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JULY 1944.

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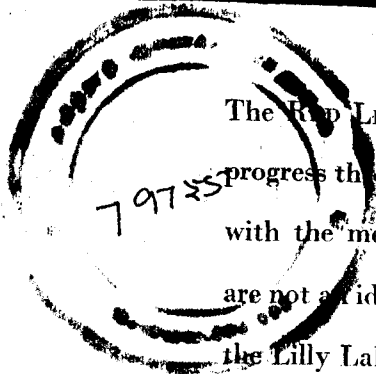
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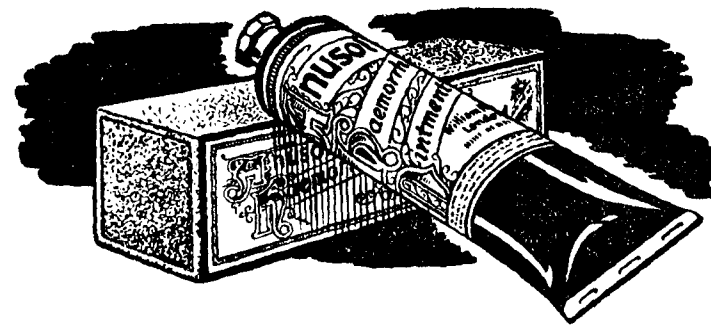
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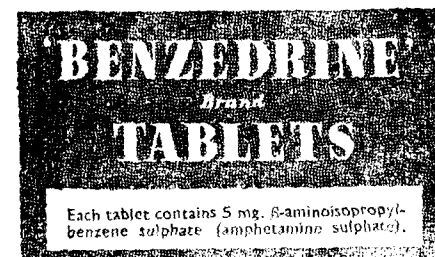
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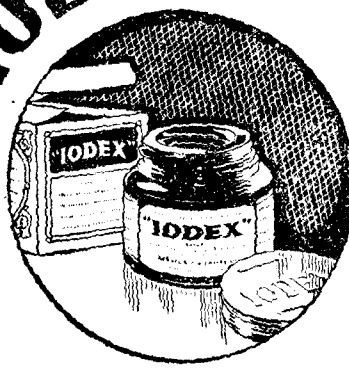
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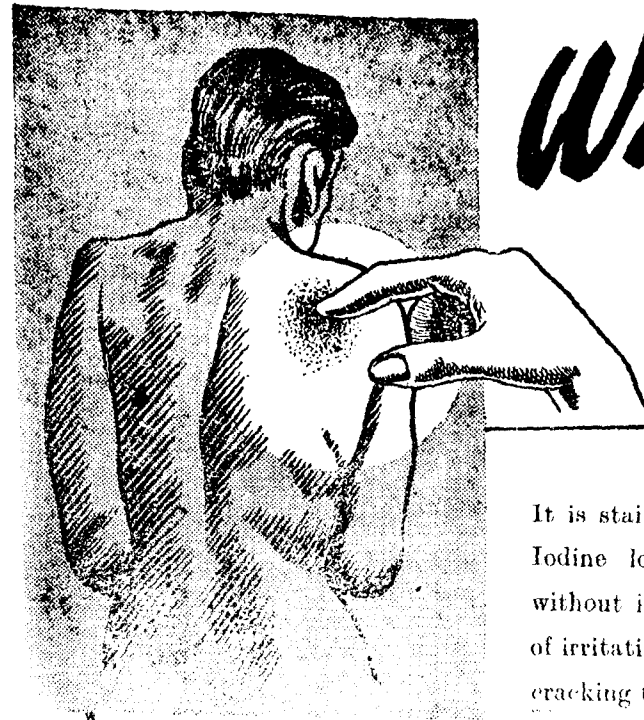
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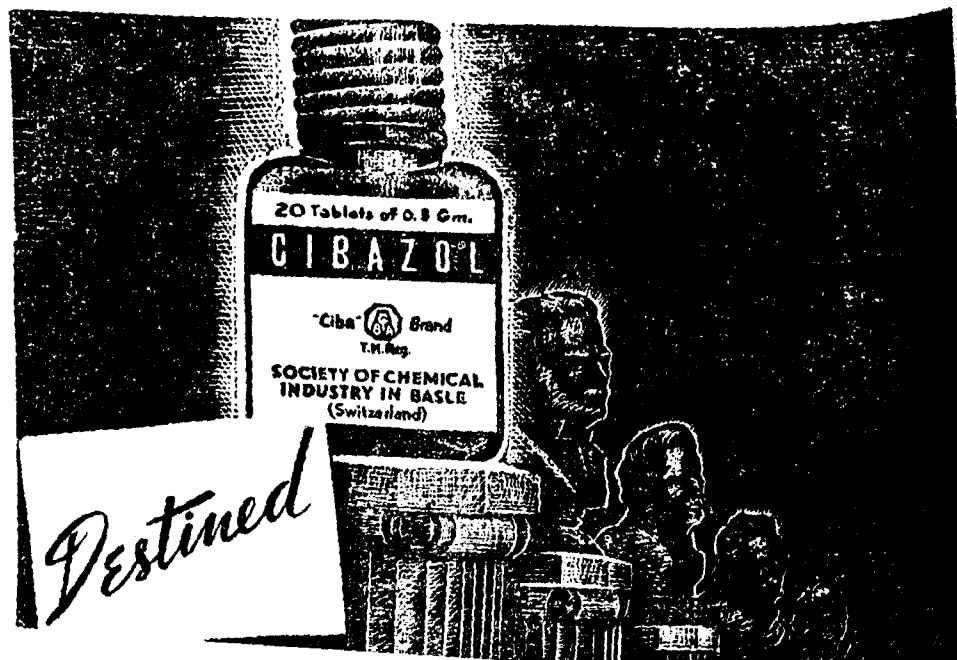
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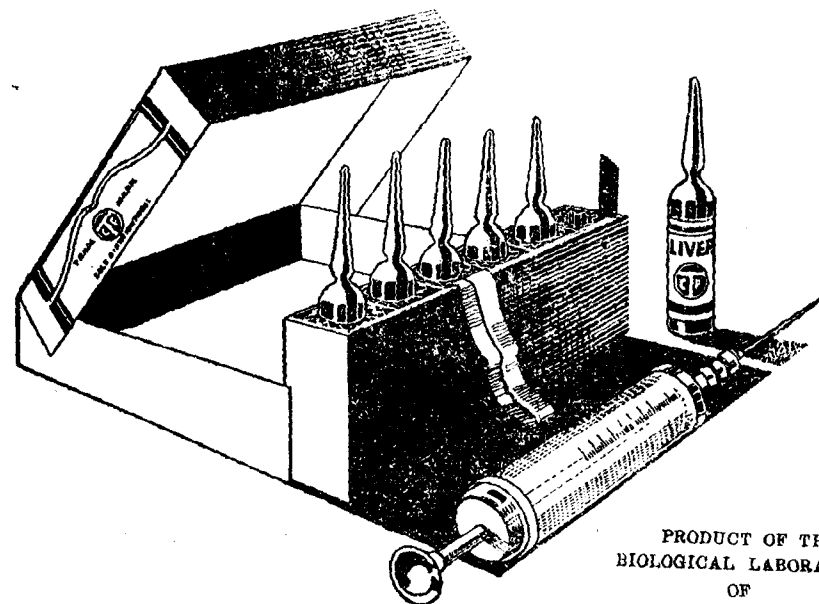
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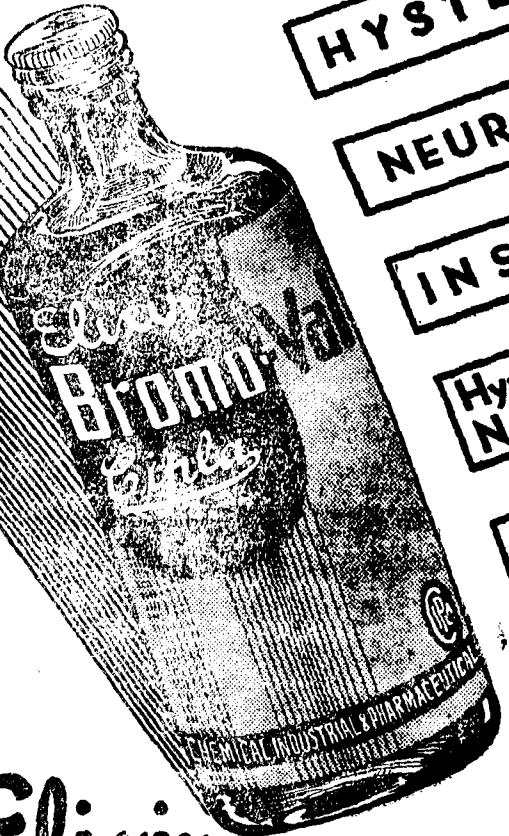
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Original Articles

The Tuberculosis Problem in the City of Madras*

BY

K. VASUDEVA RAO, M.D., M.R.C.P. (E.), T.D.D. (Wales),
Superintendent, Government Tuberculosis Hospital, Madras.

THE problem of tuberculosis in the city of Madras is so heart-rending that it calls for immediate attention and hearty co-operation of the city fathers.

To bring home to you the present position of tuberculosis in this city, I intend putting before you a few facts which came under my observation during my experience as a tuberculosis officer in the city of Madras.

Taking a pre-war year—1939, as the basis, it has been found that the number of patients who attended the Government Tuberculosis Institute, Egmore, was 11,599 of which 3,992 were found to be suffering from tuberculosis and of these 3,744 were found to be suffering from pulmonary tuberculosis and the rest from non-pulmonary tuberculosis. In this connection I would like to point out that tuberculosis may affect any portion of the body and it is not confined only to the lungs. In the above 3,744 cases, the sputum was positive for tubercle bacilli in about 1,602 cases out of which 510 cases came from out of Madras, thus Madras contributing 1,092 cases. These 1,092 open cases are the cases that were diagnosed at the institution, namely, the Government Tuberculosis Institute, Egmore. There might be very many other cases diagnosed to be suffering from tuberculosis at the other institutions in the city or by private medical practitioners which have not been taken into consideration. If these are added, I am almost certain the number will be 10 times more i.e., about 10,000.

* A Radio speech given to the City fathers and sent to 'The Antiseptic' for publication.
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A little more study of these open cases, if I am allowed to go into, will show the various factors which are operating in these cases. During this period, the members of my staff and myself have tried our best to get all the information available with regard to every open case. A particular type of enquiry card is being filled in and the enquiries done on all these 1,602 cases show that the largest number of open cases belong to the male section—1,231 and females 371.

The disease predominated among the Hindus which could be seen from the following table:

The following table gives the age periods of the 1,602 cases with sex and religion—

	Males	Females
Hindus	1,001	311
Mohammadans	132	34
Christians	86	22
Anglo-Indians	12	4
Total	1,231	371

The Hindus predominate.

Age Periods

Age periods	Hindus		Mohammadans		Christians		Anglo-Indians	
	Males	Females	Males	Females	Males	Females	Males	Females
0-10	11	8	1	1	1	—	—	—
11-15	6	13	3	—	2	2	—	—
16-20	109	70	23	7	9	3	2	2
21-25	222	70	27	12	19	6	2	1
26-30	200	76	34	7	18	6	2	—
31-35	152	27	21	4	13	1	2	1
36-40	121	27	12	3	7	2	—	—
41-45	79	8	6	—	5	—	1	—
46-50	56	9	5	—	7	2	2	—
51-55	19	2	—	—	3	—	—	—
56-60	15	1	—	—	2	—	—	—
61-65	8	—	—	—	—	—	—	—
66-70	3	—	—	—	—	—	—	—
71-75	—	—	—	—	—	—	—	—
Total	1001	311	132	34	86	22	12	4

It will be observed that the highest peak of incidence of open tuberculosis among the males and females was practically the same age found, i.e., 21 to 25 years.

