LETTER TO THE EDITORS.

ARTERIAL STRUCTURE AND ARTERIAL FUNCTION.

Sirs,—In considering the question as to where the rise of blood-pressure takes place when it is asserted that "peripheral resistance raises blood-pressure," Dr. Geo. Gibson, in your June issue, asked for a reference to any contemporary writer who has been "guilty" of the "mixed conception that a constricted vessel has its blood-pressure raised."

I beg to refer him to the writings of Dr. Geo. W. Crile, whose theory of the nature of shock depends upon the converse argument that a lowered blood-pressure must be caused by vascular relaxation. At the end of Dr. Crile's book on Blood-Pressure in Surgery, after all the experimental work has been recorded, there is a section headed "Argument" which begins as follows:—

"In shock the essential phenomenon is a diminution of the blood-pressure. Since there are no demonstrable lesions in the fatal cases, and no later effects in those that recover, we will assume exhaustion rather than structural lesions to be the cause of this fall. It must then be an exhaustion of the cardiac muscle, of the cardiac centre, of the blood-vessels or of the vaso-motor centres."

The author proceeds to show that the relaxation is not in the heart, nor in its nerves, nor in the arteries, and he concludes that therefore "shock is an exhaustion or break-down of the vaso-motor centre." Dr. Crile's theory is the popular one of the moment, and there are only a few who agree with me that it has no scientific basis, but whether it is right or wrong its author expresses no shadow of a doubt that lowered blood-pressure must be due to vascular relaxation. It would necessarily follow that a constricted vessel has its blood-pressure raised, and that this view must have a considerable popularity, as Dr. Wm. Russell suggests.

I have repeatedly urged that vascular contractions may be associated with a lowered blood-pressure. I first put this point forward, and attached to it all the importance I was able to express, fifteen years ago. I then wrote "that in inflammation, in shock, and in abdominal distension following a laparotomy contraction of the arteries is brought about by a reflex physiological mechanism;

1 Blood-Pressure in Surgery, by Geo. W. Crile, p. 401.
2 Ibid., p. 401.
3 The Lancet, Feb. 25, Mar. 4, Mar. 11, 1893; Aug. 26, 1905; vol. i. p. 497, 1907. The Brit. Med. Journal, Dec. 9, 1905. The Med. Chi. Trans., 1907.
that this contraction of the arteries, while raising the blood-pressure in the large vessels, accounts also for the smallness and feebleness of the pulse in such vessels as the radial; and consequently that this condition is due not to cardiac weakness but to the relation which the cardiac strength bears to the obstruction produced by the contraction of the medium-sized and small arteries. On another page I wrote that in certain cases "death is due to an excessive contraction of the arteries."

I think it was a new idea that the direct cause of death in certain conditions is a contraction of the arteries. Possibly Dr. Russell will not care to go so far, but I would point out that neither the full, bounding pulse of a sthenic fever, nor the weak, feeble pulse of the typhoid state is inconsistent with my view expressed above. Indeed, when a patient dies in a septic condition from a fever which is at first of sthenic type, it seems to me indisputable that there is at a very early stage a contraction of the arterioles and a big, bounding, perhaps a slow, pulse at the wrist. Later the contraction extends to or beyond the radials, and the pulse becomes smaller. At some time, varying in different patients, the heart becomes overworked by the increased resistance and its action hastens. The arterial contraction continues, increases, and extends to larger vessels, until finally the pulse may cease to be felt at the wrist many hours before the patient dies. The contraction of the arteries is the primary cause of the pulse condition. The heart, the action of which is often described as feeble, works, I believe, with tremendous power but with increasing effort to the very end. To me it seems to be certain that in the conditions under consideration the heart at the moment of death is able to go on beating; there is no discovered or described reason why it should not do so, and it would go on beating if death did not take place, because the cardiac muscle is not able to overcome the resistance of the contracted arteries, which gradually exclude the blood from the tissues and finally from the brain, starvation of which is the determining cause of death.

In the days when a 1-in-40 solution of carbolic acid was carefully and freely applied to all raw surfaces, and even to exposed peritoneum, and when a chilling spray was directed upon all wounded tissues during the whole of an operation, symptoms of shock were common and inflammatory reaction was often severe,

1 *The Physiology of Death from Traumatic Fever*, J. & A. Churchill, 1893, pp. 47-48.
2 *Ibid.*, p. 86.
even when no pus formed. When shock was thus gradually induced I have frequently traced all the changes above described until there was no perceptible pulse, and have watched the gradual return to normal conditions. Such changes are most easily distinguished in certain cases in which the heart's action continues to be slow. Both in cases of severe shock and in those of intestinal distention after a laparotomy I have observed a slow heart action with powerful impulses in the carotids and a hardly perceptible radial pulse.

