the effects on nerve-cells of the prolonged use of alcohol. Under the influence of alcohol the granular masses in the Parkinje cells disappeared, the protoplasm of the body of the corpuscle lost its characteristic structure, and the nucleus was very greatly altered and distorted in shape. Under the toxic influence of chronic alcoholism the dendrites of the pyramidal cells exhibited swellings and shrinkages. A remarkable instance of the effect of alcohol on the small pyramidal cells had been shown by Nissl and the differences between such a cell in its normal condition and after poisoning by alcohol was demonstrated by means of a lantern-slide.

FIG. 2.

From the *Zeitschrift für Psychiatrie*, Band liv., Taf. III., Fig. 5.

Finally, a remarkable constant feature of degeneration in the alcoholic person's brain was widespread pigmentation in the nerve cells. The presence of pigment in the nerve cells in the human brain was a normal fact and occurred in other animals, but in alcoholic poisoning it became exaggerated at the same time that the protoplasm of the cell shrank, so that ultimately the nerve corpuscle was represented by a shrivelled mass of pigment. In certain stages shown in a paper by Dr. H. Wright and Dr. M. Orange, published in Dr. F. W. Mott's *Archives of Neurology* under the auspices of the London County Council, the degenerating effect on the protoplasm of the bodies of the nerve cells was illustrated, clear bladder-like spaces or vacuoles being produced.

Mr. Horsley concluded by stating that he thought from a scientific standpoint that the previous contention which they had had so often put before them by their friends that small doses of alcohol such as people take at meals had practically no deleterious effect could not be maintained. He had only touched upon a very small part of the subject, but if they considered the mass of work that had been accumulated from the observations of Parkes on physical work, from those of Ridge on extremely small doses on vegetable protoplasm, and from the observations of Abbott and others on the influence of alcohol on animals in lowering their resistance to microbic invasion, they could only come to one conclusion: that from a scientific standpoint total abstinence must be their course if they were to follow the plain teaching of truth and common-sense. It was the part of the scientist to point this out but it was the part of the politician to persuade the nation to adopt it as a whole.

[The Very Rev. F. W. FARRAR, Dean of Canterbury, in moving a vote of thanks to Mr. Horsley, said that he considered that it was the duty of the nation to persuade the politicians, and he regarded the lecture as marking an epoch in reform.—Dr. W. J. COLLINS, in seconding the vote of thanks, said that Mr. Horsley had laid before them the latest results of science without bias and without partiality.—Sir WILFRID LAWSON, Bart., M.P., moved a vote of thanks to the Chairman which was seconded by Mr. T. P. WHITTAKER, M.P.—The CHAIRMAN having suitably responded the proceedings terminated.]

ST. JOHN AMBULANCE ASSOCIATION.—The Central Executive Committee of the St. John Ambulance Association has authorised the formation of a centre at Bath. An influential committee has been appointed with Colonel Wyndham Murray, M.P., as president.

OBSERVATIONS ON THE EPIDEMIOLOGY OF PLAGUE.

By T. FREDERICK PEARSE, F.R.C.S. ENG.,
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SPECIAL PLAGUE MEDICAL OFFICER, CALCUTTA.

THE accompanying chart shows the courses of the three successive outbreaks of plague in Bombay city and of one in Poona, and for comparison, but on a different scale, the course of the second outbreak of plague in Calcutta. The returns of the weekly general mortality are taken because these seem the most reliable estimate of the extent of the epidemic, the excess mortality above the average being for all practical purposes the amount of the plague. The official returns of plague are so utterly untrustworthy that in the absence of any other cause for an increased death-rate the excess mortality above the normal becomes the only guide. With regard to Bombay, it will be seen that each outbreak occupied about the same period of time. There is in each case the same rapid rise, the same fluctuating summit of highest mortality, and the same correspondingly rapid fall. The charts of each outbreak have been super-imposed in order to emphasise the corresponding weeks of highest mortality, but it must be clearly understood that these highest points did not occur at the same dates in successive years. The second outbreak in Bombay was seven weeks later in the season than the first, and the third occurred about three weeks later in the season than the second. Although the first outbreak culminated at the coolest period of the year, the second rose to its highest point as the weather was getting warmer, and the third outbreak showed its still higher range of mortality in the hot month of March. Again, the outbreaks at Calcutta and Karachi occurred during the hottest months of the year, that at Poona during the monsoon, and the Southern Mahratta country was devastated at the close of the monsoon and while the winter was coming on. These facts show that season and temperature have little or no influence in determining the rise or fall of an outbreak.

