to save the mother, I had great difficulty in extracting the fetal head from the lower thinned-out uterine cavity. The retraction ring held firmly, although the patient was deeply under morphine and chloroform. It was also interesting to note at the time of the operation that, although there was a firm band of uterine muscle around the neck of the fetus, there was no sign of this externally. I opened the upper cavity of the hourglass and caught a leg. I had to deliver the fetal head, which was below the muscular bar, by jaw traction. I mention this fact because it shows the persistence of the muscular tetany in a collapsed patient under both morphine and chloroform.

It will be noticed that I have made no reference to textbooks or papers. I wish only to give a clear clinical account of a condition which I know occurs time after time without being recognized. Many practitioners may go thirty years without seeing a case of this kind, or never see one at all.

In one of the latest books on obstetrics this condition is lightly touched upon. The author states that it is due to (1) the administration of ergot, or (2) to spasm, caused by repeated attempts at delivery. He recommends the administration of morphine gr. $\frac{1}{2}$, deep anaesthesia, and appropriate means to overcome the obstruction.

I have never seen any cases in the early stages of labour, but from the careful accounts given by the doctors in charge I feel sure that the retracting barrier had already formed when forceps were first applied. Again, the measurements of the brim in each case had been taken, and if the real trouble had been simply a mechanical obstruction at this point, the fetal head could have been delivered either with forceps or with the cephalotribe. I have seen a patient who had been more or less under deep anaesthesia and morphine for ten hours, yet the retracting uterine muscle still gripped the neck of the fetus. None of the cases had been given ergot. Of the four cases I have seen, only one recovered. Two died within a few hours of delivery, while the last died suddenly of pulmonary embolism on the fourth day of the puerperium. In my former paper I suggested that decapitation with delivery of the fetal head, followed by version, was a means of effecting delivery, but in the light of further experience I am now convinced that Caesarean section is the safest and best method of treatment. The earlier this is done the better.

CONCLUSIONS.

I would make the following suggestions:

1. Be on your guard when dealing with a short, thick-set woman with a square head and short long bones.
2. A high position of the fetal head, occurring in a primipara, should at once denote serious trouble.
3. Measure the diameters of the pelvis, but do not place too much store upon your estimate. The brim may be obliquely distorted, and this will prevent the head from entering the brim and at the same time will not show up in your calculations.
4. Cut-and-dried rules on pelvimetry are useful only in severe contractions of the pelvis.
5. Remember that to apply forceps to a fetal head which is movable and high above the brim is an obstetric operation requiring the most careful consideration, and should only be undertaken after careful examination of the condition of the uterine muscle around the neck of the fetus. If the examining hand can be passed easily beyond the shoulders of the fetus, an attempt at delivery with instruments may be made. If there is the least evidence that the uterine muscle is retracting round the neck of the fetus, Caesarean section should at once be performed.
6. When once the retraction ring has formed around the neck of the fetus, it will grip it firmly until the patient is almost at the point of death.

P.S.—Since writing this short paper I have seen another case. In spite of much bruising and laceration of the vagina an earlier diagnosis rendered it possible to save both mother and child by Caesarean section.

WE are glad to learn that Professor Kocher of Berne has recovered from a severe attack of double pneumonia and pleurisy which he experienced in the spring and is resuming his work as professor of surgery. His recovery, at the age of 75, from so severe an illness is evidence of the vigour of his constitution and will be a source of great gratification to his many friends in this country.

BREATHLESSNESS IN SOLDIERS SUFFERING FROM IRRITABLE HEART.

BY

THOMAS LEWIS, M.D., CAPTAIN COTTON, C.A.M.C.,
J. BARCROFT, F.R.S., T. R. MILROY,
D. DUFTON, AND T. R. PARSONS.

(From the Hampstead Military Hospital and the Physiological Laboratory, Cambridge.)

(Report to the Medical Research Committee.)

BREATHLESSNESS may arise from any one of a great number of causes, some of which, like mechanical impairment of the heart or lungs, are apparent, while others are more obscure. The immediate stimulus to the respiratory centre is in most cases a chemical one, so that the difference between the cases in which the cause of breathlessness is explicable and those in which it is obscure depends on whether there is an evident clue to a chemical change in the blood which may be transferred to the respiratory centre. Thus, in the commonest physiological type of breathlessness, that due to muscular exercise, carbonic and lactic acids are passed into the blood and stimulate the respiratory centre; in the case of patients recently gassed the want of sufficient functional lung surface dams back the carbonic acid in the blood and so produces breathlessness. More obscure, because the cause has to be sought directly in the blood itself, is the condition of acidosis, though this, in respect of its symptom of breathlessness, falls into the same group with that of the previous cases, since in all alike the exciting stimulus to the respiratory centre is a relatively acid condition of the blood.

