necessary, and these should be done as soon as the signs point to a further collection of pus. For routine treatment this method is necessarily somewhat difficult besides being rather uncertain, but in bilateral empyemata it is of great value. The method of opening and completely emptying the abscess cavity is not only quicker and more certain but also safer, provided that in a case presenting grave constitutional symptoms a preliminary relief of tension by aspiration renders the condition suitable for an operative procedure. After emptying the cavity of pus the question of completely closing the chest wall again must be decided on the merits of each individual case. If, however, signs supervene of further suppuration and rise of tension aspiration is performed or the incision re-opened.

The generally accepted method, therefore, of opening an empyema by resection of a portion of a rib and maintaining the opening by the insertion of a tube is probably the simplest, provided that in all cases the opening is made at the lowest limit of the abscess cavity. The tube, however, here as elsewhere, provides a very sure path for the entrance of fresh organisms. Consequently if this method is employed every precaution must be taken to prevent super-infection. This purpose is best attained first by using a long tube, whose free end is carried into a vessel containing some antiseptic; secondly, by fixing a Carrel tube around the tube entering the chest wall (Dean). With this arrangement the long tube may be removed after 48 hours, intermittent irrigation of the wound being continued till healing has commenced.

In the early stages of suppuration in the chest, by arrest of the movements of the diaphragm and chest wall and diminished expansion of the lung, a condition of immobility is brought about. Unless the tension is relieved a paralysis ensues, and if the abscess cavity is then opened and emptied the lung fails to re-expand. It gradually becomes anchored by the formation of dense adhesions which at a later stage, in spite perhaps of recovery of the lung itself, will prevent its expansion. What, therefore, as in the abdomen is primarily a physiological action becomes a pathological danger through delay. As soon as tension has been relieved in the chest by liberation of the pus the lung requires some assistance to resume its normal function, which can be best rendered by routine breathing exercises under open-air conditions.

These remarks may invite criticism on the score of lack of originality, and may appear very elementary; my object has not, however, been to put forth any new ideas, but rather to recall to mind and strongly emphasise a few fundamental principles that must be applied to the early treatment of all such conditions of suppuration as may constitute emergencies of daily practice. Whatever the type and situation of a suppurative process, and whatever additional means of treatment may be indicated, a successful issue must depend directly and primarily upon the application of these principles.

FOREIGN DECORATIONS FOR WAR SERVICES.—The King has granted permission to the undermentioned to wear the following decorations, which have been conferred in recognition of valuable services rendered during the war: Legion of Honour—Cross of the Chevalier: Sir W. Arbuthnot Lane, Colonel Sir A. W. Mayo-Robson, Sir StClair Thomson, Sir Sydney Beauchamp, Dr. E. W. Ginner, Dr. H. J. Moore Playfair, Dr. G. C. L. Vintras, and Mr. D. d'Auvergne Wright. Crown of Italy—Cross of Commander: Sir Ernest Hodder-Williams.

NEWCASTLE-ON-TYNE AND NORTHERN COUNTIES MEDICAL SOCIETY.—The first meeting of the session was held in the Royal Victoria Infirmary on Oct. 13th, Mr. S. S. Whillis, the President, being in the chair. The following cases were shown and discussed:—Dr. T. Beattie: Polycythemia, Myelogenous Leukemia, Pernicious Anæmia. Dr. Horsley Drummond: Myelitis, Aortic Aneurysm, Disseminated Sclerosis. Dr. D. W. Patterson: Infective Endocarditis, Erythromelalgia, Banti's Disease. Mr. J. W. Leech and Mr. H. M. Johnston: Cases Illustrating Sarcoma.

ARREST OF AURICULAR FIBRILLATION BY THE USE OF QUINIDINE.

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Introduction.

