half on lint to a youth whilst under lateral lithotomy, performed by Mr. Jubb of this place. Complete somnolence continued until after he had been put to bed, and was not followed by any untoward after-symptom. Chloroform is more pleasant in such quantity to take than ether, though in that respect surpassed, perhaps, by the nitrous oxide gas. As observed by Mr. Couper, however, the time relatively consumed in the administration, and the disagreeable perfume of the vapour of ether, are unimportant, if regarded as safer, and to be free from nausea and other sequelæ.

With the bichloride of methylene, except as spray, we have had no experience here, and it would seem to possess no immunity from danger in its use.

ALCOHOL AS A CAUSE OF BRIGHT'S DISEASE; AND DR. DICKINSON'S STATISTICS.

By W. ROBERTS, M.D., F.R.C.P.,

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In a previous paper, I pointed out that Dr. Dickinson's attempt to prove from the Registrar-General's Reports that renal disease was not causally connected with intemperance, broke down on a closer investigation—that, in fact, by the same rule it might be proved that cirrhosis of the liver was unconnected with intemperance, which is a manifest fallacy. In his recent rejoinder to my criticism, he rests his argument mainly on the *post mortem* statistics of St. George's Hospital for a period of thirty years, which he has collected and compared with admirable industry. First, he compares the state of the kidneys in fifty-two persons who died of delirium tremens with that in fifty-two persons who died from accident and who were presumably of sober habits. Secondly, he compares the kidneys and urine in a hundred and forty-nine persons who followed alcoholic trades with those of a hundred and forty-nine persons who followed other occupations. He finds in both series of cases that the total of renal disease was about as great in the presumably sober as in the presumably intemperate.

This conclusion is so opposed to the general opinion, and the statistics contain on the face of them some things so improbable, that I am sure Dr. Dickinson will not think me captious if I subject them to a somewhat searching criticism. I believe I shall be able to show that they only cover a part of the ground occupied by alcohol as a producer of disease; and that, in so far as they relate to the kidneys, they are misleading.

Before going further, it is desirable to define the exact scope of the controversy. It is not contended that alcohol has any important share in the production of the acute forms of Bright's disease, or of those chronic forms which begin acutely either as a result of scarlet fever or other acute disease, or of a sudden impression of cold, and which are associated with the large, smooth, white, or mottled kidney; but it is contended that alcohol is one of the most important factors in the production of those types of Bright's disease which are chronic from the beginning, and which are associated with the granular contracting or cirrhotic kidney.*

The first thing to be remarked about these statistics is, that they contain no mention of gouty lesions; yet it cannot be denied that one of the commonest morbid effects of alcohol is gout, and that one of the not uncommon results of gout is a certain form of Bright's disease. Dr. Dickinson has felt this difficulty, and endeavours to turn it by the allegation that the "gouty kidney" generally occurs in saturnine gout. But this can hardly apply to the better classes, among whom the gouty kidney is common enough. Further, Dr. Garrod doubts if lead alone, without the help of alcohol, can produce gout.

In the next place, cases of chronic Bright's disease (non-gouty) with the symptoms of granular contracting kidney, are frequently actually met with in practice, in which no definite causal antecedent can be discovered, except the abuse of alcohol. These are generally middle-aged or elderly persons, most of whom have not been intemperate in the ordinary sense of the word, but who have been in the constant habit, for many years, of taking every day alcoholic drinks to some excess. Three such cases are at the present moment under my observation, all belonging to the easy classes, and about whom it could never perhaps be said that they had been palpably the "worse for liquor." It is also remarkable how often intemperate habits are mentioned among the antecedents of the cases of chronic Bright's disease recorded by authors—especially in this country. Of fifty-one cases related by Bright, Christi-

son, George Johnson, Frerichs, and Grainger Stewart, no fewer than twenty-seven are reported as persons of intemperate habits. Dr. Dickinson thinks it a sufficient answer to this point, to say that intemperate habits are so common in this country, that this is a mere coincidence. But let him take another chronic disease, which also prevails largely in middle life and advancing years, namely, cancer, and he will find that clinical observers have not found any such coincidence. I believe that the chief reasons why these remoter effects of alcohol on the kidneys are not indicated on Dr. Dickinson's statistics are, first, that these statistics apply to comparatively young persons, who have drank hard and killed themselves early, in whom, therefore, the later and slower effects of more moderate abuse of alcohol are not developed; secondly, that the granular kidney of chronic Bright's disease has not been sufficiently distinguished, by morbid anatomists, from granulation of the kidney induced by other morbid states.