The object of the present paper is to draw the attention, and especially, at the present time, of those charged with the care of military patients, to another and more subtle cause of breathlessness. The reason for our doing so is that we have found it to exist in five out of six cases in which the cause of the breathlessness present was so far in doubt as to make a systematic examination of the chemical properties of the blood desirable. The cause of breathlessness to which we refer is the absence of an adequate supply of "buffer" salts in the blood. The patients in which this condition is present are to be found amongst those who are diagnosed as having "irritable hearts" or "trench hearts," that is, among the number of soldiers invalided during the period of training or while on active service because they are unable to tolerate physical exercise. In these cases exertion produces some of the following symptoms—namely, excessive fatigue or actual exhaustion, attacks of giddiness or actual fainting, palpitation and pain usually situated in the precordial region. The patients to whom we refer show no constant physical signs, but in the majority of them the heart rate is abnormally high when the subjects are up and about. Moderate exercise increases the pulse-rate excessively. It is the rule to find high respiratory rate in these subjects while they are at rest, and in some cases this rate may be as high as 40 per minute.

Let us now turn to the consideration of "buffer" salts, and to some explanation of the part which they play in the pathology of the cases under consideration.

The blood is at all times receiving and parting with acid and alkali. These variations in constitution, if effected in an ordinary saline solution, would produce changes in relative acidity and alkalinity which would be physiologically intolerable; their effect on the brain alone would be to excite or depress its various centres to extreme limits. The respiratory centre, for instance, would be stimulated to violent dyspnoea by the acidity resulting from trivial exercise; it would be brought to a standstill by a moderate acquisition in alkali. A given addition of carbonic acid to the blood only produces a quarter of the change in reaction which it would produce in a corresponding quantity of physiological saline solution. Thus, if 100 c.cm. of blood be taken and the same quantity of normal salt solution, 1.3 c.cm. of carbonic acid dissolved in the saline would change its reaction to the same extent as 5 c.cm. would change that of the blood. The reason of this is that the blood contains such substances as sodium bicarbonate, monacid and diacid sodium phosphates, and

protein, which tend partly to fix the acid so that in a sense it loses its chemical freedom. For the sake of example the attention may be confined to the two phosphates, Na_2HPO_4 and NaH_2PO_4 . If a little carbonic acid is added to a solution containing these two salts, a little sodium bicarbonate is formed, which is alkaline. The sodium is acquired by the CO_2 , at the expense of the phosphates, so that there is less of the alkaline Na_2HPO_4 , and more of the acid NaH_2PO_4 than before. The net result is a slight increase in the acid reaction. The blood forms a more complicated system of this type. The salts to which we have alluded are therefore called "buffer" salts, because they ease the shock to the reaction of the fluid caused by the addition of acid or alkali.

The bearing of these general observations on the cases of breathlessness which we have studied is perhaps best explained by recounting the history of our investigation of a single case—that of A. The case was referred to us by Lieutenant Drury, R.A.M.C.,* with the request that we should test it for acidosis. We found:

1. That when all the CO_2 was shaken out of the blood it was somewhat more alkaline than normal blood.
2. That with the amount of CO_2 normally present in the body it was just on the acid side of normal; and
3. That any further addition of acid increased the acidity to an abnormal degree.

We therefore tested other cases which presented a similar clinical picture in the same way, and we found that out of six cases tested and compared with four normal men of similar age the changes in reaction of the blood were as follows:

Change in Blood Reaction for the Addition of 5 Volumes of CO_2 per 100 of Blood under the Conditions Prevalent in the Body.

Normal Persons.			Dyspnoeic Patients showing Typical Symptoms of "Irritable Heart."		
R.	...	0.045 units	Z.	...	0.155 units
C.	...	0.063 "	A.	...	0.102 "
O.L.	...	0.050 "	J.	...	0.119 "
P.	...	0.051 "	R.R.	...	0.102 "
			G.	...	0.086 "
			T.*	...	0.064 "

Of these T.* proved to be a case in which the reaction of the blood was less alkaline throughout than normal. His dyspnoea was therefore explicable on other grounds than those of insufficient buffers.

The units used in the above table are those familiar to chemists as to the alteration in the logarithm of the concentration of hydrogen ions in the blood. We prefer to state the case in the following way:

If the average change in the acidity of the blood when five volumes of carbonic acid are added be called 100 the changes observed in the case of the five patients whose cases are not otherwise accounted for are:

Change in Relative Acidity expressed as a Percentage of that of Normal Men.

