8th January, 1876.

John Fletcher. Was sent for to see him on the morning of the 19th ult. Had a scotal hernia incarcerated. Had attended him for the same thing over two years ago. I reduced that, but the next day he still complained of pain and sickness. I prescribed an aperient, which his stomach did not retain.

21st. Sickness still continued. 22nd.—Sickly. 23rd.—Sickness and pain still. Administered injection, with no effect. 24th.—Vomiting increasing No swelling of abdomen, nor pain, except that caused by retching. 26th.—Admitted to Infirmary.

Progress of Medical Science.

THERAPEUTICAL NOTES.

PRURITUS.

For the intolerable pruritus common in fall and winter, many physicians use Dr. L. Duncan Bulkley's prescriptions, given in the *Transactions* of the American Medical Association. We repeat them here:—

Unguentum Anti-pruriticum.

R. Pulv. gum camphor. Chloral hydrat.

ăă 3j

Grind well together in a mortar, till they form a fluid, and add slowly, simple cerate, one ounce.

Liquor Picis Alkalinus.

R.	Potass. causticæ.	3 j	
	Picis liquidæ	3 ij	
	Aquæ	3 v.	M.

Dissolve the caustic potass in the water, and add gradually the tar, mixing them well in a mortar. Use in solution with from 8 to 14 parts of water.

PERUVIAN BARK IN SORE THROAT.

Dr. Holden recommends the following formula, as exceedingly efficacious in diptheritic scarlatina and other forms of sore throat:—

R.	Corticis peruvianæ Acaciæ pulv.	flav.,	3 ij 3 j	
	Sacch, alb.,		7 ss.	M

S. Mix one half of this powder in a tablespoonful of cream, and apply frequently with a camel's hair-brush.

SCIATICA.

Dr. Peter, of Paris, recommends bandages saturated in chloroform. They should be bound firmly over the affected part.

ON THE TREATMENT OF BRIGHT'S DISEASE OF THE KIDNEY.

By James Tyson, M.D., Prof. of Pathological Anatomy and Histology in the University of Pennsylvania.

Read before the Philadelphia County Medical Society, March 10, 1875.

Although the line of demarcation in the treatment of the different forms of Bright's disease is by no means so sharp as that which modern histology has made in their pathology, it will, nevertheless, facilitate our consideration of the subject, as well as perhaps tend to place treatment on a more rational basis, if I preface a brief recapitulation of the different forms alluded to:

1. In the first place, we have the division into acute and chronic Bright's disease. The former presents us with a single form, that of acute nephritis, the acute tubal nephritis, acute catarrhal and acute desquamative nephritis, of certain authors,— which has its most frequent occurence as a sequel of scarlet fever, and therefore in children; while it also occurs less commonly in adults after exposure, especially while perspiring, to cold and moisture. It is indicated by the presence of dropsy, bloody, highly albuminous urine, containing blood-casts and epithelial casts, also sometimes hyaline fibrinous casts.

2. Secondly, we have chronic Bright's disease, which gives us four subdivisions. The first of these is the large white kidney, also called chronic catarrhal nephritis, and chronic tubal nephritis, because it involves more particularly the cells lining the uriniferous tubules, and is generally found in continuation of the acute nephritis. The condition, while one of increased nutrition, a true hypertrophy in its earlier stages, and a fatty degeneration in its later contracting stage, cannot be considered an inflammatory one; and therefore I prefer the term large white kidney to any involving the idea of inflammation.

In this form of disease we have also dropsy, considerable albumen, at first small hyaline casts, containing an occasional oil-globule or fragment of epithelium, later more copious urine and more numerous small hyaline, and oily casts, and finally large hyaline, granular, and oil casts, indicating destruction of the gland, the rate of which may be estimated by the quantity of this kind of deposit.

Next we have the cirrhotic or chronically contracted kidney, also called the gouty kidney, and by the German pathologists the kidney of interstitial nephritis, in contradistinction to the catarrhal nephritis, because the interstitial connective tissue is believed to be the seat of the inflammation. Here again I do not think the phenomena are those of a true inflammation, but rather of a nutritive activity in the normally scanty connective-tissue elements between the tubules, to the resulting fibrillar element of which is also contributed a portion derived from the atrophy of the tubules themselves. A hypertrophy of the muscular coats of the arterioles of the kidney, and perhaps, also, as claimed by Johnson, of the entire arterial system, is also an essential feature.