It is obvious that in the conditions described there must be a stage both in the fatal septic process and in shock when the Riva Rocci hæmadynamameter will indicate a rise of blood-pressure, and that later it will indicate a fall. Such readings have been recorded and have been interpreted as meaning that at first there is a contraction of the arteries, followed later by their relaxation. I cannot accept this explanation. Vascular relaxation should give rise to an improved radial pulse if the arteries are previously contracted, and this improvement only takes place if the conditions under discussion are being recovered from. Unless there is heart failure a falling blood-pressure without an improvement in the peripheral circulation must be due to a greater vascular contraction. The other interpretation seems to me to be founded upon the "mixed conception" above alluded to, and condemned both by Dr. Gibson and by Dr. Russell.

Further confirmation of my view is found in the obvious anaemia of convalescents from prolonged fevers, and in the urgent necessity for introducing fluids into the body to insure a speedy recovery from the state of shock. After a fever the blood as a whole is reduced in quantity, and as the vascular system relaxes the disproportion between the bulk of the blood and the vascular capacity is a source of danger until new blood is formed. In shock the more watery parts of the blood serum are rapidly eliminated, and when the cause of shock is removed little more than a fresh supply of fluid to fill the distending vessels is necessary for recovery.

There is much in Dr. Russell's ingenious book that is to me novel, instructive and exceedingly interesting, but in my opinion he has not even now fully stated the power and importance of contractions of the normal vessels in many common conditions. I trust, however, that his able exposition will lead to a universal acceptance of the clinical fact that a small, feeble pulse and a lowered blood-pressure in the peripheral arteries, extending
also to the larger vessels, may be and very commonly are due to vascular contractions.—I am, yours faithfully,

13 Portman Street, London, W., 14th July 1908.

JOHN D. MALCOLM.

BIRTHS AND DEATHS IN EIGHT OF THE PRINCIPAL TOWNS OF SCOTLAND DURING JUNE 1908.

During the month of June 1908 there were registered in the towns of Glasgow, Edinburgh, Dundee, Aberdeen, Paisley, Leith, Greenock and Perth the births of 4532 living children, of whom 2310 were male and 2222 female. These births are 300 more than those registered in May, and 465 more than those registered in June of last year. The equivalent annual birth-rate is 30.0 per thousand of the estimated population of the 8 towns, a rate which is 2.9 more than that for May, and 2.7 more than that for June of last year. The birth-rate for the month in these towns ranged from 36.0 in Greenock to 22.8 in Perth. In Leith it was 32.6; in Dundee 32.3; in Glasgow 32.0; in Paisley 31.4; in Aberdeen 27.1; and in Edinburgh 24.0.

Of the 4532 births, 312, or 6.9 per cent., were illegitimate. This illegitimate rate is 0.2 above that for the previous month, but 0.8 below that for the corresponding month of last year.

Deaths registered in these eight towns during the month numbered 2184, 1130 being of males and 1054 of females. These deaths are 250 fewer than those registered in May, and 239 fewer than those registered in June of last year. The equivalent annual death-rate is 14.5 per thousand, a rate which is 1.1 less than that for May, and 1.8 less than that for June of last year. The death-rate for the month ranged from 16.5 in Greenock to 11.4 in Leith. In Dundee it was 15.9; in Perth 15.2; in Edinburgh 15.0; in Glasgow 14.6; in Paisley 14.4; and in Aberdeen 12.1. In Edinburgh, Paisley and Greenock the death-rate for the month was above that for the previous month, May, but in the remaining five towns it was below that for the previous month.

Of the 2184 deaths, 664, or 30.4 per cent., were of children of less than 5 years old; and 548, or 26.7 per cent., were of persons of over 60. The former are 157 fewer than in the previous month, and 167 fewer than in the corresponding month of the previous year; the latter are 51 fewer than in the previous month, but 4 more than in the corresponding month of the previous year.

Deaths of children of less than 1 year old numbered 390; they are 103 fewer than those deaths in May, and 83 fewer than those deaths in June of last year. The infantile mortality-rate amounts to 99 per thousand of the average monthly number of births in these