Table II gives you an idea about the number of cases that have come from each Corporation division and wards:

TABLE II

Division.	No. of patients.	Division.	No. of patients.
1. George Town	220	28. Adayar	12
2. Choolai	109	29. Mirsahibpet	11
3. Triplicane	103	30. Tondiarpet	11
4. Washermanpet	72	31. Aminjikarai	10
5. Purasawalkam	72	32. Poonamallee	10
6. Chintadripet	64	33. Krishnampet	10
7. Perambur	63	34. Old Mambalam	9
8. Royapettah	51	35. Kosapet	8
9. Royapuram	43	36. Elephant Gate	8
10. Pudupet	41	37. Kodambakkam	8
11. Park Town	40	38. Tiruvottiyur	8
12. Mylapore	39	39. Tiruvatteeswaranpet	8
13. Saidapet	38	40. Muthialpet	8
14. Mount Road	34	41. Pulianthope	7
15. Periamet	31	42. Villivakkam	7
16. Egmore	29	43. Pallavaram	6
17. Thousand Lights	24	44. Komaleeswaranpet	6
18. Mannady	21	45. Ayanavaram	6
19. Kilpauk	20	46. Tambaram	5
20. Vepery	17	47. Thyagarayanagar	5
21. Teynampet	16	48. Sowcarpet	3
22. Nungambakkam	15	49. Tawker's Choultry	3
23. Otteri	15	50. Sembiam	3
24. Vyasarpady	15	51. Korukkupet	1
25. Kondithope	13	52. Guindy	1
26. St. Thomas Mount	13	53. Vandalur	1
27. Chetpat	12	54. Avadi	1

Table III gives the occupation of the individuals treated:

TABLE III

Occupation.	No. of patients.	Occupation.	No. of patients.
Coolies	230	Fishmongers	12
Cultivators	166	Railway Servants	10
Clerks and officers	91	Mechanics	10
Mill coolies	79	Fitters	8
Beedi rollers	57	Dhobies	7
Peons and Domestic servants	40	Cobblers	6
Tailors and Emroidery workers	40	Painters	6
Compositors and Binders	36	Pilot Boatmen	6
Students	35	Village Karnams	5
Cooks	34	Hotel servers	5
Carpenters	32	Tramway drivers and conductors	4
Weavers	30	Tinkers	3
Merchants and Shopmen	29	Teasellers	3
Drivers	25	Barbers	3
Betel-shop keepers	25	Compounders	2
Teachers	23	Blacksmiths	2
Gardeners	21	Cinema workers	2
Goldsmiths	17	Pensioners	1
Sweepers and Scavengers	15	Eggs seller	1
Police Constables	15	Musician	1
Milkmen	14	Book-seller	1
Hawkers	14	Purohit	1
Masons	13	Beggar	1
Electricians	12	Photographer	1
		Flower-seller	1

Table IV (a) shows the housing conditions of the patients treated and IV (b) of their economic conditions.

Information with regard to the housing was on the following points:

1. Type of houses.
2. Number of living rooms. } Table IV (a).
3. Amount of rent paid and }
4. Number of people living together.—Table IV (b).

TABLE IV (a)

Types of houses	Males	Females
Huts	84	9
Tiled Houses	424	94
Terraced Houses	181	25

Classification according to the number of rooms occupied:

Number of rooms occupied	Males	Females	Number of rooms occupied	Males	Females
1	431	82	4	28	1
2	144	40	5	12	10
3	45	8	6 or above	12	8

Amount of rent paid by the patients:

Amount of rent	Males	Females	Amount of rent	Males	Females
8 Annas	28	5	Rs. 5 to 6	25	20
Rs. 1/8	56	4	Rs. 6 to 7	10	4
Rs. 2	24	12	Rs. 7 to 8	8	4
Rs. 2/8	75	18	Rs. 8 to 9	8	5
Rs. 3	72	14	Rs. 9 to 10	4	3
Rs. 3	95	20	Rs. Above 10	20	11
Rs. 3/8 to 4/8	153	24	Own Houses	20	16
Rs. 5	44	22			

TABLE IV (b)

Patients according to the number of families living together:

According to the number of families living together :					
Families	Number of patients		Families	Number of patients	
	Males	Females		Males	Females
1	82	10	5	119	44
2	60	8	6	75	29
3	79	9	above 6	92	30
4	163	28	Maximum number families	10	

The following table gives the income of the patients:

Income in Rupees	Number of patients		Income in Rupees	Number of patients	
	Males	Females		Males	Females
1-5	15	4	61-65	1	—
6-10	267	38	66-70	—	1
11-15	309	93	71-75	—	—
16-20	125	53	76-80	1	—
21-25	71	27	81-85	2	1
26-30	48	15	86-90	—	1
31-35	17	5	91-95	—	1
36-40	22	4	100	1	—
41-45	7	4	180	1	—
46-50	9	2	200	1	—
51-55	1	—	250	1	—
56-60	1	—	600	—	1

The greatest number of patients have an income of below Rs. 20.

The following table gives the number of persons coming in contact with the positive cases.

Sex	Number of patients	Family members of close contacts living with the patients	No. of co-tenants living in the same house as the patient
Males.	1231	3973	4638
Females.	371	1548	1526

	Males	Females	
Total number of family members	3,973	1,548	Total ... 5,521
Total number of co-tenants	4,638	1,526	„ ... 6,164
Total number of contacts among the 1602 positive cases			... 11,685
Total number of contacts examined			... 3,224
Number of houses visited for this			... 820
Average number of contacts examined for each patient			... 2.0
Number of contacts Tubercular			... 480
Percentage of the Tubercular			... 14.9 or 15%

From the above the following facts are evidenced:

- (1) that the largest number of open cases of tuberculosis were seen in both sexes between ages 21 and 25;
- (2) that the largest number of open cases are found in the George town area (220), Choolai (109), Triplicane (103), Washermanpet (72) and Purasawalkam (72);
- (3) that the poorer classes like the coolies have the greatest amount of morbidity rate, i.e., 230;
- (4) that in over-crowded and ill-ventilated houses the rate of spread and morbidity are greater;
- (5) that economically poor people suffer most and most of the cases are among people whose income is Rs. 20 and below.

The havoc that is being caused by the presence among us of these 1,602 cases, is shown by the number who came into contact both in their families—domestic contact, or outside social contact; family contact of

1,602 open cases is 5,521 and social contact 6,164 and so the total number of persons who came into contact were 11,685. My staff and myself have been trying to follow these open cases and examining all those who came in contact with these open cases. During the year under review we visited 820 houses and we found nearly 15% of these contacts were suffering from active open tuberculosis. The fact that emerges from this enquiry has been that these 15% of cases have not had any manifestation of tuberculous disease compelling them to seek the aid of a doctor but at the same time they are very great sources of infection. The danger of the open cases being amongst us can well be understood when it is brought to your notice that millions and millions of tubercle bacilli are being thrown out by each of the open case of pulmonary tuberculosis and their sputum is the fountain source of infection. It is no exaggeration when it is said that the bacilli that are brought out by one single individual can cover a distance of 12 miles when placed end to end.

Thus it could be seen that the morbidity rate of tuberculosis is great and the number of people suffering from tuberculosis who need attention is somewhere about in the neighbourhood of 16,000.

The next aspect of the question is to see what is the rate of tuberculosis mortality in this city. On an average there is one death from tuberculosis for every seven deaths and among adults one death in every third death. During the year 1940 according to the report of the Health Department of the Corporation of Madras, there were 1,064 deaths from tuberculosis. This is but a very meagre estimate as large number of deaths where tuberculosis is responsible are often reported as deaths due to other diseases such as pneumonia, bronchitis, etc. Possibly the social asceticism which is to a certain extent prevalent may also be a factor in not having a correct cause of death. Other factors such as fear of insurance acceptance or ignorance on the part of the relatives occur.

It would be seen from the following table, the Corporation divisions where large number of deaths occur:—

Division No.	Males	Females	Total	Division No.	Males	Females	Total
13	33	47	80	B. P.	414	400	814
11	34	45	79	1	11	9	20
35	36	28	64	28	12	8	20
19	25	33	58	15	13	6	19
20	30	22	52	17	16	3	19
4	24	25	49	21	13	6	19
5	24	21	45	30	9	9	18
37	21	23	44	3	9	7	16
9	20	22	42	31	9	7	16
25	18	17	35	7	9	6	15
36	18	16	34	34	7	7	14
26	19	12	31	27	9	1	10
23	21	9	30	29	4	6	10
24	14	13	27	40	3	7	10
12	10	16	26	2	5	4	9
28	18	8	26	8	4	5	9
32	16	9	25	10	8	1	9
33	9	15	24	6	3	3	6
18	16	6	22	14	3	2	5
39	8	13	21	22	3	1	4
Total	414	400	814	16	0	2	2
				Total	564	500	1064

The mortality rate as at present available from the investigations made by my staff and myself for the city of Madras is in the neighbourhood of 456 per 100,000. Basing on the European standards of working out the morbidity rate from the mortality rate, it will be seen that there would be at least 10,640 cases (worked out on the basis of figures furnished by the Health Department, Corporation of Madras—Report for 1940). As per the investigations made by the staff of the Government Tuberculosis Institute it would be 1092×10 is equal to 10,920 which is practically the same. When such amount of tuberculosis mortality is present at Madras, let us turn to the facilities that are available for the treatment of these cases.

In the city of Madras we have only one hospital at Royapettah with 62 beds and a sanatorium in the neighbourhood with 80 beds thus making a total of 142 beds. 12 beds are reserved in the Government General Hospital for tuberculous cases. Comparing the figures available in other Provinces regarding the number of beds available for tuberculous cases we are certainly better off but what we have is very small when compared to the large amount of mortality. The facilities that are made available in certain countries for tuberculosis are given below:—

New Zealand:—For every death that takes place from tuberculosis, there are 2 beds reserved for institutional treatment.

Denmark.—For every death from tuberculosis 1.8 beds are reserved for institutional treatment, or, in other words, for 100 deaths 180 beds are reserved.

Denmark which is possibly the second best in mortality rate shows 40 deaths per 100,000, the first best being New Zealand with a death-rate of 32 for every 100,000. Compare these figures with that of Madras and also the provision that has been made for treatment of the open cases.

To my knowledge in Western countries generally at least one bed is made available for every death from tuberculosis. Basing on this standard it is indeed appalling to see, for 1064 deaths we have only 154 beds. But all these 154 beds are not reserved for the patients coming from the city of Madras. These have to cater to the needs of those coming from the whole of this Presidency. The net result of this appalling insufficiency of accommodation is that a large number of open cases, who are a great source of infection and danger to the members of the society are let loose and they are the sources for the fresh spread of the disease.

What is the remedy for this?

The remedy lies in taking active measures to combat the disease. This could be divided into two groups: (1) the special preventive measures and (2) active measures against the disease.

The general preventive measures consist of (1) improvement of nutrition; (2) improvement of housing conditions and (3) improvement of medical care among school children.

The direct attack on the disease is by finding out cases—survey, establishment of clinics and establishment of institutions for isolation of cases.

The first word in combating tuberculosis is the segregation of the infected individual and the segregation could only be done if sufficient accommodation is available. As far as Madras city is concerned, the first should be the establishment of tuberculosis clinics and tuberculosis hospitals. For the whole of the city we have only one tuberculosis clinic the

Government Tuberculosis Institute, Egmore. Basing on the Western standards which with very great advantage could be copied as far as tuberculosis is concerned—as large amount of spade work has already been done—there should be one tuberculosis clinic for every 50,000 of population. The present population of Madras is about 10 lakhs and according to the above standard there should be at least 20 clinics.

It may not be practicable to start 20 clinics at a time, but I would suggest that at least 4 clinics must be started immediately in the following places:

(1) In Royapuram—to cater to the needs of Royapuram, Washermanpet, Harbour division, Seven wells, etc.

(2) In China Bazaar—to cater to the needs of George Town.

(3) In Royapettah—to serve the needs of Royapettah, Triplicane, Mylapore, etc.

(4) In Choolai—to serve the needs of Choolai, Perambur, Purasawalkam, etc.

It is not possible to build the clinics immediately owing to the non-availability and high cost of materials required for construction, but it may be possible to house those clinics in the present Corporation dispensaries which are already in existence and those clinics can work, to start with, on 2 or 3 days in a week, in the afternoon.

The clinics are the pivots on which any anti-tuberculosis work has to revolve. To house the cases that will be diagnosed as tuberculosis at the clinics, provision must be done for some institutions and to start, with a hospital of at least 100 beds is suggested to be started immediately by the Corporation of Madras.

The initial and recurring costs of a clinic and a hospital are given below:—

Non-Recurring and Recurring Cost for a Tuberculosis Clinic

NON-RECURRING		RECURRING (PER ANNUM)	
	Rs.		Rs.
Building	10,000	Pay of 2 medical officers (Rs. 200 & 100)	3,600
X-Ray Plant	10,000	Pay of 2 health visitors @ Rs. 50 plus conveyance allowance of Rs. 15 each	1,560
Furniture	1,000	Pay of 2 nurses @ Rs. 35 each	2,040
Other equipments	500	Pay of 1 X-ray assistant @ Rs. 60	720
Laboratory equipment:		Pay of 1 laboratory attendant @ Rs. 25	300
A microscope	700	Pay of 1 clerk @ Rs. 35	420
Sedimentation apparatus	100	Pay of 1 compounder @ Rs. 30	360
Blood pressure apparatus	100	Pay of 2 nursing orderlies @ Rs. 15 each	360
Hæmoglobin apparatus	100	Pay of 1 sweeper @ Rs. 12	144
Culture materials and incubator	500	Cost of X-Ray films @ Rs. 2-8-0 per film for about 20 patients per day	15,000
Laryngoscopic examination apparatus	150	Cost of medicines	3,000
Stains and other chemicals	150	Electricity, water, contingencies etc.	1,000
Centrifuge etc.	600		
Total	23,900	Total	28,540

N. B.—The cost per patient per diem at the Government Tuberculosis Institute Egmore including medicines, medical aid and other expenses during 1943 is Rs. 0-5-10.