It will be observed that there is a break in the steady and continuous rise of mortality in the first and third outbreaks in Bombay. There is also a break during the fall of the same two outbreaks. In the Calcutta curve there is also a break in the rise and a very marked secondary rise for one week during the months of the fall. It is suggested that these are not accidental, but there is nothing upon which to ground an explanation. The fluctuating summit of highest mortality in each outbreak lasting from four to six weeks is very peculiar. The severity of each outbreak in Bombay can be roughly estimated as the average weekly mortality is between 500 and 600 and on this basis it is assumed that about 22,000 lives have been lost each year from plague alone.

These characteristics of a plague outbreak in a large city are reproduced in the chart prepared from the mortality returns of the city of Poona and to a certain degree also in that for the city of Karachi. The mortality in Bombay city at each outbreak, in Karachi, and in the previous outbreak in Poona, has been roughly estimated at 1 in 40 of the population, but in the last and third outbreak in Poona it has been much more severe—more than equalling in its devastating effects the two previous attacks combined—and carrying off nearly one in every ten of the estimated population. There appears to be no explanation to account for this and there is no parallel to it in any part of India. The chart for Poona, drawn on the same scale as that for Bombay, is very similar. Considering that this city has only about a sixth of the population of Bombay the terrible severity of the last outbreak is very evident. In comparing all the lines it will be seen that the week of highest mortality occurred in three of the outbreaks immediately before the decline.

Another point of interest, but which is not shown by the chart, is the heightened general mortality for weeks and

The chart shows the general weekly mortality in Calcutta, Poona, and Bombay. The thick line indicates the general weekly mortality in Calcutta from Jan. 7th to June 10th, 1899. The thin line indicates the general weekly mortality in Poona (third outbreak) from June 16th to Nov. 3rd, 1899. The broken line indicates the general weekly mortality in the three outbreaks in Bombay, the dates taken being in the first case from Oct. 6th, 1896, to April 27th, 1897; in the second case from Nov. 23rd, 1897, to June 13th, 1898; and in the third case from Dec. 13th, 1898, to July 4th, 1899. The figures in the vertical column to the left refer to Calcutta, and in that to the right they refer to Bombay.

months before the outbreak definitely shows itself. The decline, moreover, of each attack though rapid at first terminates slowly and a plague outbreak seems to linger on for weeks and months before finally disappearing. In Bombay city, however, it is doubtful whether there has been a single week clear since the declaration of plague in September, 1896.

With reference to the various diseases making up the general mortality it is important to note that other epidemic diseases were conspicuous by their absence. Cholera, dysentery, epidemic diarrhoea, and fevers generally were all, as far as imperfect registration indicates, considerably below the average. In Bombay relapsing fever was the only concurrent fever and in each of the above towns the mortality from cholera was exceptionally low. It only remains to give the figures of the weekly general mortality in Calcutta, Poona, and the three epidemics of Bombay upon which the accompanying chart is based.

I.—CALCUTTA.

Mortality for Week Ending Jan. 7th, 1899, and Following Weeks.

562	483	604	743	631	436
520	450	628	722	517	435
543	532	799	619	443	372
521	572	716	562	387	

II.—POONA.

Mortality for Week Ending June 16th, 1899, and Following Weeks.

126	357	1082	889	410	125
159	548	1087	635	305	109
205	815	1112	584	241	76
302	1102	1182			

III.—BOMBAY.

