alkalinity of the blood was the one recently devised and described by Dr. A. E. Wright.² This process is an extremely ingenious and reliable one, and possesses the great advantage of not requiring more than two or three drops of blood in order to make an accurate determination of its alkalinity.

I first estimated the percentages of alkalinity of specimens of blood drawn from twenty-five healthy male adults, which I found to vary from 0.161 to 0.185 per cent. of anhydrous sodium carbonate, the average being 0.167. This average I have taken as representing the normal alkalinity of the blood. In the following table (Table I) are shown the results of the determinations that I made of the alkalinity of the blood of a patient suffering from subacute gout; the estimations were made throughout the duration of the attack, mostly on alternate days. The normal alkalinity of healthy blood is shown for comparison at the foot of the table:

TABLE I.—Showing the Results of the Determinations of the Alkalinity of the Blood of an Adult Man during an Attack of Subacute Gout.

Dates of Determinations, and Relations to Treatment.			Alkalinity represented as Percentage of Anhydrous Sodium Carbonate present in the blood.
February 4	(Commencement of attack and before treatment was begun)	...	0.167
" 6	(Treated with colchicum only)	...	0.167
" 8		...	0.167
" 10		...	0.156
" 12		...	0.167
" 15		...	0.158
" 17	(Treated with citrate of potassium and iodide of potassium only)	...	0.158
" 19		...	0.167
" 22		...	0.180
" 24		...	0.173
" 26		...	0.161
" 28	...	...	0.179
March 2	...	...	0.167
Average alkalinity throughout the attack			0.166
Average alkalinity of healthy blood			0.167

These results show that throughout this attack of subacute gout the alkalinity of the blood was practically the same as the average alkalinity of healthy blood.

Another erroneous assumption that has been made by some writers is that variations in the acidity of the urine can be taken as a gauge of corresponding variations in the alkalinity of the blood, and that, therefore, a fall of acidity in the urine means an increased alkalinity of the blood, and *vice versa*. That this assumption is wrong is shown by reference to the following table (Table II), in which are arranged side by side the determinations that I made on the same days of the alkalinity of the blood and of the total acidity of the urine for each twenty-four hours. The case was the one of subacute gout referred to in the previous table.

TABLE II.—Showing the Absence of any Constant Relationship between the Alkalinity of the Blood and the Acidity of the Urine of a Patient during an Attack of Subacute Gout.

Dates of Determinations.			Alkalinity of the Blood represented as Percentage of Anhydrous Sodium Carbonate present.	Acidity of Total Urine for 24 Hours reckoned as Grammes of Hydrochloric Acid.
February 4	...	...	0.167	1.392
" 6	...	...	0.167	0.953
" 8	...	...	0.167	1.096
" 10	...	...	0.156	1.374
" 12	...	...	0.167	1.583
" 15	...	...	0.158	1.529
" 17	...	...	0.158	1.629
" 19	...	...	0.167	1.581
" 22	...	...	0.180	1.602
" 24	...	...	0.173	Alkaline
" 26	...	...	0.161	Alkaline
" 28	...	...	0.17	0.608
March 2	...	...	0.1	0.622

This table shows that no constant relationship existed in this case of gout between the alkalinity of the blood and the acidity of the urine; and, moreover, that on these days when, owing to treatment with citrate of potassium, the urine remained alkaline, there was no corresponding rise in alkalinity of the blood.

The determinations of the alkalinity of the blood and of the acidity of the urine of this case of subacute gout are shown in curves in the following figure, a glance at which demonstrates at once that no constant relationship existed between the alkalinity of the blood and the acidity of the urine:

Fig. 1.—Showing the absence of any constant relationship between the alkalinity of the blood and the acidity of the urine of a patient during an attack of subacute gout. A alkalinity of blood, B acidity of urine.

CONCLUSIONS.