Non-Recurring and Recurring Cost for a Hospital of 100 Beds

NON-RECURRING		RECURRING	
	Rs.		Rs.
1. Buildings (5 wards) ...	60,000	1. Maintenance of buildings ...	4,500
Operation theatre ...	18,500	2. Replacing condemned linen ...	600
X-Ray room ...	17,800	3. Maintenance of equipment ...	900
Laboratory ...	15,800	4. Diet of patients at annas 14 per diem per patient ...	31,500
Dispensary ...	2,700	5. Medicines, hospital necessities and X-ray films etc. ...	9,500
Quarters for one medical officer ...	5,000	6. Other contingencies such as pay of watchmen, cooks, gardeners etc. ...	3,500
	1,19,800	7. Pay of Establishment: Rs.	
2. Electric sanitary fittings: ...	7,000	(a) One Asst. Surgeon @ Rs. 300 ...	3,600
5 wards ...	2,775	(b) One Sub-Asst Surgeon @ Rs. 100 ...	1,200
Operation theatre ...	2,670	(c) 8 staff nurses @ Rs. 65 plus ration allowance 22/10/- plus D.A. 10 and uniform allowance Rs. 56/4/- per annum each ...	9,918
X-Ray room ...	2,370	(d) 2 clerks @ Rs. 35 plus D.A. Rs. 8/8/- each ...	1,044
Laboratory ...	500	(e) 6 male nursing orderlies @ Rs. 15/- plus D.A. Rs. 8/8/- ...	1,692
Medical officer's quarters ...	500	(f) 4 female nursing orderlies @ Rs. 12/- plus D.A. Rs. 8/8/- ...	984
	15,315	(g) 5 male thoties @ Rs. 15/- plus D.A. Rs. 8/8/- plus bonus at Rs. 2/- p.m. ...	1,530
3. Water supply and drainage ...	13,750	(h) 3 female thoties @ Rs. 12/- plus D.A. Rs. 8/8/- plus bonus at Rs. 2/- p.m. ...	810
4. Equipment: ...	10,000	(i) Miscellaneous ...	502
Operation theatre ...	10,000	Total recurring expenditure per annum ...	71,780
X-Ray with accessories ...	5,000		
100 cots ...	3,000		
100 lockers ...	800		
100 stools ...	640		
8 almirahs ...	200		
20 teapots ...	800		
Benches, tables etc. ...	2,000		
Laboratory equipments ...	3,000		
Miscellaneous ...	35,440		
5. Linen as per sanctioned scale: ...	1,650		
Lobies 165 ...	750		
Sheets 500 ...	1,000		
Counterpanes 500 ...	625		
Draw Sheets 500 ...	275		
Pillow cases 580 ...	312		
Cot mattresses 156 ...	350		
Patna towels 660 ...	216		
White dhoties 276 ...	208		
White vests 276 ...	594		
Red sarees 250 ...	200		
Women jackets 250 ...	150		
Other sundries ...	6,330		
Total Non-Recurring cost ...	1,76,885		

Note:—The average cost per patient per diem at the Government Tuberculosis Hospital, Royapettah, Madras including diet, medical aid, establishment etc., during 1941-'42 is Rs. 5-15-9; 1942-'43 Rs. 2-12-7.

It is essential that for any organised attempt to combat this disease we require medical personnel who had special training in this branch of medicine. For this purpose I would suggest that about half a dozen medical officers of the Corporation may be chosen to undergo a special training for a period of 6 to 9 months so that they may take up the work after training.

After the international situation improves, I think, it may be within the means of the Corporation to have a mobile X-ray unit with a mobile clinic so that the X-ray can be taken from place to place in addition to having a central X-ray institute in any of the clinics. In addition to these I would suggest that it would be advantageous for the Corporation to establish a Public Health Laboratory where facilities may be made available to all the private medical practitioners of the city to get their bacteriological examinations done free, or, if this is not possible, to levy a small nominal fee. This would greatly facilitate the work of the practitioners and at the same time ensure the co-operation of the private medical practitioners which is highly essential for the home treatment of the patients. With the meagre amount of space that is available for these open cases, for sometime to come, at the outset, many of the cases may have to be treated at their homes, but with necessary precautions. The co-operation of the private medical practitioners of the city is a most important aspect in the prevention of this disease in the city.

Preventive aspect:—The results of investigations which I have mentioned to you in the first few minutes of my talk, show that economic conditions, housing conditions and public health have much in preparing the soil for the seed. These must be improved as far as possible and at the same time special attention should be paid to nutrition.

In this connection I would like to suggest that all the people who come in contact with school children, food articles, vegetables, fruits, etc., must be periodically examined. Since the Corporation has already got full-time medical officers for their schools, it may not be too much to suggest that the teachers, nurses, midwives, etc., employed in the schools, child welfare centres and other places should also be periodically examined, because in early childhood the chance of infection is exceedingly great. I have seen very many teachers coming to the Tuberculosis Institute, Egmore, and still coming into contact with young children in the school which is a dangerous thing.

Brahmachari's Disease*

(Post-Kala-Azar Infection of the Skin by *Leishmania Donovanii*)

Its Antiquity and that of Kala-azar compared. Fallacy of Hospital Statistics in determining its incidence in any locality. Pathogenesis. Do antimony-fast Cases of Brahmachari's Disease exist? Prognosis. Future evolution of *Leishmania-Donovani* Infection in Man. Treatment.

BY

PHANINDRA NATH BRAHMACHARI, M.Sc., M.D.,
Cardiologist, Medical College Hospitals, Calcutta,

Formerly Honorary Visiting Physician, Campbell Hospital, Calcutta.

Antiquity.—The history of the introduction of kala-azar into India is shrouded in obscurity. KNOWLES (1934) considered that the disease was introduced probably from China into certain parts of Southern India from which it spread to Bengal. On the other hand, it has been suggested that the Mediterranean basin may have been the original focus of the disease and that it spread thence to India by way of Greece and the Roman

Caucasus, cases having been reported from districts which would join the two foci. Though there is no mention of the disease in the ancient works on Hindu medicine yet there is no doubt that some of the cases of enlarged spleen described by TWINING (1832) were undoubted cases of kala-azar. Thus he wrote:—

"We find that children with this disease, soon become poor, languid, weakly creatures; whose breath and exhalation from their bodies, have a nauseous, sickly odour, indicative of the unsound state of their constitutions."

"Several of the characteristics of scorbutus are present during the vascular engorgement of the spleen: there is a tendency of hæmorrhage from slight causes or injuries; leechbites and blisters are apt to ulcerate during the rainy season; and at times the slightest ulcerations are apt to slough. Foul gangrenous ulcers of the lips and gums are liable to form in consequence of slight local irritation (and often without any obvious cause), whereby the jawbones become carious, and exfoliate and the teeth fall out."

"The tumefaction of the spleen occasionally comes on very suddenly, in the course of remittent fevers, and in a few days the enlargement can be seen as well as felt extending far below the cartilages of the left false ribs. The degree of enlargement which takes place is variable; it is very common to see the spleen extending downwards on a level with the umbilicus; and laterally, from its usual situation, as far as half way between the cartilages of the ribs and navel. In extreme cases the diseased spleen fills more than half the belly, extending to the right of the navel, while its lower extremity reaches the left iliac region. Several cases of this enormous tumefaction may be seen every year in Calcutta."

"The same sort of enlargement takes place here in the spleen of adults, in consequence of various debilitating diseases, (but more after protracted remittent and intermittent fevers) which are occasionally met with at all seasons."

Comparing the antiquity of kala-azar with that of Brahmachari's disease it is evident that the latter is of much more recent origin. Though it is likely that it existed before the first case described by Sir U. N. BRAHMACHARI (1922) yet it is doubtful whether it has existed in India for such a long time as kala-azar.

Fallacy of Hospital Statistics.—The following figures have been obtained regarding cases of the disease treated in large hospitals in Calcutta other than the hospital attached to the Calcutta School of Tropical Medicine during the year 1942.

(1) Medical College Hospitals	...	Calcutta	...	No case.
(2) Presidency General Hospitals	...	Calcutta	...	No case.
(3) Carmichael Medical College	...	Calcutta	...	One case.
(4) Mayo Hospital	...	Calcutta	...	One case.
(5) Hospitals	...	Assam	...	No case.
(6) Stanley Hospital	...	Madras	...	Three cases.
(7) Govt. General Hospital	...	Madras	...	One case.

In the Calcutta Campbell Hospital and the Patna Medical College Hospital no statistics are available regarding the occurrence of the disease.

As far as my knowledge goes it is rarely met in the Calcutta Campbell Hospitals.

Calculated from the statistics from these hospitals the conclusion may be arrived at, that the disease is extremely rare in localities where the above hospitals are situated.

On the other hand, statistics from the hospital attached to the Calcutta School of Tropical Medicine show that the disease is certainly much less rare as shown from the statistics in the other large hospitals in Calcutta or Madras.

NAPIER calculated that in the Calcutta School of Tropical Medicine six hundred to seven hundred cases had been collected upto 1938.

There were 47 new cases treated in 1935, 88 in 1936, 61 in 1938, 63 in 1939, and 73 in 1940. Subsequent records show that there were 346 cases between 1936 and 1940 in the same institution. The apparent increase in the incidence of the disease is no doubt due to some extent to the greater recognition of the condition by the patient and the doctor, resulting in a better attendance of the patients for treatment at the school. The increase may also be due, to some extent, to more and more patients being affected by the disease. Which of these two factors is the predominant cause can only be determined by the careful observation of the incidence of the disease among patients who had previously suffered from kala-azar in a particular locality extending over several years. In the wards of the Carmichael Hospital for Tropical Disease attached to the Calcutta School of Tropical Medicine no cases were admitted in 1939; in 1940, 3 cases were admitted two of whom belonged to Calcutta, and one from Howrah, in 1941 two cases were admitted one from the district of Barisal and one from Faridpur (East Bengal).

NAPIER stated that the disease attacked in Bengal 5% of all cases of kala-azar and that subsequent experience suggested that this was an under-estimate.

There are no records available in the annual reports of the Calcutta School of Tropical Medicine regarding the localities from which cases came for treatment.

The above figures show the fallacy of attempting to determine the incidence of the disease from statistics collected from large Calcutta hospitals other than the hospital attached to the Calcutta School of Tropical Medicine.

It is therefore evident that more observations must be made in calculating the incidence of the disease in different parts of India. So far as can be made out upto the present time it may be tentatively stated that perhaps it is most common in Bengal, next in order comes Madras, next Bihar and United Provinces and next Assam. NAPIER and KRISHNAN (1933) consider that the disease occurs in largest numbers in Madras but I consider that there is no sufficient evidence to justify their conclusion. My view is corroborated by the fact that only four cases were treated in Stanley Hospital and Government General Hospital in Madras taken together in 1940, while 73 patients were treated in the Calcutta School of Tropical Medicine in the same year. One can explain the paucity of the disease in the Government Medical College Hospitals and the Carmichael Medical College Hospitals in Calcutta by the fact that most of the cases

that come from different parts of Bengal to Calcutta naturally go for treatment to the Calcutta School of Tropical Medicine, but it is unlikely that cases of dermal leishmaniasis from Madras would come to the Calcutta School of Tropical Medicine for their treatment. The probable conclusion to arrive at would be that the disease is more common in Bengal than in Madras.

The rarity of Brahmachari's disease before the days of antimony may have been due to the fact that in those days most of the cases of kala-azar died without any chance of dermal lesions developing after its cure. The very few cases that were cured of kala-azar might have developed Brahmachari's disease but they were probably considered to be cases of leprosy. If this view were correct then there is no need of considering the theory of host-parasite adjustment or of a virgin soil to explain the occurrence of Brahmachari's disease.

Pathogenesis.—The mobilization of the parasites of kala-azar in an individual during treatment is a most remarkable phenomenon. It is now recognised that the most common channel of infection is through the skin. From the skin the parasites are carried to the internal organs, such as the spleen, where, if they survive, they give rise to visceral leishmaniasis with all its attendant symptoms and signs. During specific treatment, due probably to the development of immune bodies, most of the patients get cured. Small number of cases do not yield to treatment, and the disease may manifest itself as resistant cases of kala-azar or in some cases again the parasites as has been pointed out by me (1942) move to the skin where they may give rise to post-kala-azar dermal lesions (Brahmachari's disease). As already stated by me (1942) this phenomenon can take place only in a disease in which the parasites reside in the reticulo-endothelial system, the cells of which are mobile and travel from the skin to the viscera and back again from the viscera to the skin.