First Outbreak.—Mortality for Week Ending Sept. 1st, 1896, and Following Weeks.

667	606	772	1639	1640	1007
593	698	1051	1758	1465	970
618	663	1310	1726	1326	836
647	623	1416	1645	1258	671
720	704	1853	1911	1139	638
791	760	1711	1728	1141	558
634					

Second Outbreak.—Mortality for Week Ending Oct. 19th, 1897, and Following Weeks.

776	706	1540	2080	1303	633*
754	785	1726	2184	1202	589
741	835	1871	2137	1116	525
701	975	2067	2268	872	504
700	1061	2195	1938	725	448
682	1307	1974	1519	616	500
704					

* Doubtful.

Third Outbreak.—Mortality for Week Ending Nov. 8th, 1898, and Following Weeks.

645	798	1601	2431	1696	1047
604	792	1612	2312	1503	890
606	894	1803	2408	1305	711
616	1111	1907	2290	1099	594
679	1239	2249	2010	1127*	609*
688	1448				

* Doubtful.

Calcutta.

REMARKS ON VASECTOMY RELATIVE TO ENLARGED PROSTATE AND BLADDER ATONY.

BY REGINALD HARRISON, F.R.C.S. ENG.,
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IN a recent article¹ I ventured to state that "if vasectomy and castration in relation to prostatic enlargement had done no more than direct attention again to its pathology they would have served a useful purpose," and I then took the opportunity of illustrating some varieties in shape and structure that were included in the term "hypertrophied prostate." Hence I urged that it was about as reasonable to expect to find a uniform treatment for it as it would be in the case of what "enlarged breast" might be made to express. The mass of material which the student of morbid anatomy has now within his reach calls from time to time for its application to the purposes of classification and treatment. In no department of work is this more necessary than in the case of the urinary organs from whatever portion of the entire apparatus it is viewed.

It has been extremely interesting to follow the various communications that have been made during the last few years relative to the employment of vasectomy and castration in the treatment of prostatic hypertrophy. In some cases the results have been admirable, in others doubtful, whilst between these two extremes there have been various gradations. In my own practice since my advocacy of vasectomy in 1893 I have performed the operation in over 100 cases and I can say that whatever effects were exercised upon the prostate I have never seen any harm result or heard of any regret expressed from what followed other than that arising in a few instances from failure to obtain by comparison all the good that might have been anticipated. A number of these cases have now been under observation, or cognisance, for from one to seven years, so that ample opportunity has been permitted for noting what happened during varying intervals after operation. I have already referred to a previous communication in which stress was laid on the fact that there were structural conditions of the prostate when enlarged where shrinkage was unlikely to happen after either castration or vasectomy as illustrated by carcinomatous, fibrotic, and adenomatous glands. These all simulate the commoner form of prostatic hypertrophy and though vasectomy does no harm it is rarely followed in these instances by appreciable good.

The present communication has for its main object the demonstration of the existence of certain physical changes in the bladder arising out of chronic prostatic obstruction which are sufficient to negative or minimise the effects that would otherwise follow the induction of shrinkage of the prostate by such means as are under consideration. This point presents itself in three ways: (1) from the examination after death of persons who had undergone vasectomy or castration for hypertrophied prostate; (2) from the exploration of the interior of the bladder by suprapubic cystotomy under similar circumstances; and (3) from the examination of collected specimens illustrating various kinds of atonic bladders in persons who had been more or less dependent on the catheter with hypertrophied prostates. Of the first-mentioned I have met with two examples and of the second four. In five of these vasectomy and in one castration had been practised some months previously. In none of them was death caused or the operation of cystotomy necessitated by the vasectomy or castration, these events being brought about by independent derangements of the kidney or the bladder which arose out of the original state of chronic prostatic obstruction. All instances showed evidence of varying but marked degrees of prostatic shrinkage though the complications mentioned were uninfluenced. All had benefited to some extent from the shrinkage of the gland which had thus been artificially induced, though the improvement was chiefly confined to matters relating to catheterism and washing out the bladder, but none of these six cases had resulted in the restoration of the natural function of micturition or of catheter independence. The examination

of the third group, which included a considerable number of atonic bladders associated with enlarged prostate removed after death from patients who had been more or less dependent on the catheter for long periods of time, indicated the nature of the commoner lesions met with in the bladder under the latter circumstances.