IN the Oliver-Sharpey Lectures for 1921 Sir Thomas Lewis, describing his researches which have done so much to elucidate the mechanism of the production of auricular fibrillation and flutter, pointed out the relatively simple task involved in the treatment of these conditions and foreshadowed its solution. He showed that they depended on circus movement, and that to arrest the process all that was required was to bridge the gap between the head and tail of the circulating wave, a gap which was in point of time only 1/50th of a second and in point of distance only 10 mm. of muscle. While Lewis¹ had been pursuing these researches which led so logically to a method of attack, Frey, working in Kiel, had been investigating empirically the problem of the treatment of auricular fibrillation. In 1914 Wenckebach² reported the arrest of auricular fibrillation in a single patient by the use of quinine, g. i. per diem. The effect was only temporary. Stimulated by this observation Frey investigated the effect of quinidine, another alkaloid of cinchona, and in 1918³ reported his results in the treatment of 22 cases. In 50 per cent. of these satisfactory results were obtained, and a normal cardiac rhythm restored. In a later communication Frey⁴ reported his results in 50 cases; in 42 per cent. fibrillation was arrested. He recommended the use of 0.4 g. quinidine by mouth, three times a day, for from six to eight days, beginning with half this dose and increasing gradually to 1.2 g. daily. If cardiac failure is present, compensation should first be restored by the use of digitalis. Then, after suspension of digitalis for one week, quinidine may be given. He states that patients in whom the fibrillation is of recent origin respond best, that a much dilated heart cannot—or can only temporarily—be brought back to its normal rhythm, and that those not responding to digitalis are refractory to quinidine. In several cases the fibrillation was changed by the use of quinidine into auricular flutter.

In two patients under treatment with quinidine Frey noted what he calls temporary cerebral paralysis with sudden cessation of respiration; both cases recovered in the course of a few hours. He considers the effect to be due to a toxic action of the drug in a patient with a drug idiosyncrasy, and advises a preliminary test with small doses in all cases to determine the occurrence of such idiosyncrasies. Schott⁵ has shown in experiments on animals that intoxication with quinidine leads to sudden cessation of the respiration. Bergmann⁶ has reported the results of treatment in 9 cases of auricular fibrillation; in 6 a normal rhythm was secured, in 2 the fibrillation changed to flutter. Klowitz⁷ treated 15 patients, and in only one did he succeed in obtaining an arrest of fibrillation. Benjamin and v. Kapff⁸ have treated 27 patients. In 18 fibrillation was arrested. In 5 of the 18 successful cases there was a relapse, with return of fibrillation, which again disappeared with further treatment; in 13 of the 18 a temporary tachycardia occurred before the normal rhythm was restored. In one patient the effect appeared unfavourable, and there was increasing failure and death. These authors call attention to the possibility of embolus resulting from the restored auricular contraction, and report one instance of lung embolism and one case of sudden death which they think was probably also due to embolism. They suggest that

the two cases of sudden respiratory failure described by Frey may have had a similar causation. Faber⁹ has reported two cases with favourable results, and Levy¹⁰ two successful results out of four cases treated, of which one relapsed.

The most recent communication is that of Jenny¹¹ who was able to stop fibrillation in 17 out of 18 cases. He states that in most instances the pulse has remained regular, but as the longest period of observation was only five months, obviously no statement as to the permanency of the cure could be made. He used from 0.5 g. to 15.5 g. in different cases, and says that it may be necessary to give the drug continuously or to repeat the courses; doses of 0.5 g. quinidine were given three times a day and found to be safe up to 2 g. to 3 g. The drug should be stopped at the least sign of disturbance in sight or hearing.

The purpose of this communication is to call attention to these observations, and to make a preliminary report of our results in the treatment of seven cases of auricular fibrillation.

CASE 1.—F. M., female, No. 43343/1921, aged 58, housewife. Mitral stenosis, auricular fibrillation. Three attacks of acute rheumatism in childhood. Then quite well until eight years ago; then a sudden attack associated with dyspnoea, palpitation, and partial loss of consciousness; since then has always suffered from shortness of breath and palpitation. A similar attack seven years ago. Subsequently admitted to an infirmary with swelling of legs and abdomen, and paracentesis abdominis was performed. Sudden attack two and a half years ago, with complete loss of consciousness, following which she came to the London Hospital as an out-patient. Since then auricular fibrillation has been constantly present, and courses of digitalis have been prescribed. Patient was admitted to the London Hospital August 2nd, 1921.