The mean age of the cases tabulated by Dr. Dickinson varied from 36.8 to 40.6 years; but the mean age of the victims of the granular kidney of Bright's disease is, according to the same authority, 50.2 years. Here is a difference of age of nearly a decade, which may fairly be assumed to have an important influence. But there is something more than the difference of age. These statistics do not really test the point at issue; they apply, on the alcoholic side, to what one may call the acute effects of alcohol to persons who, in ordinary parlance, would be called intemperate. In such persons are produced cirrhosis of the liver, alcoholic phthisis, early fatty changes, and delirium tremens. But there is another very considerable class of persons who ostensibly rank among the temperate—persons who never get drunk, who run little risk of cirrhosis or alcoholic phthisis, and none at all of delirium tremens. These persons imbibe daily a few glasses of spirits or wine, or a few pots of porter, more than is good for them. This goes on steadily for a long course of years, apparently without ill effects; but at length they become gouty, or the urine becomes albuminous, or their arteries become fatty ten years earlier than they would otherwise. Are these effects less real because they are produced more slowly? There has, I believe, been too little attention paid to the effects of these long continued small excesses of alcoholic drinking, which do not, as hard drinking often does, rob a man of three-fourths or one half his life, but which curtail his life by some ten or fifteen years.

Another source of error in these statistics is the excessive amount of renal disease credited to presumably healthy persons. Dr. Dickinson would have us believe that one in every seven presumably healthy persons, of the average age of forty-one, is affected with granular kidneys. If this is to be taken to mean that one-seventh of our presumably healthy population are the subjects of chronic Bright's disease—even in the incipient stage—the statement must involve some notable fallacy. Chronic Bright's disease is not easily robbed of its victim. If this statement were true, or anything like true, Bright's disease ought to rank enormously high in our mortuary returns. But I find that all the types of renal disease only account for about one per cent. of the total mortality. But this point may be tested in another and more satisfactory manner—namely, by the examination of the urine of a number of presumably healthy persons. If the urine be free from albumen, and of normal quantity and specific gravity, it may be assumed—with only a very slight risk of error—that the producer of that urine is free from Bright's disease. I would suggest for this purpose the examination of the urine of the patients admitted into a general hospital for fractures and other injuries, which do not compromise the general health. I have made a small beginning of an investigation of this sort, but under less stringent conditions. My clinical clerks were instructed to examine the urine of all patients in the surgical wards of the Manchester Infirmary above the age of twenty years. Vesical cases were excluded, as were also patients who were moribund, or labouring under severe general ailment. Forty-two cases answered to these conditions; their average age was forty-two: twenty of them were cases of fractures and other injuries, seven had joint-disease, seven had ulcers, and the remainder had cancerous tumours or some trifling ailment. Of these, only two had albuminous urine; one was a man of intemperate habits, suffering from strumous disease of the knee-joint; the other was a syphilitic youth, with ulcer of the leg. All the accident cases—which were really the only cases fairly comparable with Dr. Dickinson's cases of death from accident—had not a trace of albumen in the urine; so that, instead of one in seven exhibiting signs of renal disease, there was only one in twenty-one, and this in spite of the inclusion of struma and syphilis. These numbers are far too few to found a safe conclusion on, but they indicate very clearly the necessity for caution in accepting purely *post mortem* data on the condition of the kidneys. Dr. Dickinson is inclined to reproach me with testing pathological observations by clinical experience. I would in my turn venture to say that morbid anatomy, without the control of clinical observation, may sometimes lead very

* I put out of view the "lardaceous" or "waxy" cases, because of their limited number, and their more obvious connection with chronic suppuration, joint-disease, and syphilis.

widely astray. There are still a good number of conditions which stand out in large outlines to clinical experience, which are vague and dim to the morbid anatomist. Need I do more than mention acute articular rheumatism, typhus, tetanus, and epilepsy?