KNOWLES (1934) considered that post-kala-azar dermal leishmaniasis was due to imperfect treatment and to this he attributed the rarity of the disease in Assam where it "had been thoroughly controlled by antimony." Others hold that the disease is rare in a virgin soil such as Assam and more common in the soil of Bengal or Madras, where the disease has existed for longer periods than in Assam. It has further been suggested that the appearance of post-kala-azar dermal lesions is indicative of host-parasite adjustment. If this is so, then it must be admitted that there is no disease other than kala-azar in which until recent times the mortality was nearly 98% and in which host-parasite adjustment has been manifesting itself within recent years in the form of a harmless local disease which may exist for indefinite periods.

Similarly there is hardly any known bacterial disease of the internal organs in which the bacteria after successful treatment of the internal organs persist in the skin, constituting a definite clinical entity.

Do Antimony-fast cases of Brahmachari's disease exist?—Such cases have sometimes been met with, but they have hardly been recorded in the literature. The earliest apparently resistant case was that of ACTON and NAPIER (1927). It was considered by them to have resisted all forms of treatment and was subsequently reproduced by MANSON-BHAR in Manson's Tropical Disease (1929) as such. Subsequently the case came under the treatment of Sir U. N. BRAHMACHARI and was completely cured

after a prolonged course of treatment with Urea-stibamine (1929). I am glad that the case was not reproduced any more as an antimony-fast case in later editions of Manson's Tropical Disease. In my opinion no case should be considered incurable and treatment should be continued for prolonged periods with the hope of getting a cure in the end.

I suggest that cases of kala-azar which show little or no improvement after 100 injections of Urea-stibamine or Neo-stibosan, may be regarded as really antimony-fast, the total quantity of the drugs being 12 grams and each dose consisting of 1 to 2 grams.

The present paper gives records of two really antimony-fast cases of Brahmachari's disease. The first was one of the nodular type and the second one of the depigmented type. The accompanying photographs show the skin lesions when the patients left treatment without being relieved.

Prognosis.—The prognosis of the disease is good so far as the life of the affected individual is concerned. It is strange that a parasite which may give rise to a fatal visceral disease does not affect the health or the longevity of the patient even when it attacks extensive areas of the skin. The prognosis of the disease varies to a great extent so far as the cure of the skin lesions is concerned. NAPIER and his co-worker (1930) were somewhat contradictory in their remarks when they stated that a very large majority of the patients treated have yielded eventually to intravenous injections of any of the antimony compounds that have been used in the treatment of kala-azar and that they may show signs of improvement, or actually be cured, after the usual course of injections. At the same time, they stated that as a general rule a much more prolonged course is necessary. MUIR and his co-worker (1930) considered that the disease yields to antimony treatment almost as readily as the visceral form. I cannot agree with these views. I agree with KNOWLES (1934) that the treatment of Brahmachari's disease is more difficult and tedious than that of kala-azar. As there are many cases which resist treatment, even after prolonged course of injection of an antimonial, it is more correct to state that while some cases yield quickly to treatment others are more resistant than cases of kala-azar generally. Cases showing slight areas of depigmentation in the skin quickly respond to treatment, as also those with small papules. Those rare cases that do not give any history of a previous course of antimonial treatment, more quickly yield to it. On the other hand, cases are not infrequently met with in which depigmentation of the skin persists after 50 or 60 injections or even more of an antimonial such as Urea stibamine or Neo-stibosan. It may be generally stated that while slight and early depigmentation may easily yield to treatment extensive areas of well-marked depigmentation may take a very considerable time to disappear and sometimes nodules or papules may disappear, but depigmentation may still persist even after prolonged treatment.

It may be stated as a general rule that the earliest cases yield most quickly to treatment.

Future evolution of Leishmania-Donovani infection in man.—The following changes may take place in the future evolution of the parasite of kala-azar:

When the parasite attacked man in the earliest stages of its evolution the disease was a visceral one, probably after a transient local lesion.

Brahmachari's Disease.—By Phanindra Nath Brahmachari.

Antimony-fast Cases of Brahmachari's Disease



Fig. 1.



In course of time this visceral localization has been changing and the disease has been more and more manifesting itself by dermal lesions especially after antimony treatment. It is possible that the dermal lesions will increase in course of time and visceral localization diminish, till finally *Leishmania Donovanii* infection of man will more or less manifest itself as Brahmachari's disease, and kala-azar will be rare in course of time. Specific drugs perhaps would help in this evolution of the parasites as specially during specific treatment do the parasites tend to move to the skin with the help of cells of the reticulo-endothelial system and other leucocytes. This is perhaps happening to-day since the introduction of antimonial treatment. A similar view was also expressed by KNOWLES (1934). He considered that in many parts of Madras *Leishmania-Donovani* infection has been in the present day manifesting itself mostly in the form of dermal leishmaniasis and less of kala-azar. He held that the epidemic waves of kala-azar of by-gone times in the Madras Presidency have left here scattered foci of the disease representing the last members of the infection together with a large residue of cases of dermal leishmaniasis.

From this one is likely to conclude that Brahmachari's Disease is a later manifestation of *Leishmania-Donovani* infection in man than kala-azar. Whether kala-azar would be more or less replaced by Brahmachari's disease at some future period, however distant it may be, I am unable to say. Perhaps the parasites may again revert to their original life cycle in man due to certain unknown factors and give rise to visceral leishmaniasis which may be sometimes of an epidemic character.

Probably the evolutionary changes in the parasite are not progressively the same in the different parts of the world, and therefore though the disease may be more ancient in China or the Mediterranean basin than in India yet dermal lesions are scarcely met with in the former places at the present time.

Can it be assumed by analogy that oriental sore which is caused by *Leishmania tropica* might have originated as a visceral lesion. This conclusion is justified by the fact that the virus of *Leishmania tropica* may give rise to a visceral infection pointing out, as it were, to its reversion to its primitive life cycle if injected into a susceptible animal intravenously.

Treatment.—The treatment of Brahmachari's disease is unsatisfactory in the sense that, frequently, it has to be a prolonged one. The following lines of treatment have been adopted by me:—

(1) Intravenous injection of Urea-stibamine or Neo-stibosan; (2) intravenous injection of Sodium Antimonyl Tartrate or Tartar Emetic; (3) intravenous injection of a pentavalent antimonial together with inunction of metallic antimony in a state of fine sub-division; (4) intramuscular injection of Urea-stibamine or Neo-stibosan or Berberine Sulph or the antimony analogue of tryparsamide, or Sodium Sulphomethyl stibanilate or Neo-stibosan; (5) combination of the above treatments; and (6) combined treatment with antimonial and arsenical preparations.

The combined treatment of introducing antimony into the system by different routes, e.g., (1) intravenous, (2) intramuscular, (3) inunction seems so far to have given the best results. In some cases the alternate administration of trivalent and pentavalent antimonials seem to be attended with somewhat quicker results.

Inunction of metallic antimony in a state of finest sub-division over the skin lesions would enable antimony to come in contact with the leishmania most effectively and quickly, as it has been observed by me that antimony administered in the form of ointment is absorbed through the skin and can be subsequently detected in the urine.

Apart from the fact that Brahmachari's Disease may be a possible carrier of kala-azar, it is very necessary that its treatment should be improved so that the sufferers may be saved from the misery of an ugly disfiguration which may be mistaken for leprosy and is most disagreeable to himself, his friends and relations, as well as to the public with whom he may come in contact.

In many cases Neo-stibosan was used in the beginning of the treatment of the disease but as the result was unsatisfactory in some cases it was replaced by Urea-stibamine which was found to be more effective.

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Indian Village Obstetric Emergencies

BY

F. R. W. KINKEAD ALLEN,

Master in the Art of Obstetrics and Doctor in Medicine of the University of Dublin.

(Continued from page 368 of June 1944 issue.)

CHAPTER V—(contd.)

Delay in 2nd stage i.e., external "os" fully dilated—(contd.)

XIV. In cases of phthisis, cardiac decompensation, debilitating fevers, exhaustion etc., in cephalic presentations forceps should be applied or in breech presentations extraction should be performed if the interests of the mother demand it.

XV. In cephalic presentations the discharge of meconium-tinged amniotic fluid when the parietal bosses have passed the ischial spines is an indication for the application of forceps.

XVI. Changes in the foetal heart rhythm or the foetal heart rate exceeding 160 or dropping below 100 beats a minute calls for forceps delivery in a cephalic presentation when the parietal bosses have passed the ischial spines or for extraction in a breech presentation.

XVII. In the circumstances mentioned in XIV, XV and XVI if the parietal bosses in a cephalic presentation are above the ischial spines the alternatives are axis traction or Saxtorph's manoeuvre with forceps or, provided the integrity of the uterus is not endangered, version and extraction.

XVIII. If the head is floating, in the absence of serious disproportion, delivery can usually be effected more safely and rapidly by version.

XIX. In the absence of forceps and destructive instruments, when out of reach of a hospital, version may be the only alternative to death for both mother and child. In such an event rupture of the uterus would have to be risked. With the head low in the pelvis intra-nasal pituitrin as detailed in Chapter III, 20, or injections of pituitrin as detailed in maxim 11 at the beginning of this chapter would have to be tried.

XX. Do not forget that it may be possible to obtain a live child by post-mortem Caesarian section upto 15 minutes after the death of the mother and that any kind of a knife will do. Do not cut the cord until it ceases to pulsate and do not omit resuscitation for the child even it appears to be dead. Mouth to mouth breathing is best after clearing the infant's throat of mucous and liquor.

CHAPTER VI.

3rd Stage

I. Maxims.—1. Delay is usually due to one or more of the following:—

- (a) Handling, rubbing, squeezing the uterus by the conductor.
- (b) The woman's bladder being full.
- (c) The woman and/or her uterus being exhausted.

Rarer causes:

- (d) Rupture of the uterus.
- (e) Adherent placenta.

2. By "P.P.H." is meant any abnormal loss of blood after the birth of the child (a loss of over 500 c.c.m. (7/8 pint) is abnormal), or any loss of blood which produces or is likely to produce symptoms, i.e., the pulse and respiration rates increase, the face becomes drawn and ashy, the mucous membranes lose their pink colour, the woman feels chilly and becomes restless, while in serious cases the temperature falls, the woman experiences air hunger and a cold sweat or becomes unconscious. A large full blooded woman can afford to lose more blood than a small anæmic woman.

The bleeding is usually external but may occasionally be concealed in the uterus, broad ligament or peritoneal cavity.

3. It is important to control "P.P.H." before the onset of shock.

4. As a rule, time taken in giving an injection of Morphine, washing up properly and, if assistance is available, commencing an anæsthetic is seldom wasted. If blood or serum is not available for transfusion the best use must be made of intravenous glucose or subcutaneous normal saline.

5. Shock alone, without antecedent hæmorrhage, is rare and is only likely to follow premature and injudicious efforts at expression of the placenta, manual removal of the placenta, rupture of or inversion of the uterus, prolonged labour, damage to pelvic floor or fulminating infection.

6. Once shock has appeared a very fine judgment is required as to whether there is a concealed hæmorrhage.

7. If shock alone is present perform no manipulations for expression or removal of the placenta. Give Morphine, tea, glucose, transfusions or infusions [see Chapter I] and wait for 24 hours at least before attempting removal of the placenta.

II. **Simple delay.**—1. Provided there is no "P.P.H." or shock, delay within reason does not matter.

Do not rub, squeeze or handle the uterus.

1/4 grain of Morphine will be more effective and less harmful.

2. An hour after the birth of the child get the woman to kneel or squat and endeavour to urinate. This will probably bring out the placenta. If it fails pass a catheter and give 1/4 grain Morphine.

3. When the fundus of the uterus has risen to or above the umbilicus or is found at this level—

(a) If the uterus is well contracted the placenta is in the lower segment or vagina.

(b) If the uterus is large and soft it may contain a second foetus (see Chapter V (IX) 4 or blood clots in addition to the placenta (see (III) (A) (2) (b) below).

(c) Having diagnosed the placenta in the lower segment or vagina either make the woman use her abdominal muscles to express it or make her squat up so that gravity may bring it out. If both fail take the fundus of the uterus in your right hand, thumb on the anterior surface and fingers on the posterior surface and, without massaging or squeezing, push downwards and backwards with the uterus in the direction of the pelvic inlet to push the placenta down the vagina to the vulva.

(d) If the uterus has not risen to the umbilicus it shows that the placenta is still in the body of the uterus. (In rare cases of rupture it may be in the abdominal cavity or broad ligament). If you desire to avoid the risk of shock and sepsis, wait at least 24 hours before resorting to expression or manual removal. You must not attempt to express a placenta which is in the abdominal cavity or broad ligament [see (8) below].

