1st East Anglian Field Ambulance, Royal Army Medical Corps: Captain Ernest V. Gostling to be Major (dated March 13th, 1913).

2nd East Anglian Field Ambulance, Royal Army Medical Corps: Thomas Aloysius Flynn to be Lieutenant (dated May 5th, 1913).

3rd Home Counties Field Ambulance, Royal Army Medical Corps: Alexander Hampton Brewer to be Lieutenant (dated May 2nd, 1913).

1st Wessex Field Ambulance, Royal Army Medical Corps: Frank Arthur Roper to be Lieutenant (dated April 22nd, 1913).

1st Southern General Hospital, Royal Army Medical Corps: Captain Walter J. Clarke resigns his commission (dated June 4th, 1913).

6th Battalion, The South Staffordshire Regiment: Surgeon-Captain Frederick Edge resigns his commission, and is granted permission to retain his rank and to wear the prescribed uniform (dated June 4th, 1913).

Attached to Units other than Medical Units.—Lieutenant Cecil Johnson to be Captain (dated Dec. 4th, 1912).

For attachment to Units other than Medical Units.—James Henry Crane to be Lieutenant (dated Jan. 22nd, 1913). Ernest Stanley Stork to be Lieutenant (dated April 23rd, 1913). Major James Stratton Warrack, from the 1st Home Counties Field Ambulance, Royal Army Medical Corps, to be Major (dated June 4th, 1913).

SPECIAL RESERVE OF OFFICERS.

Royal Army Medical Corps.

The under-mentioned Lieutenants to be Captains: William H. L. McCarthy (dated May 17th, 1913) and John Inkster (dated May 24th, 1913).

THE JAPANESE NAVAL MEDICAL DEPARTMENT IN THE RUSSO-JAPANESE WAR.

In the *Archives de Médecine et Pharmacie Navales* for March Médecin de 1re classe (Staff-Surgeon) Dr. Chemin presents a seventh, and, it is said, a final, instalment of his interesting translation of the official medical history of the Japanese Navy in the Russo-Japanese war. The war lasted from Feb. 9th, 1904, to Oct. 16th, 1905, and the average daily strength of the navy was 42,500, of whom there were in all killed or wounded 3692.

	Total.	Per 1000.
Killed	1883	44·7
Died from wounds	127	2·8
Invalided	153	3·5
Recovered	1529	35·8
	3692	86·8

The table speaks volumes for the efficiency of the Japanese surgeons. Included in these totals are the 1080 men drowned and 143 injured in the sinking of their ships. Of those actually damaged in battle half were injured in one or other of the three great battles, and their disposal was as follows:—

Casualties in the Fleet Actions per 1000 Injured or Killed.

	Russo-Japanese war.	In our French wars about 1800.
Killed in the three great battles	165	250
Died from wounds	55	75
Invalided	75	95
Recovered and went to duty ...	705	580
	1000	1000

Killed means people who died without having their wounds dressed even once; they were more numerous formerly, the ligature being scarcely known. The ships that lost most heavily were the *Yoshino* class, who had 37 per cent. casualties; the marine artillery at the siege of Port Arthur lost 35 per cent.; and the battleships 27 per cent., as did the torpedo flotillas. Admiral Togo's proud flagship *Mikasa* lost in battle more heavily than any other ship, 28 per cent., but 86 per cent. of the *Hatsuse's* crew were drowned when she foundered. Officers lost 17 per 1000; the navigating staff, always on the bridge (perhaps that is why), lost far more than any other group. The engine-room staff had few wounds, but many deaths from drowning. The painstaking Japanese have divided the injuries into two groups, 2840 the direct result

of the war (shells, fragments of ship, bullets and drowning) and 852 indirectly due to the war—rupture of membrana tympani or crush of hand by the mechanism of one's own gun. There are very neat and elaborate tables which will be useful to historians, and the medical profession should be grateful to the Japanese surgeons for so carefully putting so many interesting facts on record.

UNITED SERVICES MEDICAL SOCIETY.

The annual general meeting of this society will take place at the Royal Army Medical College, Grosvenor-road, S.W., at 5 P.M., on Wednesday next, June 11th.

Correspondence.

"Audi alteram partem."

THE OLIVER-SHARPEY LECTURES.

To the Editor of THE LANCET.

SIR,—In common, I feel sure, with all who are interested in cardio-vascular physiology and pathology, I have read with pleasure and instruction Dr. A. D. Waller's Oliver-Sharpey Lectures on the Electrical Action of the Human Heart, reported in your issues of May 24th and 31st.