We may be sure that the phrase "granular kidneys," as it occurs in *post mortem* records, means something else besides the granular degeneration of Bright's disease, as practically and clinically understood. And, in fact, the kidneys do often show granular surfaces in cases which have no real claims to be classed with genuine Bright's disease. I can only speak with exactness of some of these cases. In my work on *Urinary Diseases* (second edition, p. 350), I have pointed out the confusion into which morbid anatomists have fallen in not distinguishing the granular appearance of the kidneys consequent on long sustained passive congestion of the kidneys, from the granulation due to Bright's disease. The anatomical identity of these two conditions, although generally assumed, has been denied on high authority, and clinically, they are certainly distinct.* A real combination of regurgitant heart-disease, or cirrhosis of the liver, with chronic Bright's disease, is comparatively rare; and when it exists, the renal affection never fails to vindicate its existence by its appropriate symptoms. But, as a rule, the passive renal congestion which accompanies impediments to the circulation is quite a subordinate condition, and has none of the clinical features of chronic Bright's disease. The urine is of high specific gravity and scanty, and it either contains no albumen at all, or only small quantities. The renal disorder has no progressive tendency of its own; it does not produce the special secondary effects of Bright's disease, and it oscillates in its intensity strictly with the activity of the primary disease.

There are also morbid conditions of the kidney of a temporary character, which probably leave behind them certain alterations in the cortex of the organs much resembling granulations. When we reflect how many middle-aged persons have suffered in earlier years from scarlatinal and other forms of acute renal disease—from syphilitic tubercles of the kidney, from renal gravel—how many women have suffered from renal congestion during repeated pregnancies, and yet how recovered with practically sound kidneys from all these ailments—it is scarcely a matter for wonder that the organs should, years afterwards, bear upon their surfaces irregularities, depressions, and even more or less granulation with thickening and adhesion of the capsules—not as indicating any existing disease, but, like a scar on the skin, as footprints of a bygone disorder.

LONDON DRINKING WATER.

By J. ALFRED WANKLYN, Esq., Corresponding Member of the Royal Bavarian Academy of Sciences.

THE following, which is the second of a series of monthly analyses of the London waters, is intended as a substitute for the analyses published by the Registrar-General.

COMPANIES.	Free Ammonia.*	Albuminoid Ammon.*
Kent Water Company.—Deptford Railway Station, Nov. 20..	0.01	0.02
New River.—Cab Rank, Tottenham Court Road, Nov. 21....	0.01	0.05
East London.—Cambridge Heath Gate, Dec. 3	0.01	0.18
THAMES COMPANIES.		
Chelsea Company.—Cab Rank, Horse Guards, Dec. 2.....	0.02	0.13
West Middlesex.—Cab Rank, Portland Road, Dec. 3	0.00	0.10
Southwark & Vauxhall.—Cab Rank, St. George's Road, Dec. 4	0.00	0.18
Grand Junction.—Cab Rank, Woodstock Street, Dec. 4	0.00	0.17
Lambeth.—Cab Rank, Palace Road, Dec. 2	0.00	0.16

* Parts in a Million (Milligrammes per litre).

The two first specimens of water were collected on the 20th and 21st of November, and the rest on the 2nd, 3rd, and 4th of December. The analyses have special interest, inasmuch as they exhibit the effect of continued floods on the quality of the metropolitan water-supply. As will be observed, only two of the companies have maintained the quality of their water; viz., the Kent and the New River. All the other companies have suffered in a greater or less degree. The West Middlesex has suffered only slightly. The Chelsea comes next; whilst the East London and Grand Junction Companies (which usually supply excellent water) have, during these floods, sunk to the ordinary level of the Southwark and Vauxhall and of the Lambeth Companies.