The facts dealt with in this paper lead to the following conclusions: (1) The alkalinity of the blood of gout is not necessarily diminished during an attack; (2) no constant relationship exists between the alkalinity of the blood and the acidity of the urine during an attack of gout.

REFERENCES.

- ¹ *Deut. med. Woch.*, 1895, Band xxi, p. 655. ² *Lancet*, September 18th, 1897.

THE OUT-PATIENT TREATMENT OF INSANITY IN GENERAL HOSPITALS.*

By CROCHLEY CLAPHAM, M.D.,

Physician Superintendent, the Grange, Rotherham; Physician to the Royal Hospital, Sheffield.

A CERTAIN amount of provision for the out-patient treatment of mental disease amongst paupers has been made at our large county asylums, and good work has been done, but there remains a large number of patients of the class slightly above paupers who decline to present themselves for treatment at an asylum and whom it is advisable to provide for elsewhere.

Dr. Henry Rayner (late Superintendent of Hanwell), Lecturer on Mental Diseases at St. Thomas's Hospital, London, has endeavoured to fill this gap by instituting an out-patient department for mental diseases at that hospital, and has advocated the establishment of similar departments at other public hospitals. Dr. Rayner commenced his work at St. Thomas's early in 1893, and I was induced, by his representations and by a paper which he read at the British Medical Association meeting held at Newcastle in that year, to set up a corresponding department at the Royal Hospital,

* Read at a meeting of the Yorkshire Branch of the British Medical Association.

Sheffield, with which I am connected as Honorary Physician.

The weekly Board of the hospital having sanctioned the proceeding, I started in the autumn of 1893 by putting up a notice in the out-patient waiting room to the effect that patients suffering from mental disorder would be seen at 1.30 P.M. every Tuesday, and that it was advisable that patients of this class should be accompanied by a near relation. The response to my notice was immediate and continuous, and for the last four years and a half cases of mental disease have never failed to present themselves on the day set apart by me for their treatment. During this period every form of insanity has passed under my notice at the hospital, and with very gratifying results.

The great object aimed at in the establishment of this department has been to get at the insane of the lower classes in the early stages of their malady, which are so much more amenable to treatment than the more advanced ones. To this end I spoke in my notice of mental disorder and not of insanity, as the latter term is singularly abhorrent to these people.

The first thing to determine, on a case presenting itself, is whether or no it is a suitable one for out-patient treatment, or whether it would be more properly dealt with inside an asylum, thus providing the poor with expert advice gratuitously on a most important point.

Where I have thought it advisable to recommend asylum care, I have always found the relatives ready to fall in with my suggestion, though previously, perhaps, averse to taking action in the matter. However, the great proportion of patients presenting themselves have proved suitable for out-patient treatment and home management.

In the absence of such a department as I am describing, these patients would too frequently be put through a routine narcotic treatment, which might or might not be suited to the case; or, on the other hand, would receive no treatment whatever. Such as these are the cases which would be specially benefited by the advice and treatment of an expert mental physician attached to the staff of a general hospital; and I am anxious to support Dr. Rayner's contention that such a physician should, wherever practicable, be attached to at least one hospital in each large town.

My own experience is that patients of this order readily avail themselves of the facilities afforded, and are brought at an early stage of their disease to the hospital consulting room, with, in many cases, eminently favourable results. Dr. Rayner, in a letter to me on this subject, says: "What strikes me with wonder is the ease with which arrest can be effected in these early stages." I can quite bear out this statement.

Another function of this department is to render aid in the supervision and after-care of patients discharged as convalescent from county asylums with their mental equilibrium still somewhat unstable.

A CONTRIBUTION TO THE PHARMACOLOGY OF THE MAMMALIAN HEART.

By ARTHUR CUSHNY, A.M., M.D. ARBERD.,

Professor of Materia Medica and Therapeutics in the University of Michigan, U.S.A.