Condition on Admission.—Poorly nourished, rather wasted woman; no cyanosis, oedema, or dyspnoea; pulse absolutely irregular in volume and frequency, rate very variable at wrist, 62–112. Blood pressure: systolic 160, diastolic 96. Heart moderately enlarged to left; apex beat diffuse and wavy; systolic murmur at apex conducted outwards; pulmonary second sound accentuated. A polygraph tracing taken on August 4th, 1921, showed complete irregularity of both radial and jugular pulses, and absence of *a* waves.

Treatment and Progress.—On August 6th, quinidine gr. v. t.d.s. started. On the 11th, pulses perfectly regular, rate 50–60; no pulse deficit; a loud rumbling diastolic murmur not previously heard was present at the apex. Little change in the patient's objective condition, but she stated that the fluttering sensation in her chest constantly present for the last two and a half years had completely disappeared. On the 12th, quinidine discontinued; on the 13th, sudden onset of pain in left loin followed by four stools and hæmaturia. A little later pain in right upper abdomen; on examination tender palpable left kidney; diminished breath sounds and crepitations at right base; pulse still regular. On the 17th, condition improved; pain had gone, hæmaturia had ceased; occasional irregularity of pulse suggesting extra systoles. On the 18th, pulse completely irregular in volume and frequency; polygraph tracing showed complete irregularity of radial and jugular pulses with absence of *a* waves (see Fig. 1). On the 19th quinidine, gr. v. t.d.s., was started at 7.45 A.M.; at 9 P.M. patient suddenly complained of numbness and inability to move left leg. On examination, leg was cold; pulsation absent in dorsalis pedis but present in femoral artery; tendon reflexes were normally active; no disturbance of sensation; pulse completely irregular; diastolic murmur at apex had disappeared. On the 20th, leg warmer; good pulse in dorsalis pedis. On the 21st, tachycardia 130–140; pulse more regular. On the 22nd, tachycardia still present; polygraph tracings showed irregularity of radial and jugular pulses with absence of *a* waves. On the 23rd, pulse absolutely regular; tracings showed regular radial and jugular rhythms, and presence of *a* waves superimposed on *v* waves in the jugular pulse tracing. On the 24th, pulse-rate very variable, 76–112; polygraph tracings again taken; normal rhythm with presence of *a* waves. On the 29th, intermittent tachycardia continued. Tracings showed regular radial and jugular rhythms, *a* waves present and superimposed on *v* waves (see Fig. 2). Considerable improvement in patient's general condition. Complete absence of palpitation. A loud crescendo presystolic murmur was now heard at the mitral area. The pulse continued regular and on the 31st quinidine was reduced to gr. v. one dose a day. On the same night between 10 P.M. and 12 midnight the pulse became absolutely irregular. At 2 A.M. on Sept. 1st the pulse was regular and remained so until 10 A.M. on the 2nd, when it again became absolutely irregular. At the same

time the presystolic murmur disappeared from the mitral area.

Summary.—A case of auricular fibrillation of about two years' duration. Reacted well to digitalis when under out-patient treatment and admitted fully compensated. Quinidine by the mouth was started and fibrillation ceased with return to normal rhythm on the fifth day after a total amount of gr. xcv. had been administered. The quinidine was discontinued and embolic infarction of left kidney and right lung occurred while the pulse remained regular. Six days after the quinidine was stopped fibrillation set in again; quinidine was at once re-administered and embolism of the left popliteal artery occurred before the heart returned to its normal rhythm. After four days of quinidine administration and when a total amount of gr. lx. had been given auricular fibrillation gave place for a second time to normal rhythm. Quinidine was then reduced and fibrillation recurred, but reverted to a normal rhythm in two hours, fibrillation again recurring eight hours later. Return to normal rhythm after fibrillation was proved by polygraph tracings and the appearance of a presystolic murmur.