From one or other of these sources I was able to demonstrate (1) that the usual effect of vasectomy is to induce shrinkage of the hypertrophied prostate; (2) that though this shrinkage affords a readier access to the bladder for catheters and such like purposes it did not necessarily follow that voluntary and natural micturition was thereby restored, failure in this respect being mostly due to structural changes in the bladder itself arising out of long-continued obstruction; and (3) that the latter consists of certain kinds and degrees of sacculation, pouching, and trabeculation of the bladder whereby its voluntary expulsive power is permanently and frequently irrecoverably damaged.

That the usual effects of vasectomy on the hypertrophied prostate is to bring about in the course of time its shrinkage or atrophy is, I think, generally admitted. Further, it would seem that if the prostatic shrinkage thus induced is not followed by the cessation or improvement of the symptoms characteristic of prostatic obstruction, and which includes the return of natural micturition, it may be assumed that either the gland itself is at fault in other ways, as indicated in my previous paper, or that the bladder is now solely responsible for the failure.

The examination of the cases and specimens referred to previously in the three groups indicates that by far the commonest cause of bladder failure under these circumstances does not necessarily arise out of deterioration or loss of muscular substance requisite for expelling urine but is largely due to altered relationships between the mucous and muscular investments of the viscus. Take, for instance, as illustrations of the latter the sacculated, pouched, trabeculated, and herniated mucous coat bladders which are the common results of protracted obstruction. The power is there but it is misdirected. The mode in which a comparatively small sac in the bladder is capable of throwing the whole muscle "out of gear," to use a very apt term, is capable of demonstration in various ways.

Some years ago I saw a man, under 50 years of age, without any stricture or prostatic obstruction, who had met with an accident whilst hunting and was partially thrown from his seat when there was reason for believing that he had a distended bladder. He experienced some pain in the region of the bladder immediately, but was thought at the time not to be seriously hurt. Some weeks afterwards I was consulted by him for offensive urine and for difficulty in emptying his bladder, for which, since the accident, a catheter had been necessary. Eventually he became entirely dependent on the latter and died in the course of a few months from chronic cystitis. A herniated pouch of mucous membrane just above the trigone which would admit the last joint of the thumb revealed the primary cause of his illness from his accident to his death. There was no evidence during life or after death of spinal injury. It was clear that this protrusion of a pouch of mucous membrane through the meshes of the muscular coat had brought the natural function of the bladder somewhat abruptly to an irreparable end.

Again, from time to time I see a man, aged 50 years, from whom I removed a single urate stone of about the size of a small walnut, situated, or rather embedded, in a pouch just within the posterior wall of the bladder. The operation consisted in seizing the stone (which could only be felt with the side of a sound) with the lithotrite and putting it in a part of the bladder area where it could be readily broken into fragments and removed with the wash-bottle and evacuator. The embedded condition of the stone had previously interfered with the patient's expulsive powers and he was, before I saw him, largely dependent upon his catheter though he had no large prostate to account for this. After the stone was removed from the sac, though he was completely relieved from pain and regained his general health, he became entirely dependent upon his catheter—a circumstance which could only be explained by the structural alteration the bladder had thus undergone and which at his age is not likely to be remedied. I have had more than one instance of this and have recently reported two cases²

¹ THE LANCET, August 5th, 1899, p. 331.

² Two Cases where Multiple Calculi were Removed from large Narrow-necked Sacculi connected with the Male Urinary Bladder. Transactions of the Royal Medical and Chirurgical Society, vol. lxxxii., 1899.