Z.	A.	J.	R.R.	G.
290	200	220	200	150

These results were all obtained by an indirect method of determining the blood reaction based on the affinity of the blood for oxygen; but as it was clearly unsatisfactory that a result so important should depend on data obtained by

* The present research arose from one conducted by Lieutenant Drury, R.A.M.C., on the carbonic acid in the alveolar air and the effect of breathing small quantities of that gas. As he was shortly afterwards detailed for service abroad, we lost his very valuable assistance.

† Variations in the "buffer" value of human blood have been found by Levi and Rowntree.—*Archives of Internal Medicine*, vol. xvii, p. 525.

a single method, the direct determination of the concentration of hydrogen ions by the electrometric method was made on one of these cases, Z., and compared with that of two normal cases.

Change in Reaction produced by addition of 5 Volumes of CO_2 per 100 of Blood measured by Electrometric Method.

Normal.			Dyspnoeic.		
R.	...	0.045 units	Z.	...	0.150 units
P.	...	0.05 "			

Expressed as a percentage of the normal, therefore, the change in acidity of Z.'s blood would be about 320 per cent. of the normal.

The measurement of the quantities involved is near the experimental error of whatever method may be employed for the purpose, yet we consider that the agreement in the figures is striking enough to warrant our conclusions. Nevertheless, we have obtained additional confirmation of our argument from the fact that we were actually able to impose the properties of normal blood upon that of one irritable heart patient by the addition of "buffer" salts. The blood of J. before we did this had a change of reaction of 0.119 as compared with the normal 0.045 to 0.06.

The vertical figures denote the logarithms of the concentration of hydrogen ions in the blood (pH).

It was compared with the blood of J. B., a much older person, normal in the sense that he is not unduly dyspnoeic for his age on taking exercise, though he possesses a history of functional cardiac trouble. The comparison is shown graphically in the figure in which the reaction of the blood is plotted vertically in units corresponding to pH .

The line indicated by the letters JB-JB shows the changing reaction of J. B.'s blood when it contains the quantities of carbonic acid shown successively by the abscissae. The line J-J shows that of a case of irritable heart. Note that the line, especially at the lower end, which represents the usual carbonic acid conditions in the body, is much steeper in the case of J. than in that of J. B. When disodium hydrogen phosphate (Na_2HPO_4) is added to the blood of J. to the extent of 0.003 gram per c.cm. the inclination of the line is changed, as shown in the dotted line O-O, whilst if 0.003 gram of sodium phosphate per c.cm. and 0.0008 gram of sodium bicarbonate be added, the points marked J' J' are obtained, which are nearly on the curve of the person (J. B.) with normal respiratory phenomena.

These observations seem to us to be important, first of all because they demonstrate an altered state of the blood which goes far to explain the otherwise inexplicable breathlessness in certain patients who suffer from the condition described as irritable heart. They are important from another standpoint. The temptation to cover our ignorance of the pathology of the malady as a whole by referring many of its symptoms to instability or to irritability of the central nervous system was a powerful one, so long as clear deviations from normality could not be found. Our observations carry us from the abstract to the concrete, and tell us that we deal with an affection in which disordered metabolism plays a conspicuous rôle. If we are inclined hastily to judge the temper of our fellow-men, holding that poorness of spirit and lack of zeal contribute to the histories of physical inefficiency as they are related to us, such findings bid us beware, reminding us that there may be no more potent restraint to the display of spirit than the early conditions of ill health.

THE ESTIMATION OF MYOCARDIAL EFFICIENCY.

By J. STRICKLAND GOODALL, M.B., M.R.C.P.LOND.,
F.R.C.S., F.R.S. EDIN.,

LECTURER ON PHYSIOLOGY AND SUB-DEAN, MIDDLESEX HOSPITAL;
ASSISTANT PHYSICIAN, NATIONAL HOSPITAL FOR DISEASES OF
THE HEART, LONDON; CONSULTING CARDIOLOGIST, RED
CROSS AND AUSTRALIAN AUXILIARY HOSPITALS.

THIS article is based on 2,000 observations made on healthy and diseased hearts during the past five years.

The majority of those made on the "healthy" heart, and the reactions worked out in connexion with them, were carried out on students in the Middlesex Hospital, and to all those gentlemen who so willingly and unselfishly sacrificed their Saturday afternoons in order to climb endless stairs—often to the point of extinction—the writer offers his most grateful and sincere thanks, and assures them that their time was not wasted. In this connexion he would also thank Mr. Dale Richards for his assistance in some of the work. The observations on pathological hearts were carried out partly on my own private patients and partly on cases at the National Hospital for Diseases of the Heart.