CASE 2.—C. G., female, No. 43343/21, aged 32, housewife. Mitral stenosis, auricular fibrillation, myocardial failure. The patient was quite well until 21 months ago, when she had a "stroke" in the night, and on the following morning was found unconscious in bed with paralysis of the right side and later aphasia. She was admitted to Tottenham Hospital; ever since the attack patient had complained of shortness of breath, cough, and palpitation. Two weeks ago shortness of breath, cough, and palpitation became worse, with swelling of abdomen and legs. No history of rheumatic fever, chorea, or rheumatism.

On Admission.—August 4th, 1921, extreme dyspnoea, orthopnoea, and cyanosis; pulsation of congested veins of neck. Almost pulseless; pulse-rate almost uncountable at apex beat, but absolutely irregular in force and rhythm; apex beat $6\frac{1}{4}$ " from mid line; systolic murmur at apex beat conducted outwards. Accentuated pulmonary second sound; liver and spleen palpable; ascites and oedema of legs; old right hemiplegia. Immediate treatment: oxygen, intravenous strophanthin, venesection, and digitalis. Rapid improvement.

Treatment and Progress.—On the 8th, pulse 80; absolute irregularity in volume and frequency. Apex beat 4 inches from mid-line; no ascites; no oedema. On the same day, fully compensated when at rest in bed. Polygraph tracing shows complete irregularity of radial and jugular pulses with absence of *a* waves (see Fig. 3). On the 19th quinidine, gr. vii. t.d.s., started. On the 20th, after four doses of quinidine (i.e., gr. xxx.), pulse perfectly regular, rate 70–80; loud crescendo presystolic murmur now heard at apex beat. On the 22nd, pulse regular, polygraph tracing showed absolute regularity of radial and jugular pulses and the presence of *a* waves on the jugular pulse-tracings (see Fig. 4). On the 28th, pulse slow and regular; loud crescendo presystolic murmur at apex beat. Occasional extra-systoles not transmitted to radial pulse; polygraph tracing shows normal rhythm with no extra-systoles and presence of *a* waves; considerable improvement in general condition; complete relief from palpitation. Quinidine reduced to gr. vii. once a day. On Sept. 5th, pulse still regular; polygraph tracing showed presence of *a* waves.

Summary.—Admitted with failing compensation; reacted well to digitalis. Return to normal rhythm 36 hours after commencement of quinidine administration, and after a total dose of gr. xxx. had been given. Quinidine reduced to gr. vii.ss. once a day. Rhythm remained normal. Return to normal rhythm proved by polygraph tracings and the appearance of a presystolic murmur.

CASE 3.—R. N., male, No. 33074/21, aged 36, carman. Aortic and mitral incompetence, auricular fibrillation, myocardial failure. Acute rheumatism seven years ago; second attack six years ago; then well until two years ago, when he was admitted to London Hospital with complete irregularity of pulse, mitral systolic and aortic systolic and diastolic murmurs, enlargement of liver, ascites and oedema of legs. Since then always suffered from shortness of breath, palpitation, and swelling of legs.

On Admission.—August 18th, 1921, cyanosis, dyspnoea, and palpitation; no ascites or oedema of legs; apex beat 1 inch outside nipple-line; systolic murmur at mitral area; systolic and diastolic murmurs at aortic area. Pulse irregular in volume and frequency. Polygraph tracing showed complete absence of *a* waves and absolute irregularity of radial and jugular pulses (see Fig. 5). On the 20th, patient much better; pulse still irregular; compensated at rest in bed. Quinidine gr. vii.ss. started t.d.s. On the 24th, pulse still irregular; quinidine gr. x. t.d.s. At midnight 25th–26th, pulse regular; bradycardia; tracings taken showed normal rhythm of radial and jugular pulses with *a* waves present (see Fig. 6). Considerable improvement in patient's general condition. No palpitation. Murmurs unchanged. Quinidine reduced to once

FIG. 1 (Case 1).