THE effects of drugs on the mammalian heart have hitherto been experimentally studied chiefly by an indirect method, the graphic registration of the arterial blood pressure by means of the manometer, the results thus obtained being interpreted by the aid of the changes observed in the frog's heart. Although the value of this procedure is undoubted, it seems high time that some of the direct methods which have given such valuable results in the hands of the physiologist should be introduced into the pharmacological laboratory. The numerous factors on which the tension of the arteries depends render deductions as to the condition of the heart very insecure, while the reaction of the frog's heart to poisons is often very different from that of the mammalian. A systematic examination of the effects of some of the more important drugs has therefore been carried on by direct methods in the pharmacological laboratory of this University during the last three years.

METHODS.

The rabbit's heart was observed directly by dividing the sternum and opening the pericardium, while the cardiometer of Roy and Adami was employed to register the movements of the cat's heart, and a somewhat modified form of their myocardiograph¹ was used in experiments on dogs. The rabbits were anaesthetised with urethane, the cats and dogs with morphine and chloroform acetone.

1. DIGITALIS SERIES.²

A number of drugs of this series (digitalin, digitoxin, strophanthin, antiarin, erythrophlocein, convallamarin, oleandrin, etc.) were examined, and all gave very similar results. After therapeutic doses, the rhythm of the heart was slowed, the relaxation of the ventricles was considerably increased, and their contraction rendered more complete. The auricular systole was generally weaker, while its relaxation appeared unaltered, although the last could not be measured accurately. The contraction volume, or amount of blood rejected at each ventricular contraction, was increased, the total output per minute augmented to a less extent.

When the vagi were paralysed by the injection of atropine previously, digitalis produced a very different result. The rhythm was then unchanged, while the ventricular systole became much more perfect than before, and its dilatation during diastole was either unchanged or was less than that before the injection. The changes in the auricle were identical with those in the ventricle. The contraction volume and the output per unit of time were augmented in equal proportion, and often to a much greater extent than when the vagi were intact.

The changes seen in hearts in which the inhibitory mechanism is uninjured are, therefore, due to two distinct and separate factors—the stimulation of the regulating apparatus (chiefly in the medulla oblongata) and the direct action on the cardiac muscle, which is observed in the heart after the former has been paralysed by atropine. The slow rhythm and increased dilatation of the ventricle are due to the inhibitory stimulation, which would also lead to a less perfect systole were this effect not opposed by the direct muscular action. In the auricle the inhibitory action overbalances the muscular, so that the latter at first appears to be wanting, but is shown to be present by the atropine experiments and also by more careful examination of the other tracings. The vagus effect would also lead to a lessened contraction volume and output were it not for the compensatory increase in the ventricular systole. It is to be remarked that the muscular action does not prolong the systole in the mammalian as it does in the frog's heart. The blood pressure rises in this stage, partly from the increased output of the heart and partly from contraction of the vessels.

Poisonous quantities of these drugs may cause two entirely different results, according to which of these two factors is thrown into the greater prominence. If the inhibitory stimulation is the more pronounced, the rhythm becomes extremely slow and somewhat irregular, the ventricular relaxation is more marked than after therapeutic doses, and the systole is often reduced in strength. The auricle generally ceases in diastole, and the output falls to a very low point. These symptoms are often accompanied by cyanosis and dyspnoeic and convulsive movements. The heart then gives a few rapid contractions and the ventricle passes into delirium. On the other hand, if the muscular action is the more highly developed, the ventricle assumes an independent rhythm, which undergoes rapid acceleration. If the auricle continues to beat at a different rate, there results a periodic variation in the extent of contraction and relaxation both in the auricle and ventricle, which gives a characteristic appearance to this part of the tracing. With advancing intoxication these periods become shorter and less regular, and, after a stage of extreme irregularity, the ventricle ceases its contractions and goes into delirium cordis, while the blood pressure, which has been slowly falling as the heart becomes more irregular, descends to zero.

2. ACONITINE³ AND VERATRINE.

In therapeutic doses these produce slowing of the rhythm, increased relaxation and less complete contraction of the