4. When the placenta is delivered examine it carefully to see that no portion or succenturiate lobe has been left behind. Such a portion or lobe must be removed at once under full antiseptic precautions [see (8) below].

A portion of membranes left behind will be cast off in the lochia.

5. For an hour following the birth of the placenta palpate the uterus gently occasionally and watch the pulse rate.

(a) If the pulse rate increases and the uterus becomes flabby give 0.5 mgm. Ergometrine into the uterine muscle through the abdominal wall and rub up the uterus to contract and express clots. The Ergometrine can be reinforced by 2 to 5 oxytocic units of posterior lobe Pituitary extract given into the uterine muscle.

(b) If the uterus remains well contracted do not maul it about, i.e., no squeezing or rubbing. There is no need to give an Ergot preparation as a routine.

(c) If the woman desires to wear a binder apply one.

6. If for any reason it appears undesirable to wait 24 hours for the

separation of the placenta and in any case before you try expression or manual removal, inject 500 c.c. of Sterile Glycerine, Saline or of a fairly strong solution of Glucose or of Sodium or Magnesium Sulphate up the umbilical vein of the placenta and clamp the vein.

This may help the expulsion of the placenta into the lower segment.

7. 24 hours or so, after the birth of the child, if the injection of saline up the umbilical vein has failed, give your patient a 1/4 grain of Morphine "S.C." and if she is unable to empty her bladder, pass a catheter. If this is not followed by expulsion of the placenta, provided you are sure the uterus is not ruptured nor the systolic "B.P." below about 100 mm. Hg.—

Dublin method of expressing the placenta:

(a) Administer an anæsthetic if possible.

(b) Seize the fundus in your right hand and with your thumb on the anterior surface and your fingers on the posterior surface rub the uterus up to a good contraction and squeeze the body of the uterus in your hand to force the placenta out into the lower segment.

(c) Then use the uterus as explained in (II) (3) (C) above to push the placenta down to the vulva.

This method of expression is liable to cause great shock and if it fails do not persist but perform manual removal.

8. *Manual removal* is indicated if the systolic blood pressure is under 100 mm. Hg., if the uterus may be ruptured or if the Dublin method has failed.

(a) Prepare the woman as described in Chapter I.

(b) If possible administer an anæsthetic.

(c) Hold the fundus with your right hand. Pass your left hand into the uterus.

(d) If the placenta is in the uterus work your fingers between the placenta and the wall of the uterus. Make sure you leave no portion or succenturiate lobe behind. Make sure there is no rupture of the wall of the uterus. Remove the placenta and your hand slowly to give the uterus time to contract down and thus avoid unnecessary hæmorrhage.

(e) While removing the placenta if you find that there is a rupture of the uterus the rent should be plugged.

(i) Take a large handful of sterile Sulphathiazol impregnated gauze (or boil up strips of clean sari in weak aqueous "Dettol," "Cyllin," or Carbolic Acid solution, allow cooling off to 118°F. and wring out).

(ii) Steady the fundus, with your right hand, through the abdominal wall.

(iii) With your left hand carry the hand full of plugging material to the rent in the uterus and plug it moderately tightly. Leave the tail or tails of the plugging material in the vagina.

(iv) Perform a posterior colpotomy by drawing the cervix downwards and forwards with bullet forceps, with scissors cut the posterior fornix transversely close up against the cervix, widen the incisions by opening the scissors, by blunt dissection with the scissors or a finger

covered with gauze, separate the rectum from the cervix, snip forwards keeping close to the posterior wall of cervix without actually cutting into it and you will open into the peritoneal cavity, widen the opening with your finger. Insert a gauze drain.

(f) If the placenta is not in the uterine cavity it must have escaped through a tear into the broad ligament, which is unlikely, or into the abdominal cavity, according to the situation of the rupture. You have now to decide:

(i) Whether you can get the woman to hospital, which is the best course if practicable;

(ii) Whether you will open her abdomen as described in Chapter III (19) (A) (3); or

(iii) If the placenta is in the broad ligament whether you will pull it back by traction on the cord and increase the size of the tear; or

(iv) If the placenta is in the abdominal cavity whether you will remove it through the colpotomy incision.

(v) In the first two cases cut off the cord as high up as possible, plug the rent and in the second in addition do a posterior colpotomy with insertion of a drain as described in (e) above.

(vi) In the third case after removal of the placenta plug the rent.

(vii) In the fourth case after cutting off the cord as high up as possible plug the rent and make the colpotomy incision as described in (e). Widen the colpotomy incision by stretching with your fingers until you can pass in your hand. Squeeze the placenta by any portion, but do not mistake bowel or omentum for placenta, pull out the placenta. Push back any bowel or omentum which prolapses. Insert a loose plug to prevent prolapse of bowel or omentum and to serve as a drain in the colpotomy wound.

(viii) If necessary, repair the perineum and treat every case as being potentially septic by giving one gramme of Sulphathiazol and an alkaline mixture every four hours. Do not forget light nourishing food, sweetened fruit drinks, honey or sugar-cane and at night opium if necessary to secure sleep. If the woman is dying do not spare the opium by day as well as by night.

(ix) Next day loosen the uterine and colpotomy plugs by gently pulling a little on the tails in the vagina. Repeat this daily until they can be removed entirely about the fifth day.

III. "P.P.H."—(A). Before the placenta is born:

1. Causes.

(a) Tears of the birth canal.

(b) Partial separation of the placenta.

(c) Separated but retained placenta.

2. (a) *Tears*:—Especially after placenta prævia, versions, instrumental deliveries or other abnormalities, when the bleeding commences immediately following the birth of the child and the uterus is found to be firmly contracted a tear of the birth canal is the probable cause of the "P.P.H."

In cases where the uterus can be made to contract after massage, if there is no diminution in the flow of blood, the cause is most likely to be a tear.

(i) Bring the patient to the edge of the bed. A good light is essential.

(ii) Ensure that your hands and the vulva are sterile.

(iii) Pass a catheter.

(iv) Inspect the external genitalia. If the perineum, labia and clitoris are not the source have an attendant press upon the fundus to bring the cervix into view. If fundal pressure is insufficient employ a speculum and bullet forceps to draw down the cervix.

(v) If the source of the bleeding is not the cervix separate and inspect the vaginal walls.

(vi) By the time you have done all the above the placenta will in most cases have been expelled into the lower segment or vagina. If it has not use your right hand to express it as described in II, 7, above, provided the systolic "B.P." is over 100 mm. Hg.

(vii) If this fails or if the systolic "B.P." is under 100 mm. Hg. remove it with your left hand as described in (II), (8), above.

If you have found no source of bleeding so far examine the interior of the uterus for a rupture [see (ix) below].

(viii) Repair the tears.

Remember in the perineum the levatores ani and the deep and superficial transverse perinei and the bulbo-cavernosi muscles must be brought accurately together by deep and superficial stitches. The deep stitches must not pass into the rectum. If the sphincter ani is torn it too must be repaired.

Remember that when the cervix is torn the muscle has retracted between the two layers of the mucous membrane and that the stitches must draw muscle layer to muscle layer.

If unable to repair the cervix apply ring forceps for 24 hours. As a temporary measure other tears may be plugged with sterile material.

Dress the wounds with gauze soaked in 1 in 1,000 neutral salt of Proflavine or Acriflavine in Glycerine for rapid healing [see (B) (2), (a) (ix) below].

(ix) The uterus may be ruptured accompanied by concealed hæmorrhage into the abdominal cavity or into the broad ligament, with or without revealed vaginal "P.P.H."

(x) The classical signs and symptoms of ruptured uterus were given in Chapter III (19) but rupture is probably more common than diagnosed because the signs may be slight and the only symptom may be shock.

(xi) Bleeding into the broad ligament may stop naturally. By passing an aspirating needle (in the correct direction parallel to the uterus) through the posterior fornix intra-abdominal bleeding can be diagnosed.

(xii) The best treatment for a rupture is hysterectomy or occasionally, repair of the rent, provided the hæmorrhage can be controlled and the patient got over her shock and to hospital.

(xiii) According to its degree, shock should be treated while plugging materials are being got ready for treatment as described in (II) (8) above before removal of the patient to hospital.

(b) *Partial separation of the placenta* is liable to precede or accompany placenta prævia or to follow too rapid emptying of the uterus (especially when originally over distended), prolonged labour in which the patient and/or her uterus become exhausted, traction upon the cord, premature attempts at expression of the placenta, meddlesome massage of the uterus or the misuse of the Ergot.

(c) *Retention of the placenta* may be brought about by the same agencies, the uterus being either exhausted and unable to expel the separated placenta or a constriction ring (hour glass contraction) holding up the placenta.

(x) *Moderate hæmorrhage*:—If blood oozes steadily from the vagina or you notice the woman commences to show signs of blood loss and the uterus filling up with blood—

(i) Seize the fundus of the uterus in your right hand and rub up a good contraction. (If your left hand is sterile keep it so).

(ii) Express the placenta as described in (II) (7) above.

(iii) If this fails, while continuing to massage the fundus, try and compress the aorta, with the ulnar boarder of your right hand, against the vertebral column.

(iv) Get an attendant to raise the foot of the bed on bricks, blocks, stones or anything handy.

(v) If the child is alive and able to suck have it put to the breast or get some other child or relative to oblige.

(vi) Get an attendant to hold the fundus and tell you when it relaxes.

(vii) If your left hand is contaminated give the patient 1/4 grain Morphine before you wash up.

(viii) Empty the woman's bladder with a catheter.

This may be followed by extrusion of the placenta into the lower segment or vagina. If not—

(ix) If possible, have the woman anæsthetised.

(x) Perform manual removal as described in (II) (8) above and massage the fundus of the uterus between your external and internal hands. Withdraw your internal hand slowly as the uterus contracts down upon it. [See (B) (2) (a) (ix) below].

(xi) If you find a constriction ring below the placenta, grip the fundus with your right hand and making the fingers of your left hand into a cone endeavour to dilate the constriction by forcing the cone into it. If this fails get an attendant to crush an ampoule of Amyl Nitrite under the patient's nose or to give her "I.M." 1/3rd ccm. of 1/1000 Adrenalin solution. [It appears possible that an injection of 100 mgm. Pethidine or of an ampoule of Trasentin 6 H. (Ciba) may be preferable, but so far precise reports are lacking and the drugs are not yet available in India]. When relaxation occurs dilate the constriction and remove the placenta.

It may be difficult or impossible to dilate a constriction ring quickly unless the patient is deeply anæsthetised. [See (B) (2) (a) (ix) below].

(y) *Severe hæmorrhage continuing*:—If the blood commences to pour out of the vagina in large quantities or the woman collapses and becomes almost pulseless do not hesitate but perform manual removal immediately as described in (II) (8) above. Then inject 2 to 5 oxytocic units of posterior lobe pituitary extract and 0.5 mgm. Ergometrine into the uterine muscle if the latter does not contract rapidly. [See (B) (2) (b) below and (C) below].

(z) *Severe hæmorrhage which has stopped or nearly stopped and left the woman collapsed*:—You may be called to a case which has had a considerable hæmorrhage as shown by the blood in the bed or on the floor. By the time of your arrival the bleeding may have stopped or be reduced to slight oozing.

Treat such a case for shock and hæmorrhage first [see (C) below] but do not give drugs to raise the "B.P." 24 or more hours later when the woman recovers from the shock, or sooner if bleeding recommences, if the placenta has not reached to vagina, after careful preparation against sepsis, remove the placenta manually and be on the watch for puerperal sepsis. Do not employ the Dublin Method of expressing the placenta in such a case.

(B) *After the placenta has been delivered*—

1. Causes:

(a) Tears of the birth canal.

(b) Atony of the uterus.

(c) A portion of the placenta or a succenturiate lobe retained.

2. (a) *Tears* except for rupture of the uterus or in cases where the placenta is expelled with great rapidity, are likely to cause bleeding before as well as after delivery of the placenta. The treatment is as described in (A) (2) (a) above.

In "rupture of the uterus" possibly there may be prolapse of the bowel or omentum.

(i) Cover the prolapsus, if any, by a clean cloth wrung out in weak aqueous solution of "Dettol" "Cyllin" or Carbolic Acid.

(ii) Treat the woman for shock.

(iii) If there is a prolapsus boil up swabs in normal saline and let them cool off to 100°F.

(iv) If you have no sterile Sulphathiazol impregnated gauze boil up strips of clean sari in weak aqueous "Dettol" "Cyllin" or Carbolic Acid solution. Allow cooling off to 118°F.

(v) Prepare the perineum and vulva but do not douche the vagina.

(vi) Cleanse the prolapsus with warm, previously boiled, normal Saline at 100°F.

(vii) Anæsthetise the woman.