FIG. 3 (Case 2).

FIG. 5 (Case 3).

FIG. 7 (Case 4).

FIG. 9 (Case 5).

FIG. 2 (Case 1).

FIG. 4 (Case 2).

FIG. 6 (Case 3).

FIG. 8 (Case 4).

FIG. 10 (Case 5).

a day. On the 27th, sudden onset of pain in left side; no physical signs in lungs; spleen not palpable, but tenderness on palpation under left costal margin; probably splenic infarction. On the 29th patient felt very well; pain completely gone; no palpitation. Polygraph tracings showed complete normal rhythm.

Summary.—Auricular fibrillation of two years' duration, admitted with failing compensation; reacted well to digitalis; return to normal rhythm five days after commencement of quinidine administration and after a total amount of 157 gr. Embolic infarction of spleen 24 hours after return to normal rhythm; quinidine reduced to gr. x. once a day; remained regular.

CASE 4.—F. C., No. 4335/21, aged 45, housewife. Auricular fibrillation, mitral incompetence, myocardial failure. Had chorea 35 years ago; three attacks of acute rheumatism 24–19 years ago, since when she has always suffered from shortness of breath, palpitation, and præcordial pain. Admitted to the London Hospital 15 months ago, eight and a half months pregnant, with auricular fibrillation, severe dyspnoea, ascites, and oedema of legs. Reacted well to digitalis and passed successfully through a normal labour, giving birth to a live child; since then has been attending as an out-patient; auricular fibrillation constantly present.

On Admission.—August 2nd, 1921, poorly nourished woman; congested facies, dyspnoea, orthopnoea, and cyanosis. Pulse absolutely irregular in volume and frequency; heart moderately enlarged to left; mitral systolic murmur and accentuated pulmonary second sound. Congestion and oedema of lungs; palpable liver; ascites and oedema of legs.

Treatment and Progress.—Immediate treatment, digitalis, to which the patient responded well; this was stopped after nine days on account of nausea. On the 18th the patient was much better; cedema and ascites were absent; pulse-rate was much less and pulse deficit reduced. Polygraph tracings revealed absolute irregularity of radial and jugular pulses with absence of *a* waves (see Fig. 7). On the 19th quinidine, gr. v. t.d.s., was started; on the 24th digitalis was re-started on account of tachycardia; on the 31st quinidine was increased to gr. x. t.d.s. Frequent polygraph tracings revealed the persistence of fibrillation and the absence of normal rhythm, and this condition continued up to Sept. 2nd, when, however, the patient's general condition was much improved, and a completely irregular rhythm seemed to have given place to one in which phases of comparative regularity alternated with phases of tachycardia, but polygraph tracings failed to reveal presence of *a* waves. On the 3rd digitalis was stopped. On the 5th at 6 A.M. the pulse was absolutely regular and continued so all day. Polygraph tracings showed a completely regular cardiac rhythm with presence of *a* waves (see Fig. 8).

Summary.—A case of auricular fibrillation of about 15 months' standing which reacted well to digitalis. Normal rhythm was restored after quinidine had been administered for 18 days and a total quantity of 315 gr. had been given.

CASE 5.—S. E., No. 43563/21, aged 35, housewife. Myocardial failure, auricular fibrillation, mitral stenosis, and incompetence. Shortness of breath on exertion dating from an attack of influenza three years ago. A year and a half ago an attack of bronchitis. On first getting up after three weeks in bed, sudden loss of consciousness, followed by exhaustion, breathlessness, palpitation, and pain in the chest, which have persisted ever since. Hæmoptysis, followed by oedema of legs, nine weeks ago. No history of chorea, rheumatism, or rheumatic fever.

On Admission.—August 20th, 1921, poorly nourished, sallow woman; heart enlarged outwards and downwards; dyspnoea; systolic and diastolic murmurs at mitral area; absolute irregularity of pulse; tachycardia; a few crepitations at both bases; palpable liver; no ascites or oedema of legs.