* Ferichs has pointed out that a similar confusion has arisen with regard to granulation of the liver in true cirrhosis, and granulation from congestion and other causes.

REPORTS

OF

MEDICAL AND SURGICAL PRACTICE IN THE HOSPITALS OF GREAT BRITAIN.

THE LONDON HOSPITAL.

SPECIAL KINDS OF FRACTURES.

(Under the care of Mr. RIVINGTON.)

Oblique Fracture of the Lower Ends of the Humerus.—Two cases of oblique fracture of the lower end of the humerus have come under Mr. Rivington's care. Each case occurred in an old man over seventy years of age. One of these patients had been shut up in a cupboard for two days before his admission into the hospital, and his arm was extensively ecchymosed from the shoulder to the elbow. The fracture extended obliquely across the humerus from a point above the inner condyle to another just below the external condyle, and crossed the olecranon fossa. Flexion and extension were not interfered with. The patient died from the effects of the injury and neglect after it, and the specimen is in the London Hospital Museum. In the second case, there was great breadth, especially when the joint was flexed; crepitus was obtainable only on grasping the arm with one hand and the forearm with the other, and moving the two parts of the limb from side to side. Flexion and extension were readily effected, as well as rotation of the radius, without eliciting crepitus. When the two condyles of the humerus were grasped and flexion made, no crepitus could be felt, because the lower fragment was then fixed in apposition with the shaft of the humerus. The inner condyle moved with the forearm, and the outer with the humerus. Hence it was concluded that the fracture extended obliquely across the humerus from above the inner condyle to a point below the external, as in the previous case.

Fracture of the External Tuberosity of the Tibia.—Mr. Evan Llewellyn, of Mile End, asked Mr. Rivington to see a patient of his who had met with an accident. She was a housekeeper, sixty years of age, had slipped off a chair while cleaning a window, and had fallen on her right knee. There was a good deal of swelling below the knee, due to effusion of blood. When the joint was extended nothing could be made out, and the fibula appeared to be uninjured. On flexing the knee-joint, however, and grasping the outer tuberosity, crepitus became apparent, that part of the tibia being moved with comparative ease. Ice was recommended to be applied in the extended position of the joint, and the patient did well. The joint swelled considerably after the first twenty-four hours, as had been predicted; but no unfavourable symptoms presented themselves throughout the progress of the case.

Compound Fracture of Frontal Bone: Recovery.—Charles Moss, aged 33, was admitted into the London Hospital, under the care of Mr. Rivington, on December 23rd, 1871. He had been kicked by a horse on the forehead, and there was a lacerated wound extending transversely from the right side above the right eyebrow to the middle of the left eyebrow. The frontal sinus was laid open on the left side, and a fracture extended upwards from that point through both tables of the bone. The finger introduced could feel the dura mater. Some loose fragments could be felt in the wound, and a piece of the orbital portion of the bone was detached. The loose pieces of the bone were removed, but the portion which formed an integral part of the orbit, and had the tendon of the superior oblique muscle attached to it, was retained. The edges of the wound were brought together with sutures for the greater part of its extent, and a piece of lint dipped in carbolic oil inserted at its left extremity. Ice was applied to the head. The temperature had risen next day to 104.5 deg. On the 25th, it was 103.5 deg., and did not fall again till the 31st, when it went down to 101 deg. There was some delirium at first, especially at night, and a great deal of purulent discharge. The eyelids both became infiltrated, so as to close the eyes completely, and the conjunctivæ became inflamed and oedematous. There were no rigors at any time. There was occasional headache. By the 2nd of January, 1872, he was much better, the eyelids were much less swollen, and the eyes had begun to clear. By the 4th, the wound had quite healed on the right side, and the eyes were nearly well. He continued to improve, and was soon able to sit up. By the end of January, he was able to leave the hospital cured.