(viii) Repose the prolapsus through the rent in the uterus and plug the latter as described in (II) (8) (e) above. If you use the sari strips or other recently boiled material have it 118°F.

(ix) Repair the perineum and treat every case as being potentially septic. Give an alkaline diuretic mixture and one gramme of Sulphathiazol every four hours during the day *i.e.*, five doses. Give also light nourishing food, sweetened fruit drinks, honey and sugarcane juice. Give opium, chloral and bromide at night to secure sleep. If the woman is dying do not spare the opium by day as well as by night.

(b) *Atony of the uterus* :—The uterus is large and soft. On being rubbed up clots of blood are extruded when the uterus can be got to respond by contracting.

(i) Have a live baby capable of sucking vigorously put to the patient's breasts or get a relative to oblige.

(ii) Have the foot of the bed raised on bricks, stones or stools.

(iii) An attendant must be made to knead the uterus while you inject 2 to 5 oxytocic units of Post. Lobe Pituitary Extract and 0.5 mgm. Ergometrine into the uterine muscle through the abdominal wall.

(iv) If the uterus still fails to contract or remain contracted pull the fundus sharply forward over the pubes and get an attendant to hold it there while you get the materials ready for—

(v) Plugging or packing the uterus. Everything must be sterile. Before commencing to pack explore the cavity of the uterus for a piece of placenta. During the packing either you must hold the fundus in your right hand or an attendant must hold it to ensure that the packing material reaches right and tight up to the fundus. The best material to use is sterile Sulphathiazol impregnated gauze. If you have none boil thin strips of sari or pugarie in a weak aqueous solution of "Dettol" or "Cylin" and allow them to cool down to 118°F.

(vi) Catheterise the patient.

(vii) If you do not possess a Holmes' uterine packer pull down the anterior lip of the cervix by means of a bullet forceps and under direct vision feed in the strips of packing material right up to the fundus, which must be tightly packed while it is held by an attendant.

(viii) Count how many strips you use and leave the tail of each strip low down in the vagina. Remove the strips by pulling on the tails in 6 to 24 hours. Plugging properly performed stops hæmorrhage.

(ix) If for any reason when plugging is indicated, you are unable to plug, provided there is no rupture give a long hot intra-uterine douche. Use 4000 to 5000 ccm. of 2% "Dettol" or 1% "Cylin" or Carbolic Acid in water (4 to 5 litres = roughly one gallon) at a temperature of 116°F. In the absence of a rubber douche nozzle I consider a stomach tube or flatus tube unlikely to perforate the uterus and advise their use in preference to Bozemann's largest size or other glass or metal douche nozzle. See that there is a free return space for the fluid. You should not force fluid through the fallopian tubes. Have an attendant hold the fundus or do so yourself to ensure the douche nozzle reaching there. [See (B) (2) (a) (ix) above].

(c) *A small portion of placenta or a succenturiate lobe retained* :—It must be removed by your left hand and then withdraw the hand slowly as the uterus contracts down upon it kneading the uterus with the "abdominal" right hand simultaneously. [See (II) (8) above].

(C) The woman is by now both *exsanguinated* and *shocked* :—

(a) Raise the foot of the bed and give Omnopon, Morphine or Opium.

(b) If the weather is warm give her a drink of not too warm sweet tea. If the weather is hot, cool the sweet tea by adding cold water.

If the weather is cold wrap her trunk up well with blankets and if it is very cold place a hot water bottle in each axilla *but not* next to the patient's skin *or she will be burnt*. Give plenty of warm sweet tea.

(c) Arrange for a blood transfusion or failing that for a serum or plasma transfusion if possible.

(d) Give 20 to 40 ccm. of 50% Glucose solution "I.V."

(e) Commence an intravenous drip injection of Normal Saline at 20 to 30 drops a minute and continue until you have given about 2 pints (— 1,000 ccm.).

If the weather is cold HAMILTON BAILEY has pointed out that it is no good warming the blood or saline container. Warm the recipient. Every half hour fill a 20 ccm. syringe with 50% Glucose solution and passing the needle into the lumen of the rubber tubing, by which the saline is travelling to the vein, add the Glucose solution to the saline slowly.

If you cannot find a vein in the arm remember the marrow of the manubrium sterni will absorb fluid almost as quickly, but avoid perforating both plates of the manubrium or the fluid will enter the mediastinum. Or there is the vein just anterior to the medial malleolus. If you are unable to utilize the sternum or if you cannot find any vein give the Saline subcutaneously or rectally by Murphy's method but in this case do not add the glucose to the Saline. Give the glucose by mouth.

(f) Apply a broad tight abdominal binder and bandage the lower limbs commencing at the toes and working towards the trunk.

IV. Inversion of the uterus may be partial or complete :—

1. Cover the uterus with cloths wrung out in hot weak "Dettol" "Cylin" or Carbolic Acid solution.

2. Treat the shock as detailed in (III) (C) above. If there has been no bleeding only a small transfusion or infusion will be required but plenty of glucose.

3. There is great danger of sepsis. Commence Prophylactic Sulphathiazol. [See III (B) (2) (a) (ix) above].

4. If the woman survives the hæmorrhage, shock and sepsis she will have to be sent to hospital for operation at a later date.

V. Shock alone—see Chapter I, 25.

VI. Pulmonary embolism is very rare. Prop the woman up in the fresh air and allow her to breathe freely. Give oxygen by means of an intranasal catheter or mask if available.

VII. Do not massage or permit massage of the uterus in the puerperium. Make the woman lie on her face as much as possible. Whether the child is alive or dead, if the breasts become engorged and painful

and/or the uterus unvolutes badly give 5.0 mgm. Stilboestral daily. This will not interfere with lactation.

Unless the genital tract has been plugged or stitched get the woman sitting upright the following day and out of bed in a couple of days for short periods.

IX. Severe secondary P.P.H. coming on after several days is due to a portion of retained placenta, which must be removed, or to retained membranes or massage of the uterus, in which case the uterine cavity must be plugged for 12 to 24 hours with sterile material Ergometrine "I.M." and Pituitary Extract fail to control the bleeding.

CHAPTER VII

Hæmorrhages via the Vagina

Most cases are more easily treated in hospital.

A. 1st Trimester: (1) Threatened or incomplete abortion. (2) Ectopic pregnancy (must go to hospital). (3) Hydatidiform mole. (4) Injuries or neoplasms. (5) Erosions.

B. 2nd Trimester: (1) Miscarriage with retention of placental parts. (2) Ectopic pregnancy (must go to hospital). (3) Hydatidiform mole (1st few weeks). (4) Placenta prævia (later weeks). (5) Injuries or new growth. (6) Erosions.

C. 3rd Trimester: (1) Placenta prævia. (2) Abruptio placentæ. (3) Injuries or neoplasms. (4) Ectopic pregnancy (must go to hospital). (5) Spontaneous rupture of the uterus. [see Chapter III, 19, iii]. (6) Erosions.

I. In abortion or miscarriage there is usually colicky pain in the lower abdomen, backache and frequency of micturition. A vaginal examination must be made.

1. Treat by rest in bed, Omnopon or Opium. If the patient is costive give gentle enemata. If you diagnose the abortion as only threatened, inject "I.M." 10 milligrammes Progesterone and repeat 2 mgms. daily.

2. If the hæmorrhage is sufficient to produce symptoms repeated $\frac{1}{2}$ uterine emptying unit "I.M." doses of Post. Lobe Pituitary Extract may assist the Ergot and "I.M." injections of 5 to 10 mgms. Oestradiol every 4 hours.

3. If it fails to evacuate itself, the uterus must be emptied, taking all antiseptic precautions. A finger of the left hand peels the products of conceptions from the uterine wall while the right hand on the abdomen holds the uterus. A ring forceps may be used for removing the "debris," but a finger must ensure that the cavity is finally empty. A wick of sterile impregnated gauze leading from the fundus aids contraction if oozing is troublesome. This gauze should be removed after 12 to 24 hours.

4. If the cervix will not admit a finger tightly plug the vagina, especially the fornices and external "os," for 24 hours with impregnated sterile gauze or balls of wool boiled in a weak aqueous solution of 'Dettol,' Carbolic Acid or 'Cyllin.'

5. If you suspect criminal interference or if the case is febrile, unless the hæmorrhage threatens the woman's life, do not evacuate the uterus until the temperature has been normal for at least 72 hours and you have

started the woman on prophylactic Sulphonamide or Penicillin and given Tetanus and Gas Gangrene antitoxin in sufficient quantity (Chapter I, 19). Meantime treat as explained in (2) above and treat the patient's shock and hæmorrhage. [Chapter VI, (III) (C)].

6. When the uterus is empty, if hæmorrhage continues, Ergometrine should be given and it may be reinforced by "I. M." injections of Posterior Lobe Pituitary Extract.

II. Vesicular or hydatidiform mole produces vague abdominal discomfort due to the over rapid growth of the uterus. The foetal parts cannot be felt and the foetal heart cannot be heard. Small white vesicles may be found in the vaginal bleeding.

Treat by evacuating the uterus as described above.

III. Abruptio (ablatio) placentæ or separation of the normally implanted placenta is usually accompanied by severe hypogastric colic, shock, rapid pulse, pallor, restlessness, collapse and air hunger. The uterus becomes hard and tender while the foetal heart sounds and movements disappear. If shock is slight the "B.P." may be above normal.

1. Vaginal bleeding only occurs in the milder types which are luckily the more common.

2. When hæmorrhage is concealed the condition has to be differentiated from *Acute Hydramnios*, in which pallor and air hunger are absent and in which the uterus is tense rather than hard and a tense bag of waters may be found on vaginal examination. In acute Hydramnios relief is obtained by rupturing the membranes high up and only permitting slow drainage or by paracentesis through the abdominal and uterine walls.

Treatment.—(1) Ensure that the bladder and lower bowel are empty.

(2) A tight abdominal binder helps to compress the uterus on the placental site. Better still employ a sheet or sari and by means of two lathis twist and thus tighten down the sari to constrict the patient's abdomen against the bed, or if she is being sent to hospital, against the stretcher. When twisted tight the lathis must be bound to the side of the bed or stretcher to prevent them unwinding.

(3) In mild cases rupture the membranes and, if labour is slow in starting, apply intranasal Post. Lobe Pituitary Extract, give oral Quinine and "I.M." 10 mgms. of Oestradiol as described in Chapter III, 20 (g).

(4) In more severe cases, especially if there is albumen in the urine, in addition to the above endeavour to obtain a blood or serum transfusion and treat the shock [Chapter VI, II, 6] and weak pains as described in Chapter IV, 1. Such patients require ample Morphine and Glucose and, if blood or serum is not available, Saline infusion.

(5) If the cervix does not dilate proceed as described in Chapter IV, (III), (C).

(6) Plugging the vagina tends to aggravate shock. It is therefore a last resort. If done at all it must be done properly, with antiseptic materials and preparations, or it is worse than useless.

(7) Be prepared to deal with P.P.H. and have sterile impregnated gauze or material boiled in weak aqueous "Dettol," "Cyllin," or Carbolic

Acid solution ready to pack tightly the uterine cavity if the usual methods fail.

(8) See Chapter I, 7, 24, 25, and 28, and Chapter VI, (III).

IV. **Placenta prævia** occurs in the second half of pregnancy. Painless bright red vaginal bleeding coming on (a) without apparent cause and (b) without albumen in the urine is due to placenta prævia.

1. Never perform a vaginal examination on a suspected case of placenta prævia until everything is ready for the application of Willett's forceps or version and if possible a blood transfusion. Always treat shock before performing examinations or manipulations unless continuation of the bleeding compels immediate action.

2. It is much easier to deal with a case of placenta prævia in hospital but the patient must be watched *en route* for fear of a recurrence of the hæmorrhage. In the ambulance the patient should wear a tight abdominal binder or be tied down to the stretcher as explained in (III) (5) above. If she commences to bleed Post. Lobe Pituitary Extract should be injected "I.M." half unit by half unit waiting 20 minutes between each injection to avoid producing tonic uterine contraction. If available apply a pad of sterile dressing to the vulva and keep it in position with a T-bandage.

3. Mild cases with slight bleeding should receive the treatment for threatened abortion [See (I) (1) above]. The bleeding may cease for a time but it is liable to recur with greater intensity. Hospital is the safest place.

4. If the bleeding is too severe to permit of the patient being sent to hospital or if no hospital is available apply a tight abdominal binder, give 10 grains of Quinine by mouth, inject "I.M." 10 milligrammes Oestradiol and get everything ready and the patient prepared as rapidly as possible.

(a) Rupture the membranes. Apply Post. Lobe Pituitary Extract as described in Chapter III, (20) (g). In cases of partial placenta prævia this may suffice especially if a tight binder is skilfully applied.