Treatment and Progress.—Tinct. digitalis $\mathbb{M}$ xxx., four doses. After three days much better; no dyspnoea while at rest in bed. On the 22nd polygraph tracings revealed complete irregularity of radial and jugular pulses with absence of *a* waves. Quinidine, gr. vii.ss. t.d.s., was then started; but on the 27th it had to be suspended on account of diarrhoea and abdominal pain. On the 28th there was no diarrhoea or pain; quinidine was re-started. On Sept. 1st patient developed a scarlatiniform rash. On the 5th pulse was still irregular. Repeated polygraph tracings showed the continuation of auricular fibrillation up to this date.

Summary.—A case of auricular fibrillation of unknown duration which failed to be arrested by 300 gr. quinidine administered over a period of 15 days. Ill-effects observed attributable to the drug were abdominal pain, diarrhoea, and a scarlatiniform eruption.

CASE 6.—J. L., No. 33010/21, aged 37, carman. Auricular fibrillation, myocardial failure. Six months ago short of breath; five months ago his right foot suddenly went numb, cold and white. Admitted to the London Hospital with auricular fibrillation, palpable liver, mitral systolic murmur

and cedema of lungs; reacted well to digitalis and was discharged. Return of exhaustion and shortness of breath one month ago.

On Admission.—August 2nd, 1921. Well-nourished man; dyspnoea, orthopnoea, and cyanosis. Pulse absolutely irregular in volume and frequency; heart enlarged to left; mitral systolic murmur and accentuated pulmonary second sound; crepitations at both bases. Palpable liver; no ascites or oedema.

Treatment and Progress.—Reacted well to digitalis, which was stopped on August 18th. On August 23rd, 1921, polygraph tracings were taken which revealed absolute irregularity of radial and jugular pulses with absence of *a* waves (see Fig. 9). Quinidine, gr. vii.ss. t.d.s., was then started. On the 27th, quinidine was increased to gr. vii.ss. six-hourly. On Sept. 1st quinidine was increased to gr. x. six-hourly. At 4 A.M. on the 5th one dose of tinct. digitalis, $\mathbb{M}$ xxx., was given. The pulse remained completely irregular until 6 A.M. on the same day, when irregularity gave place to a normal rhythm. Polygraph tracings (see Fig. 10) demonstrated complete regularity of radial and jugular pulses with presence of *a* waves.

Summary.—A case of auricular fibrillation of about 15 months' duration which reacted well to digitalis. Normal rhythm was restored after quinidine had been administered for 18 days, when a total quantity of gr. 375 had been given. Ill-effects observed attributable to the drug were abdominal pain, diarrhoea, and profuse sweating.

CASE 7.—R. P., No. 3564/21, aged 42, housewife. Auricular fibrillation, mitral incompetence. Rheumatic fever ten years ago. Since then quite well until 18 months ago, when she started to have shortness of breath, pain in the chest, and palpitation. One year ago admitted to the London Hospital with auricular fibrillation, dyspnoea, and oedema. Did not react well to digitalis, but improved with prolonged rest in bed. Since then shortness of breath on exertion, palpitation, and præcordial pain.

On Admission.—August 20th, 1921, very obese Jewess. Short of breath; absolute irregularity of pulse; heart enlarged to left; mitral systolic murmur and accentuated pulmonary second sound. No cedema. Fairly well compensated.

Treatment and Progress.—On the 22nd tracings showed absolute irregularity of radial and jugular pulses with absence of *a* waves. The same day quinidine, gr. vii.ss. t.d.s., was started. On the 24th tinct. digitalis, $\mathbb{M}$ xxx., four-hourly, was started and continued for six doses. On Sept. 1st patient developed a scarlatiniform rash. On the 2nd, quinidine was still being administered. Polygraph tracings revealed the persistence of auricular fibrillation, and there was no alteration in the nature of the cardiac rhythm.