I shall not presume to offer any remarks on the purely physical section of his lectures, in which he has almost succeeded in rendering mathematics tolerable to the uncalculating mind, which is relieved when it discovers that the learned lecturer appears to have borrowed his phonetic symbol for the cantering rhythm from the Bucolics of Virgil, for I venture to think that Dr. Waller could complete the melodious line in which "*tegmine*" is the ultimate dactyl.

It is, indeed, upon this canter that I chiefly desire to comment, only premising my remarks on this subject by stating that I have also read with much interest Dr. Waller's account of auriculo-ventricular dissociation on pp. 1515-16, and merely remark in this connexion that I do not think the anatomy of the auriculo-ventricular bundle is yet sufficiently determined to be assumed to be common knowledge, nor, such as it is, a final conception of that structure.

The cantering rhythm is, as we know, a very common phenomenon met with at all ages and in hearts both with and without organic valvular disease. Explanations of the canter—that is, of the so-called "third sound"—which have hitherto been given have not been generally accepted. A year ago I asked an expert electrocardiographer what Einthoven's instrument had to say to the matter, but he confessed he had not noted the sign specially. It was with much pleasure, therefore, that I observed that Dr. Waller had dealt so fully with this sign, so well known to clinicians. His term "*hemitachycardia*" is, I think, likely to become current, and it is certainly to be preferred to "*cantering*," "*galloping*," or any other sesquipedalian word. One occasionally has the good fortune to meet with cases which in themselves reveal the whole gamut of cardiac irregularity, and are in themselves an epitome of variations in cardiac motion.

THE LANCET of Oct. 8th, 1910, contains a minute study of such a case which was under my care at the Great Northern Central Hospital, under the title "*The Meaning of the Audible Signs in Mitral Stenosis*." This case exhibited tachycardia, bradycardia, cantering rhythm, complete ventricular irregularity (auricular fibrillation), and the normal rhythm with which it was discharged from hospital. The only part of this paper to which I shall refer is my explanation of the cantering, or, as I called it, the triple rhythm, for which *tegmine* with a long and soft final vowel is certainly a more euphonious symbol than the "*lup-lup-dup*" which I employed. Dr. Waller makes the final vowel short, which distinguishes a quick from a slow canter, although the canter cannot be quickened much without being abolished and giving place to the ordinary first and second sounds of the heart, a condition which is constantly induced by making a patient evincing the canter in the recumbent position sit up in bed, when, with the acceleration and augmentation dependent upon movement, the added sound frequently disappears. In the paper which you published, and which was originally

read at a meeting of the Association of Physicians of Great Britain and Ireland held at Liverpool, my view that the third sound "was due to an unregistered but regularly recurring ventricular extrasystole" was received with some scepticism, and while I do not think that my explanation accords altogether with Dr. Waller's, I am glad to note that he also attributes the added sound in the canter to a ventricular extrasystole of regular recurrence.

In my paper I also mentioned a case of quadruple rhythm or cor bigeminum, which one could at will convert into the triple or cantering rhythm, and again reproduce the bigeminal heart according as one directed the patient to stand or lie. The quickened rate of standing eliminated the first short diastolic pause and left systole, extrasystole, and long diastole to account for the three sounds, while the resumption of recumbency also reproduced the eliminated short diastole and gave systole, short diastole, extrasystole and long diastole to account for the quadruple sound or bigeminal heart. In the text case of the paper there was, however, no quadruplication of sounds, but the triple canter in which a ventricular extrasystole was considered to interpose between the normal first and second sound of the heart, and produce the unregistrable but audible third sound. This I still believe to be the explanation of the sign, and it only requires the elimination of the extrasystole to give the normal rhythm. With increased force and acceleration of the heart beat this occurred in the case in question. Dr. Waller¹ in his electrocardiogram of the canter shows *two* complete cycles followed by an extrasystole, and while remarking that it was "odd" only one sound should accompany these complete cardiac cycles, adds that it is by no means unusual. Personally, I venture to think that it is not only odd but also very unusual.

The heart of this case as shown² when "hemitachycardia" was fully established, was probably quicker than in Fig. 20, for the spacing between the apices of the extrasystoles is considerably less, and, admittedly, there is only *one* complete cycle in the interval. In this case, also, there are only two sounds registered which I take to be the first and second sounds of the heart, for the spacing of the sounds is the same as in Fig. 19. Here I would suggest that the third sound, which is essential to the canter, was certainly due to the extrasystole which is registered, but the sound itself has not been reproduced by the phonograph, although it was doubtless audible to the ear. So that since I read the paper to which I have referred we have advanced in the appreciation of the conditions underlying the cantering, triple, or hemitachycardial rhythm, in so far as we have now graphic evidence of the extrasystole causing the "third sound," but we have not yet succeeded in registering, except by the ear, the tone sound caused by the extrasystole.