(b) Where the placenta is situated centrally or covers a large portion of the external "os" it will probably be necessary, in addition to the above, to perforate the placenta or push it aside and if the presentation is a vertex with the occiput anterior or transverse without disproportion to apply Willett's forceps to the child's scalp or to perform Braxton Hick's version pull out an ankle until the knee appears outside the vulva. In case of a breech presentation or of a transverse presentation pull out an ankle until the knee is outside [See Chapter V, (IV and V)].

(c) To the handle of the Willett's forceps or to the ankle attach, by means of a cord passing over the foot of the bed, a weight, just sufficient to keep light traction operating but not sufficient to damage the child, usually a weight of about one seer is sufficient. If contractions are poor and the child is small inject "I.M." Post. Lobe Pituitary Extract half unit by half unit watching the result between each injection to avoid tonic contraction.

(d) Do not try extraction before the external "os" is fully dilated or the cervix will be badly torn.

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80	4.8
70	4.6
60	4.4
50	4.2
40	4.0

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Immunology in Immunity

Immunology is a branch of science which involves the study of reactions through which animal being might develop an immunity to resist infection by micro-organisms that cause disease. The study of the mechanism of immunity was just in its beginning towards the end of the last century when it was a wonder to find soluble poisons in fluids in which organism of Diphtheria and Tetanus had been artificially cultivated. With the advancement of serology it became apparent that the animals could be rendered immune to such a poison and that the blood serum of such animals developed a substance or substances that might combine specifically with the poison in question and render it innocuous. The nature of this immunizing process is still obscure. But it has been amply demonstrated that the injection of serum of animals rendered immune, saves human beings from the attacks of the toxins liberated by those micro organisms.

This aspect of the newly developed science was not overlooked, and soon organisation grew up for producing the various immune sera on industrial scale for ameliorating the conditions of the suffering public in general. The physicians all over the country now realise the importance of the local productions of various remedies for passive immunity. Active immunisation, however, is the

process by which the antigen (bacteria and bacterial toxins) can be directly introduced into the living tissues with the object of inducing the formation of specific antibodies. This requires a perfect knowledge and a command on the whole technique that are associated with the science of immunology. These might involve amongst other things an idea of action of lysins, precipitins and opsonins, a knowledge on the different chemical reactions and the conception of the changes caused by some surface phenomena. A conquest in all these directions would only permit a manufacturing concern to offer products—the toxoid—for active immunisation.

In order to serve the humanity then and that again at all times by offering sera and toxoids in quality and quantity, a national out-look, a public spirit and a desire for sacrifice amongst capitalists and the labourers of the concern are absolutely essential. Being handicapped in divergent ways the idea seems to be a utopian one in India. But a strong determination may create success as has already been done in the laboratories of the Bengal Immunity for development of Science of Immunology, and thereby offering to the physicians of the various sera and toxoids as their armaments to fight against dreadful diseases.

(e) In case of a recurrence of the bleeding or of dystocia substitute the cranioclast for the Willett's forceps when the size of the external "os" admits and be ready for cleidotomy and embroyotomy. If you have drawn out an ankle the order of the above operations will be reversed.

(f) Do not plug the vagina to control the haemorrhage except when the "os" is too small to permit of version or of the application of Willett's forceps. It is worse to plug the vagina, with unsterile material, dirtily and badly than not to plug it at all.

(g) Be ready for P. P. H. and puerperal sepsis.

(h) Endeavour to procure a blood or serum transfusion and give intravenous Glucose and subcutaneous Morphine for shock [Chapter VI, (II), (6)].

(i) It may be necessary to plug the uterus after delivery is over if other methods fail to control haemorrhage from the placental site as described in [Chapter VI, (III) (B) (2), (b), (v)].

Intestinal Perforation in Enteric Fevers*

BY

R. D. PENDSE, M.B., B.S.,

Poona City.

INTESTINAL perforation in enteric is happily not a common complication, the p.c. being about 3-4 or so. But it is on the other hand responsible for about 30 p.c. of deaths from all causes in enteric fever. The complication is so grave that without operative interference mortality is cent per cent. But if diagnosed early 30 p.c. cases can be recovered in best hands. Any delay in diagnosis makes the thin chance of successful operative interference recede further and further. So the diagnosis should be very prompt, with as least delay as possible. For this it is not sufficient only to remember that perforation occurs in enteric fever, after three weeks or so, but on all occasions one must be on the look-out for the early signs and symptoms of this complication, and in differential diagnosis of every new sign or symptom that may arise after three weeks, perforation must be considered and excluded. It is by methodical attention to all points in the symptomatology and signs that one can hope to make the diagnosis at the earliest. Diagnosis becomes easier and straightforward as time goes on, and all the features of general peritonitis make their appearance, but by that time the patient has gone beyond all hope.

Even in spite of being watchful and alert, early diagnosis is quite a job. Why it should be so, why the early diagnosis should be difficult? It appears to the writer that there are two factors responsible for this. The first is that the patient is generally already seriously ill, generally for three weeks or so, and the condition of the patient at the time of perforation, may vary greatly from the fully developed typhoid stage, to a comparatively good condition that may reasonably be expected at the end of three weeks of continuous high fever. This difference, this variation of the general condition of the patient within wide limits—in ambulant typhoid it is said that perforation may be the first thing noticed—that is possible,

* Specially contributed to 'The Antiseptic.'

makes the first reaction of the patient's system to perforation also to vary within a wide range. There is no one clinic and characteristic picture. In one case the patient may be seized with agonising pain, sharp and unbearable, while another unconscious, or one bordering on it, may not be able to appraise pain. In one there may be sudden collapse, with fall in temperature, and yet in another the system already overpowered with infection, cannot react to anything else, so there is hardly any change in the condition.

Primarily there is this inherent possibility of the patient's general condition being so different from patient to patient, and consequently possibility of varied types of reactions to perforation. In addition wide variations in any one sign and symptom can occur due to the time that might have elapsed before examination is done. The course of events on general broad out line can be divided into three stages, 1st stage of early collapse due to peritoneal shock, 2nd stage of remission with abatement of symptoms but definite localizing abdominal signs, and the 3rd stage of terminal toxæmic collapse caused by general peritonitis. All signs and symptoms are not present in the same degree and extent in all the three stages. Some are definitely to be found in one stage while others are conspicuous in other stages. Not only this but the intensity and definiteness of the same symptom or sign may change as the case progresses.

Second factor that contributes towards mistakes in diagnosis is the period of remission that occurs after the primary collapse. The doctor visits the patient second time after some interval, finds him more comfortable, pain much less, temperature not sub-normal, but returning to its previous level, and thus causing no alarm, and on the whole the patient or the relatives state that the particular medicine, or more likely particular injection, that was administered immediately after collapse, has done the patient immense good. Naturally the doctor thankfully takes the credit, wishfully persuading himself that all the frantic calls to come immediately and do something for the collapsed patient, and this anxious incident of dramatic nature, might after all have been due to some quite trivial cause in the already exhausted and weak patient, and may even rebuke himself for being alarmist in suspecting perforation. Patient's relatives always hate an alarmist doctor. This remission, this feeling of well-being on patient's part, and relying too much on general improvement in general features to the exclusion of the abdominal signs on the doctor's part, combine to make the second factor which makes the diagnosis difficult in the second stage of remission.

So, for an early diagnosis reliance should not be placed too much on any one particular sign or symptom. Its presence or absence should be firstly correlated to the patient's condition immediately before perforation, and secondly with the possible time that might have elapsed between the suspected perforation and the time of examination. Then only the presence or absence of any sign or symptom can be intelligently interpreted. This will be evident when signs and symptoms are considered separately one after another.

Symptoms.—1. *Onset*:—It is usually dramatic. Perforation occurs on 21st day or after. It becomes rare after definite decline, but may even take place during early convalescence. Perforation takes place at the time of separation of sloughs and is generally situated within the last foot of

ileum. It may be ushered in by shivering. It can not be definitely predicted in which case it will occur and in which case it will not occur. But cases attended by much meteorism, diarrhœa, or gastro-intestinal upsets are potential candidates for this complication. Great toxæmia should make one alert and watchful. Intestinal hæmorrhage sometimes immediately precedes this complication, and the collapse due to hæmorrhage is not recovered from but gradually merges into toxæmic collapse of general peritonitis.

2. *Pain*:—It is invariably present unless the patient is unconscious and unable to feel pain. Pain is the most dominant symptom and the first to attract attention. It is very characteristic; sharp, of increasing severity and paroxysmal. It is visceral in type and not definitely localized. It is usually referred to hypogastrium or right iliac fossa. In the third stage of general peritonitis character of pain changes from visceral to peritoneal, becomes localized and dull and is less prominent, though it is never absent in conscious patient. Pain is very severe and is one factor in causing the primary collapse.

3. *Vomiting*:—It is variable but usually present. It is not related to food, slight at onset, becomes frequent afterwards, may become incessant and distressing.

4. *Rigor*:—In the usual type of enteric rigors spell some other complication than the original infection. And perforation may take place after a definite rigor and shivering. But there is not uncommon variant type in which chills are a regular feature. In diagnosis of perforation presence or absence of a definite rigor has no great significance. When it coincides with other prominent signs, and when it happens to occur for the first time, even then it is only of secondary significance, suggesting that some new complication has taken place, but no particular complication.

5. *Collapse*:—In the first stage it is due to peritoneal shock caused by leakage through perforation, and also to unbearable pain. In the second stage of remission patient somewhat recovers, and his collapse not quite marked. In the third stage collapse is due to bacterial toxæmia resulting from general peritonitis and absorption of toxins from the peritoneal surface. This gives rise to hippocratic facies, which only means patient is almost near death. This terminal collapse is never recovered from. In cases where hæmorrhage precedes perforation or in cases already collapsed before the pre-existing collapse soon becomes profound and the end comes rapidly. In these cases presence of collapse does not help in the diagnosis.

6. *Hic-cough*:—Appears late and really not of much help in diagnosis.

7. *Motions and urine*:—Diarrhœa may be troublesome from the beginning but constipation of first few weeks may give place to loose motions prior to perforation for a few days. With the onset of general peritonitis and consequent intestinal perisis, distension and constipation results. Sometimes after perforation there is increased frequency of urination and is due to irritation of pelvic peritoneum.

8. *General features*:—These vary greatly. In the early stage patient resents interference of any sort and prefers to lie with his knees drawn up, and on one side. His face is clearly very anxious and there is increased pallor, his head and face covered with clammy cold perspiration. As

general peritonitis supervenes and the patient, becomes dehydrated, his expression becomes pinched, and appears to shrivel up. There is great aggravating of existing prostration. In the end Facies Hippocratica is clearly obvious.

From the above consideration it will be seen that of all the symptoms occurrence of severe pain is fairly constant, and collapse when not present already is the second constant finding.

Signs. 1. *Pulse*:—It is increased. But sometimes, possibly due to vagus effect, for the first hour or so it may be of surprisingly good quality. The teaching that if the pulse is not above 100, nothing serious is happening in the abdomen is not always infallible. But soon it becomes rapid, the rate mounting steadily, the so-called 'rising pulse.' In cases that are carefully watched from hour to hour this rising pulse is very pathognomonic.

2. *Respiration*:—It is generally increased, and becomes completely thoracic.

3. *Temperature*:—If shivering occurs just prior there is some rise in temperature. But with perforation it generally falls quite low, and may become subnormal. In the second stage it again almost comes to its previous level. With the advent of toxæmic collapse it again falls or sometimes the temperature becomes fixed at a higher level for two or three days, without showing any variation during 24 hours, and then the perforation occurs. Except the sharp fall that occurs immediately after perforation temperature is not of much value in arriving at the diagnosis.

4. *Local signs*:—These are very important. Most conspicuous from the beginning are the abdominal muscles rigidity, spasm and increased tension. These depend upon the condition of abdominal muscles, and therefore more prominent amongst young and well developed subjects. It is less conspicuous in old fat and feeble, and when there is much toxæmia. Rigidity is due to peritoneal irritation and is a muscular response. The respiratory movements become gradually, completely thoracic. Rigidity becomes less marked during later stages. If there is no previous distention or meteorism the abdomen may be scaphoid but is quite exceptional. Generally the abdomen is distended in the first instance due to meteorism, but soon there is intestinal peristalsis and general peritonitis. Any increase in the pre-existing distention is not an early feature and should not be depended upon in the early stages. Simple distention with gas and meteorism is not accompanied by rapid or mounting pulse, nor by fall of temperature. On the contrary simple distention causes rise in temperature. There may be flat note in the flanks due to exudate, and this dullness may be shifting with the position of the patient. There may be, on the other hand, pneumoperitoneum due to escape of gas in the general peritoneal cavity. Then there may be obliteration of liver dullness.

Tenderness on palpation and easily brought out by pressure is generally quite marked. It is localized in the beginning but becomes diffused as general peritonitis develops. Tenderness is more marked when the perforation is situated near the parietal peritoneum. On rectal examination there may be fullness or tenderness in the pelvis.