Summary.—A case of auricular fibrillation of about one year's standing which reacted poorly to digitalis. Quinidine (262 gr. administered over a period of 12 days) failed to produce a return to normal rhythm. Ill-effects observed, attributable to drug, were abdominal pain, pain in the back, sweating, and scarlatiniform rash.

Discussion of Results.

From the foregoing case reports it will be seen that out of 7 cases of well-established auricular fibrillation in 5 the administration of quinidine by the mouth has been associated with return to the normal cardiac rhythm. In all these five cases the change has been demonstrated by polygraph tracings, and in two of them by the appearance of a crescendo presystolic murmur at the apex. In one of these five relapse occurred soon after the drug was stopped, and again a second time when the dosage was reduced. In two the dosage was only decreased and there was no relapse into auricular fibrillation. In two of the five cases, shortly after the onset of normal cardiac rhythm, embolic infarction of internal organs occurred. The formation of clots in the appendix of a fibrillating auricle is not uncommon, and the loosening of these by restored auricular contraction and their projection into the general circulation is easily understood. The only reference we have been able to find in the literature to a similar occurrence is in a paper by Benjamin and v. Kapff, who report one instance of lung embolism and one of sudden death possibly due to the same cause. In one, tachycardia was associated with the administration of the drug. In one case an alteration of cardiac rhythm, characterised by an alternation of phases of comparative regularity with bouts of irregular tachycardia, preceded return to normal rhythm.

The pharmacological action of quinidine is still undetermined. Electro-cardiographic study has shown

that, under quinidine, alteration in both shape and size of the auricular and ventricular waves occur and are accompanied by a prolongation of the P.R. interval.⁵ Hoffman¹² has shown that the excitability of the auricle of the excited heart to electrical stimulus is diminished by quinidine, and that stimuli which would normally produce fibrillation of the auricle fail to do so after quinidine has been added to the perfusate. These results indicate an effect on the excitability and conductivity of heart muscle. Lewis has pointed out that the existence of the circulating wave depends on three factors—the length of the path, the refractory period of the heart muscle, and the conductivity. It seems reasonable to suppose that quinidine abolishes fibrillation either by increasing the conductivity or by prolonging the refractory period of heart muscle, with the result that the gap is bridged and circus movement ceases.

The restoration of a normal rhythm in five out of seven cases of auricular fibrillation by the use of quinidine is of considerable physiological interest. Its practical value in therapeutics remains to be determined. We would point out from the first that the use of quinidine is not without danger, the risk of embolism being a real one. In our cases the improvement in the general condition was not striking, and much less marked than might have been anticipated, and perhaps little more than might be attributed to the prolonged rest in bed. Certainly no dramatic change in the patient's general condition occurred when normal cardiac rhythm was restored. If the drug is stopped, tendency to relapse seems to be the rule. That quinidine will take a place in the treatment of cardiac disease seems probable, but the nature of its action and the limitations of its therapeutic value remain to be determined.

In conclusion we would express our thanks to Miss Rutherford and Miss Harvey, sisters of the Medical Unit Wards, for the extra trouble they have taken in the supervision of the nursing of these patients.

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ACIDOSIS AND TOXIC SYMPTOMS OF
SEVERE DIARRHŒA IN INFANCY.

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THE toxic symptoms of severe diarrhœa in infancy, especially when associated with an increased pulmonary ventilation without cyanosis, have been shown by Howland and Marriott¹ and by Schloss and Stetson² to be accompanied by a state of acidosis.* This so-called intoxication was found to be coincident with (1) a decreased tension of the carbon dioxide of the alveolar air; (2) an altered hæmoglobin dissociation curve; (3) an increased tolerance to sodium bicarbonate; and (4) a decreased reserve of available base in the blood plasma.

The administration of alkali has been suggested and used in treatment. In Great Britain but little attention has been paid to this group of cases. Accordingly, at the suggestion of Dr. Leonard Findlay, the present investigation into the alkali reserve of the plasma in cases of diarrhœa in infancy was undertaken.