Is it not possible that the "large electrical effect" of the extrasystole, which, as Dr. Waller remarks (p. 1517), "gives no pulse wave," may also fail to influence the phonograph, and probably for the same reason—namely, because it occurs while the ventricle is comparatively empty of blood? That the *audibility* of the heart sounds is largely dependent upon the presence of blood in the heart and large vessels was long ago demonstrated by Halford (1860), and this view is not invalidated by the fact that cardiac muscle is not without a share in producing the same phenomena. One word more.

Dr. Waller (p. 1519) dismisses as unimportant the question as to the initiative of nerve or muscle in the production of ventricular extrasystole. If "it matters little," and I would respectfully question this opinion, even when expressed by one who by inheritance and personal distinction is so closely associated with authority in physiology, it is certainly a point of very considerable interest, and may matter more when we know more of the conditions underlying cardiac sensibility and motion. The former of these is at present largely denied, and the latter, for reasons which do not appear to some sufficient, endowed with a degree and kind of automatism which, if true, would render the existence and influence of the nervous system rather superfluous. But the absolutely regular recurrence for a lengthened period of an independent ventricular extrasystole, together with the very power I have mentioned, posturally to convert a bigeminal heart into one with a

cantering rhythm, and the latter into normal action, appears to me to indicate so intimate a control, reflex and direct, of all the phenomena usually described as "properties" of muscle, as to make the consideration of the neural factor in cardiac sensibility and motion a matter not only of scientific interest but of practical moment.

I am, Sir, yours faithfully,

ALEXANDER MORISON.

Upper Berkeley-street, W., June 2nd, 1913.

THE PHYSIOLOGY OF THE OPEN-AIR TREATMENT.

To the Editor of THE LANCET.

SIR,—My delay in answering Dr. Francis Hare's inquiry in THE LANCET of May 17th is due to the need I felt of investigating the original papers of Robin and Binet and finding out on what experimental facts they based the conclusions cited by Dr. Hare from "Progressive Medicine." Robin and Binet found an increased respiratory exchange and increased absorption of oxygen—which did not appear as CO₂ in the expired air—in tubercular patients with the exception of cases of meningitis and peritonitis. They also brought forward some evidence to show that the children of tubercular parents have a higher respiratory exchange, but their evidence on this point is very inconclusive. They conclude that the increased exchange and absorption of oxygen is part of the defensive mechanism, and that the more the disease is accentuated and the more the organism is overcome by the attack of the bacillus the less is the oxygen absorbed by the tissues. By the expression "oxygen absorbed by the tissues" they mean oxygen breathed in over and above that which appears as CO₂. It is to be expected that the febrile condition of consumptives would increase the respiratory exchange, the breaking down of tissue substance, and absorption of oxygen by the tissues.

I should not, however, be prepared to accept the figures arrived at by Robin and Binet without further investigation carried out by the exact methods recently introduced by Benedict, or by Douglas's excellent method of studying respiratory exchange. Whatever may be the results of such further inquiry, the fact remains that metabolism includes anabolism and katabolism, and that I had both in view when I discussed the influence of open air and exercise on metabolism. Anabolism and growth take place as the result of katabolism. We cannot feed a muscle strong, but we can develop it by exercise. The katabolic products excite the vigorous circulation and respiration, and the excretion of these products renews the appetite. The katabolism produced by the invasion of the bacillus differs from the normal katabolism; the latter, provoked by cool moving air and exercise, builds up and strengthens the defensive mechanisms. It goes without saying that the exercise must not be carried to the point of exhaustion.

I am, Sir, yours faithfully,

LEONARD HILL.

London Hospital Medical College, June 3rd, 1913.

THE LATE DUKE OF ABERCORN AND THE WEST LONDON HOSPITAL.

To the Editor of THE LANCET.

SIR,—With the concurrence of His Royal Highness Prince Arthur of Connaught, President of the Hospital, we ask you to be good enough to allow space in your influential paper to bring under the notice of your readers the following particulars concerning an effort which is now being made in memory of the late Duke of Abercorn, who as the former president and chairman of the hospital was throughout a lengthened period closely associated with its daily life and work.

It is generally felt that no more fitting tribute can be paid to his memory than by erecting new accommodation for the nursing staff and to name it the "Abercorn Home for Nurses." The matter was submitted to His Royal Highness Prince Arthur of Connaught on the occasion of his visit on May 21st, and he then authorised us to state that it met with his entire approval. At present the nurses are crowded into seven private houses in various parts of the hospital premises.

¹ THE LANCET, p. 1517, Fig. 20.

² Loc. cit., p. 1519, Fig. 22.