the dorsum of the tongue until it comes in contact with the pharyngeal wall. Then, by a simple leverage action of the wrist, the whole tongue with the epiglottis and hyoid bone may be made to move upwards and away from the pharyngeal wall as far as the thyro-hyoid membrane will allow such a movement; and in a moment all obstruction to the entrance of air in and out of the lungs is thus removed. As long as the spoon handle thus supports the tongue, hyoid bone, and epiglottis, none of these structures can possibly interfere with the entrance of air into the lungs during the movements of artificial respiration (see Fig. 2).

Fig. 2.—A photograph of the same body, showing the appearance of the neck below the chin in the middle line (a) when the spoon handle is held in proper position during artificial respiration. In this case examination showed that the whole tongue was well removed from the pharyngeal wall, and that the epiglottis was raised, and the aryteno-epiglottic folds were quite tense.

In all cases of deep insensibility, as in *anæsthesia*, and in the apparently drowned, this method of removing all possible obstructions to the entrance of air into the lungs during artificial respiration is at once both safe and reliable, and in combination with Howard's method of pumping air in and out of the chest gives the most satisfactory results. The sword swallower has no difficulty in passing the sword down his gullet when his head is thrown well back; and there is even less difficulty in lifting up the tongue, epiglottis, and hyoid bone in the way I have described.

MENTAL PECULIARITIES IN HÆMOPHILIA.

By CLINTON T. DENT, F.R.C.S.,
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IN the interesting case of hæmophilia described in the BRITISH MEDICAL JOURNAL of April 2nd (p. 883), passing allusion is made to a point which, though, according to my experience, very common in these patients, has hitherto been little noticed. In the hope of eliciting information on the point, corroborative or other, I venture to trespass on your columns.

Patients subject to hæmophilia—bleeders in short, for that term is surely better than "*hæmophilics*"—constantly exhibit mental peculiarities of definite form. The most important and the most common mental peculiarity is an inability (it is more than an unwillingness) to tell the truth about their condition even when they have had repeated and alarming experience of their defect. Frequently they will persist in obstinate denial of their liability to bleed, even when the hæmorrhage is going on, and resisting all efforts to check it. I well recall a strongly-marked instance of this unfortunate and misleading propensity that occurred some years ago in hospital practice. A youth, about 16 years of age, was admitted for persistent hæmorrhage following the extraction of a carious lower molar tooth. Every kind of remedy was tried, but the bleeding, though often checked for the time, broke out again and again. Eventually the inferior dental canal was plugged, but after a while the hæmorrhage recommenced and the boy died. From first to last he denied stoutly that he had ever suffered from bleeding, though the condition of hæmophilia was obvious enough to all. Though sane in all other respects, and aware of his serious condition, he maintained this attitude throughout. We were anxious

to communicate with his friends, but he flatly refused to give the address of his relations. Ultimately and by chance the address of his mother was discovered, and she was sent for. There was no need to break the news gently to the woman, for the moment she arrived she said, "I know what has happened; my son has had his tooth taken out, and he is bleeding to death. The same thing almost happened two years ago, and on many occasions his condition has been most serious from slight scratches or wounds." The youth furiously resented his mother's appearance at his bedside, and died actually cursing her, and all who had to do with him, while asserting almost with his last breath the *idée fixe* that he did not bleed more than others.

This distressing case, though exceptional in its circumstances, is no isolated experience, and I doubt not many others have met with patients subject to similar delusions. Hæmarthrosis may easily simulate tuberculous disease in appearance, and most surgeons would probably admit—if only to themselves—that once in a way they have at first been deceived by such cases.

In these days of early and free operative measures the precaution of ascertaining whether there is any history of hæmophilia should, in the case of joint disorders, be a matter of routine. It is not, as I have shown and always taught, sufficient to rely on the patient's word alone, however categorical and clear his statements may seem to be.

The suggestion that bleeders should be tattooed does not seem to be a happy one. Apart from the obvious practical difficulty of gaining consent, the tattoo punctures would be likely to give rise to trouble. If bleeders and epileptic patients could be induced to wear round their necks a small label setting forth their infirmity, they would probably derive advantage. But epileptic patients are far more likely to adopt any such device than bleeders.

THE ALKALINITY OF THE BLOOD IN GOUT.

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It is well known that an attack of gout may be accelerated by the ingestion of certain foods or beverages harmful either as regards their quality or quantity. One view, which has been commonly held for some time, is that such substances when they accelerate an attack of gout reduce the alkalinity of the blood, and so hasten the precipitation of sodium biurate from that medium, or, as is so frequently stated, they cause "an acidity of the system." It is assumed by those who hold this view that a nitrogenous animal diet diminishes the alkalinity of the blood and so causes deposition of sodium biurate, and that a similar result is caused by the acids contained in wines and beers; whilst, on the other hand, it is assumed that a subsequent rise in alkalinity causes solution of the previously-formed deposits. In the Goulstonian Lectures of last year I showed by a series of experiments that a diminution of the alkalinity of blood serum neither diminished the solubility of sodium biurate in that serum, nor did it produce any effect on the rate of decomposition of sodium quadriurate by such serum, and that, therefore, it was extremely improbable that a diminution of the alkalinity of the blood could in any way affect the gouty process.

Although so many writers have inclined to the view that a diminished alkalinity of the blood is associated with gout, it is remarkable that such a view has no basis of positive experimental evidence whatever. As a matter of fact the proof is in the opposite direction. The experiments of Klemperer¹ show (1) that the alkalinity of the blood of gout is but very little, if at all, diminished; (2) that corresponding variations in the alkalinity may frequently be met with in healthy individuals; and (3) that a diminution of the alkalinity of the blood is a common pathological condition in diseases that are in no way associated with uratic precipitation, such as acute rheumatism, leukæmia, diabetes, carcinoma, and pyrexia. I therefore thought it advisable to make systematic determinations of the alkalinity of the blood throughout the duration of an undoubted attack of gout, and to compare such alkalinity with the average alkalinity of specimens of blood drawn from a number of healthy individuals.

The process that I employed for these determinations of the

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.,

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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.