* The term "acidosis" is used in the sense suggested by F. G. Hopkins at the recent meeting of the Section of Physiology and Pharmacology of the British Medical Association, as a diminution in the potential neutralising power of the blood.

Methods.

The estimations were made according to the "micro-method" of Van Slyke.³† The blood was taken by the investigator from the superior longitudinal sinus directly into a glass syringe containing a few grains of potassium oxalate (approximately 0.5 per cent. of the weight of the blood drawn). By the use of the longitudinal sinus changes in the blood due to stasis, crying, and struggling were obviated. Admission of air to the syringe was avoided. Sufficient admixture of blood and oxalate was obtained during transference from syringe to centrifuge tube, and this did not expose the blood to loss of carbon dioxide or absorption of oxygen to any greater extent than occurs with the use of the M'Rae needle as described by Van Slyke.³ Analyses were made at once. Duplicate determinations were carried out whenever there was sufficient plasma.

The alkali reserve is expressed as the volume per cent. of carbon dioxide which the plasma binds as bicarbonate at body temperature and the normal carbon dioxide tension of the alveolar air. Corrections are made for the temperature and barometric pressure at the time of analysis and the whole reduced to standard conditions of 0° C. and 760 mm. Hg pressure.

In the present paper the term "alkali reserve" will be used in relation to the numbers expressing the cubic centimetres of carbon dioxide bound as bicarbonate in 100 c.cm. of plasma. This seems permissible as an abbreviation, for the percentage of carbon dioxide is used as a measure of the available base. Since corrections are made for the free carbonic acid in the plasma, it seems also less confusing and more exact than the use of the phrase so frequently employed, "percentage of carbon dioxide." The sugar estimations were made according to the Lewis-Benedict⁴ colorimetric technique. Routine urine analyses as made by the resident physician showed neither increased glycaemia nor ketosuria.

1. Normal Infants.

The blood was taken, as described, from healthy breast-fed infants gaining in weight, brought to the dispensary for circumcision. As a delay of one-half to one hour between the withdrawal of the blood and the separation of the plasma and corpuscles was unavoidable, the alkali reserve is probably slightly lower than it would have been had the determination been carried out at once.‡ The number of hours after the last feed is obviously approximate. The value ranged from 43.3 to 63.3 (Table I.). This wide

TABLE I.—Alkali Reserve of Plasma (Van Slyke) in Normal Infants.

No.	Age.	Carbon dioxide vol. %.	Hours after feeding.	No.	Age.	Carbon dioxide vol. %.	Hours after feeding.
1	6 wk.	51.6	3	16	4 mth.	51.6	+
2	1 mth.	56.5	3	17	11 wk.	52.4	1½
3	2 mth.	53.6	3	18	9 "	55.0	2½
4	5 "	50.0	3	19	3 mth.	54.6	+
5	5 wk.	56.5	3	20	9 wk.	56.5	2½
6	2 mth.	49.7	2½	21	11 "	56.2	3
7	4 "	49.7	2	22	1 mth.	63.3	2½
8	6 "	49.0	?	23	4 "	62.2	2
9	6 "	43.3	8	24	7 wk.	54.8	+
10	6 wk.	54.2	1	25	5 "	56.4	+
11	6 mth.	53.8	1	26	1 yr.	46.8	5
12	3 "	54.0	2	27	1 "	55.5	1½
13	5 "	50.0	2	28	1 "	58.6	2
13	9 wk.	52.6	12	29	8 mth.	60.4	2½
14	3 mth.	50.7	3	30	8 wk.	53.0	2½
15	7 wk.	51.6	3				

+ = At once.

† It is realised that a certain error occurs in the separation of plasma and corpuscles, and the subsequent subjection of the plasma to alveolar carbon dioxide tension, but the smallness of the error, and the greater facility of this technique for clinical purposes, determined its adoption.

‡ Christiansen, Douglas, and Haldane⁵ state that when serum and corpuscles remain in contact even for an hour there is a lowered alkali reserve of the plasma, due to ionic interchanges between cells and plasma.