the fact that indifferent characters having such disabilities never fail to allege their existence either as an excuse for the non-performance of duty or for the avoidance of punishment. So long as a soldier can demonstrate the existence of a disease in any organ, so long will it be impracticable to punish him for malingering to avoid his duty, on the one hand, or the penalty of his offences on the other. It is this contingent circumstance *and not any well-grounded belief in the disqualifying nature of many alleged disabilities which leads to the rejection of recruits for blemishes which in no way affect the efficiency of a willing soldier.*"

[1] Director-General A.M S., whose report was written when he held the rank of staff surgeon and was engaged in recruiting duties.