The measure of a heart's efficiency is its capacity for doing work, and this is true whether one is dealing with the physiological heart of the healthy young adult, the worn-out heart of the old man, the "super" heart of the well-trained athlete, or the pathological heart, the seat of definite valvular disease, myocardial atonia, inflammation, or degeneration. Furthermore, it must be clearly borne in mind that many hearts work perfectly, exhibiting no defects at all, when the person is at rest, but exhibit serious derangements and reveal definite evidence of myocardial impairment when called upon to do extra work under load. Such defects are usually revealed by the development of irregularities—murmurs, pulsations, etc.—which did not previously exist.

WORK REACTIONS.

(a) The Healthy Heart.

The physiological heart of the healthy young adult responds to increased work by increased contraction—the rate increases, the blood pressure rises, and the respirations increase. The increase in the heart-rate, in the blood pressure and respirations, is gradual, while a definite relationship is still maintained between the pulse-rate and respirations. The rise in the pulse rate, in the blood pressure, and respirations is proportional to the amount of work done, but it is rare in healthy people to be able to raise the heart beat beyond 180 a minute excepting by the most violent forms of exertion.

The height to which the blood pressure rises is, apart from training, etc., largely influenced by age, but for a healthy young adult 220 mm. of mercury may be taken as the limit. On ceasing exercise the heart-rate, blood pressure, and respirations, fall rapidly; the blood pressure usually reaches normal (that is, starting point) before the heart-rate. The respirations being the first to return to normal.

(b) The Athlete's Heart.*

The slow "super heart" of the well-trained athlete also responds to increased work by increased contraction and a raised blood pressure, but whereas the heart of the "ordinarily" trained individual gradually increases in frequency, that of the athlete suddenly "practically" doubles its rate.

(c) The Diseased Heart.

The pathological or diseased heart in which the myocardium is at all impaired responds to increased work, not by increased contraction, but by dilatation, so that, although the frequency is increased (often out of all proportion to the amount of exercise taken), the blood pressure either fails to rise or actually falls, according to the amount of myocardial damage present and the amount of work done. Furthermore, the dyspnoea and general distress are often marked, while the normal pulse-respiration ratio is frequently disturbed. Finally, irregularities of various kinds (such as extrasystoles,† auricular fibrillation, and alternation, etc.), murmurs, or pulsations, not previously present, may make their appearance.

As regards the practical application of the above principles, the methods usually adopted by the writer and found to be quite satisfactory are briefly as follows:

1. The Simple Stair Test.

The patient is weighed and his weight recorded in pounds. He is next made to lie down on a couch, and his pulse-rate, systolic blood pressure (brachial), and respirations recorded as below:

NAME	SEX	AGE	WEIGHT	DATE
	PULSE	B.P.	RESP.	
NORMAL				
AFTER 1ST EXERCISE				
AFTER 2ND EXERCISE				

NOTES:—

He is next made to do a definite amount of work in a given time, his pulse, blood pressure, and respirations again being recorded immediately after his return to the couch. As it is most important to get the readings as quickly as possible, two observers are employed, one standing on either side of the couch; one observer takes the blood pressure (the armlet being left in position during the exercise and the manometer being a fixture on the wall), while the other takes the respirations, and whichever completes his observation first takes the pulse. These are recorded on the card as before. These observations are then repeated periodically—for example, every minute—until pulse, blood pressure, and respirations are normal.

For ordinary purposes a very convenient test is to make the patient raise his body weight 20 ft. in not longer than 30 seconds. This is easily done by making him run up a flight of stairs of the required number. His pulse, blood pressure, and respirations should be normal at the end of three minutes after the exercise, if his myocardium is in good condition. The work done (in foot pounds) is easily obtained by multiplying the body weight (in pounds) by the distance through which it was raised (in feet). For purposes of diagnosis it is not necessary to calculate the actual work done, but if the reaction is to be used for remedial purposes, this is essential. In examining the results obtained, the most important points to pay attention to is whether the blood pressure rises or not immediately after the exercise. For practical purposes the results can be classified as follows:

1. *Good reactions*, when pulse, blood pressure, and respiration all increase on exercise and are normal again at the end of three minutes.

	Pulse.	Blood Pressure.	Respiration.
Normal	116	152	22
After exercise	124	180	24
Three minutes after exercise	116	152	22

W. = 2,580 ft. lb. in 30 seconds.

* The writer is of opinion that different effects are produced on the heart by different kinds of exercise—for example, long distance cycle racing produces quite a different heart from rowing.

† Much difference of opinion exists as to the nature and significance of extrasystoles. The writer definitely holds the view that any heart which shows no extrasystoles when at rest, but systematically develops them on exertion, has a damaged myocardium.