5. *Blood counts*:—There is rise in total white cell counts and is

progressive. However no one depends on that and waits exclusively for that to happen.

Out of the above signs abdominal rigidity and tenderness are early signs and should be carefully looked for.

Intestinal perforation is not a common complication and therefore the general practitioner is not quite familiar with all its aspects. This results in late diagnosis and the surgeons justly blame the attending doctor. The writer, who is a general practitioner himself, wishes to put forward the plea that it is better to be somewhat alarmist in one's own mind, and when any suspicious symptom or sign arises it should be the first rule to consider perforation and completely satisfy oneself that it has not taken place. Local abdominal signs are more trustworthy than the symptoms, and careful consideration of them all as to how they fit in with the previous condition of the patient and with the different stages will help towards the early diagnosis. Then only patients and surgeons will get a fair chance.

Schilder's Disease*

(Encephalitis Periaxialis diffusa)

BY

A. V. S. SARMA, M.B., B.S., D.C.H. (Eng.),
Hon. Assistant Physician, Stanley Hospital, Madras.

Definition.—“A malady characterised anatomically by a progressive and massive demyelination of the white centre of the cerebral hemispheres, proceeding from a single focus or from two symmetrical foci, and producing the clinical picture of progressively increasing failure of cerebral function, local at first, but advancing in terms of the function of the contiguous regions which are not affected, by the spread of the disease from its starting point.”

Ætiology.—Nothing is known definitely. Sexes are equally affected. The disease is either infective and inflammatory (BICLOWSKY and HENNERBERG) or familial and degenerative. The condition starts in childhood (even in the second year).

Pathology.—The characteristic lesion consists of a primary demyelination and later destruction of the axis cylinders of oval centres producing a translucent jelly-like appearance, over growth of neuroglia, and a general round celled infiltration of the white matter of the brain.

Demyelination commences most commonly symmetrically in the occipital white centres, and less frequently in the temporal or prefrontal white centres. The corpus callosum and the brain stem are involved but the spinal cord is spared.

Symptoms.—Blindness (with no change in the optic disks and with pupils normally reacting to light), bilateral deafness and later bilateral ataxia and astereognosis, bilateral spastic paralysis and complete amentia occur, generally in the order given.

The initial lesion may be in the temporal or frontal region with an

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altered order of symptoms; and if the initial lesion is unilateral, hemianopia or hemiplegia may herald the disease. Headache, vomiting and papilloedema appear in some cases due to increase in intracranial tension and these may suggest an intracranial tumour. Fits are common and, though rarely, fever occurs in some cases. C.S.F. is normal or may contain an excess of protein and lymphocytes.

Diagnosis.—Cerebral blindness or bilateral deafness with progressive amentia means Schilder's disease. Intracranial tumour may cause confusion, but high grade papilloedema and consecutive optic atrophy do not occur in Schilder's disease. Encephalography may also help in the differential diagnosis. Disseminated sclerosis may be ruled out at the age at which Schilder's disease occurs.

"Any locally commencing progressive destruction of the brain may be an example of this malady."

"There is really no other disease affecting children or young subjects which presents the following triad of symptoms: (1) progressive loss of vision of the cerebral type; (2) progressive spastic paralysis; (3) progressive mental dissolution." (BRAIN and STRAUSS).

Course and Prognosis.—Schilder's disease progresses to death while rarely arrest occurs or improvement may result from use of mercury, arsenic and iodides. The duration averages to 9 months.

Treatment.—Treatment is at present empirical. Arsenic mercury and iodide give temporary benefit. Sedatives are indicated for convulsions.

Illustrative Case.—Kamala Devi, Hindu, female child, three years old, was admitted on 19-2-1943 and discharged on 19-4-'43.

Complaint:—(1) Blindness. (2) "weakness" of neck muscles, (3) inability to walk.

Family history:—The child is the third amongst four. The first pregnancy ended in an abortion; second is a girl of 6 years; third is the are healthy.

Present illness:—The child is said to have been well till a fortnight ago when she stood up for passing a motion and then she flopped down. Later the child had fever and fits and also vomited a couple of times.

Clinical condition:—The child is in fairly good state of nutrition. Anterior fontanelle closed. Bridge of the nose is depressed. Neck is short. Walks with a broad base, and gait is spastic and this is only with support from another. Muscles in good nutrition.

Nervous system:—The child appears mentally normal. No abnormal movements seen. Cranial nerves: I, III, IV, VI—Nil abnormal. VII—nil particular. Folds of the face normal. VIII—Hearing not impaired. IX, X, XI—nil abnormal. Vision lost (detailed report on fundus noted below).

Sensory system:—Nothing noteworthy.

Motor system:—All tendon jerks exaggerated. Abdominal reflexes present in all quadrants (?) Planter reflexes—extensor in response. No signs of meningism or meningitis (Kernig's sign or retraction of head absent). Abdomen—Liver just palpable. Spleen not palpable. Umbilical hernia present. Cardiovascular—Nil abnormal. Lungs—Clear.

Investigations done in the ward:—Urine—Nil particular. Blood—20-2-'43 no malaria parasite. Polymorph. Leucocytosis present. Faeces—Nil particular.

X-rays of both Tibiae 1-3-'43:—No nibbling of upper and medial ends of tibiae seen.

Wassermann and Kahn of mother's blood:—Both negative (11-3-'43).

22-3-'43. Blood cholesterol 450 per cent.

31-3-'43. " 470 "

18-3-'43. Lumbar puncture done—clear fluid. Pressure 60 drops per minute. No spinal block. C. S. F.—15 cells per cm.m. Pandy's test for protein negative. C.S.F.—Chlorides 730 mgm.%. Protein—60 mgm.%. Sugar 63 mgm.%.

24-3-'43. X-rays of wrist and palm. Two carpal ossifying centres seen. (No sign of retarded growth). No signs of rickets. Humerus—nil abnormal.

13-3-'43. X-rays skull—nil particular. No separation of sutures seen. Pituitary fossa—nothing abnormal.

25-3-'43. X-rays spine—nil abnormal. No changes in spine seen suggestive of gargoylism.

29-3-'43. X-rays of cervical region—seven cervical vertebrae seen (normal).

Study of fundi: (22-2-'43) Pupils, size normal. React to light. Ocular muscular movements normal. After 1 per cent. Unguentum Atropinae—the patient is seen not to appreciate light.

Fundus: (24-2-'43) Discs—Margin regular. White in colour. Vessels—Arteries thinner than normal, veins slightly larger. Retina—Looks lazy throughout. No exudation or haemorrhage seen. Picture suggestive of optic atrophy.

31-3-'43. Pupils still dilated and inactive with direct and consensual.

Fundi show yellowish white crystals all over (cholesterol?) The picture is suggestive of a primary optic atrophy in both eyes. The child kept a normal temperature throughout her stay in the hospital.

Weight of child:—18 lbs. (21-2-'43); 19 lbs. (7-3-'43); 20 lbs. (28-3-'43); 21 lbs. (4-3-'43).

Comment:—From the clinical data the following points are important: (1) Onset of the condition with fits and vomiting. (2) The presence of central blindness. (3) The presence of spastic paraplegia. (4) Increase in cells and protein content of C.S.F. (5) The presence of non-consecutive optic atrophy. (6) The presence of high blood cholesterol.

W. RUSSEL BRAIN writing about the etiology of Schilder's disease says: "The early infantile examples have been attributed to a failure of development of the myelin sheaths, later cases to a failure to maintain their nutrition. This in turn has been described to a defect of the Oligodendrocytes (GREENFIELD) a disturbance of the power of the glial cells to regulate lipid metabolism (SCHOLZ), and the presence of abnormal lipoids in the blood. (BIELCHOWSKY and HENNEBERG)."

A trial of treatment with thyroid in this case was not useful during the two months under observation.

Conclusion. This case is diagnosed as Schilder's disease, a rare condition challenging the clinician's attention. In this case gargoylism might arise but no characteristic bony changes are present along with an enlarged liver and high cholesterol; further spastic paraplegia and central blindness are against gargoylism. The umbilical hernia and high blood cholesterol might suggest cretinism, but retarded growth is absent as also a favourable response to thyroid. The writer feels that gargoylism and cretinism also have to be remembered in the differential diagnosis of Schilder's disease and this fact might also be stressed in the literature on the subject.

[The writer is greatly indebted to DR. G. R. DINKER RAO, M.D., Professor of Medicine and Principal, Stanley Medical College, and First Physician, Stanley Hospital, Madras, for permitting him to study and report this case under his care.]

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The Treatment of Hyperpiesia and Symptomatic Hypertension*

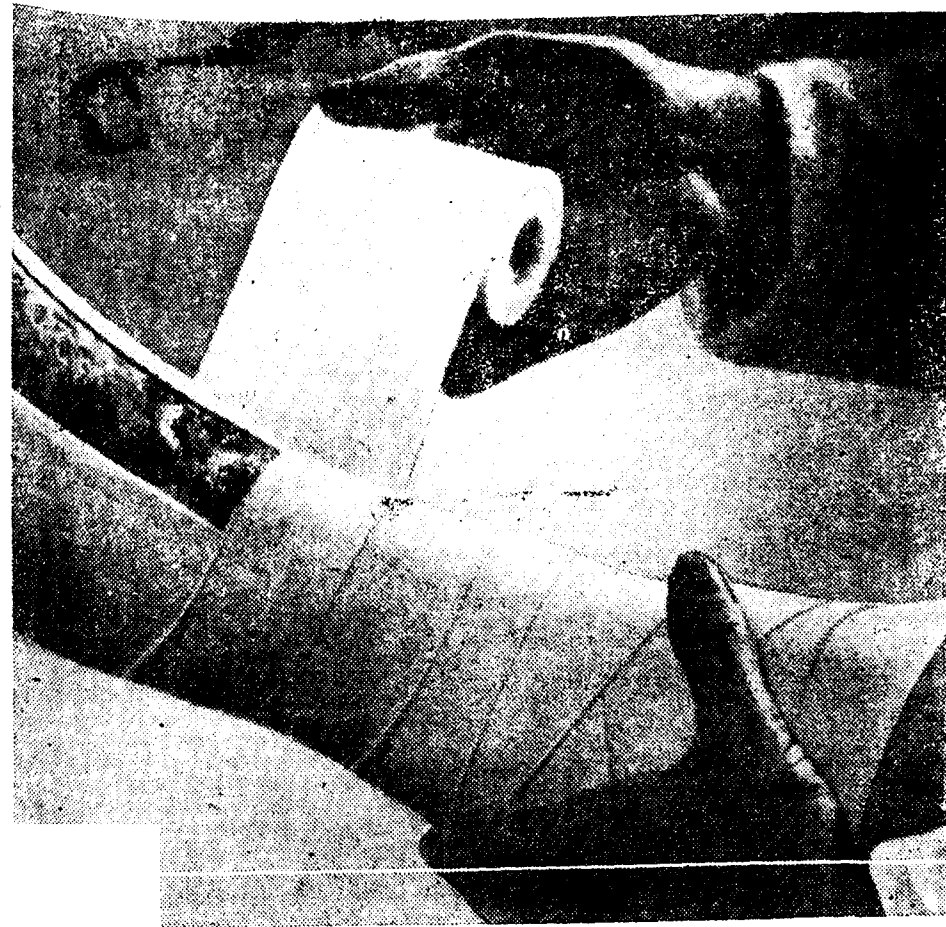
BY

H. M. KAR, B.Sc. (Alld.), M.B., B.S. (Luck.), P.M.S.,
Medical Officer-in-Charge, Civil Hospital, Sikandarabad, U.P.

THE treatment of hyperpiesia is one of the many unsatisfactory chapters of therapeutics, the fundamental causes being rather obscure. RALPH MAJOR said: "If our knowledge of the aetiology of arterial hypertension is shrouded in a haze, our knowledge of an effective therapy in this disease is enveloped in a dense fog." Until and unless we have a clearer conception of the etiological factors underlying hyperpiesia, anything in the nature of an effective or specific therapy is impossible. But this does not make us helpless, as much can be done to lengthen the life of a patient by controlling the pressure, increasing his efficiency and re-co-operates. We need treating the man rather than his malady in a majority of cases. Before a scheme of treatment can be regarded thorough, one should make a complete survey of the whole position when he is to treat a case of hyperpiesia. The patient's body, mind and spirit must all come under investigation.

This disease is usually first recognised in a person between forty and forty-five years of age. In the middle aged and elderly it is usually accepted that 150 mm. is the maximum normal systolic pressure but the demarcation line, in my opinion, seems to be an arbitrary one and a further rise of 20 mm. of mercury may be of no importance in the absence of any other evidence of high blood pressure. It must be noted that such pressure may not be of any significance on a single day examination, as it

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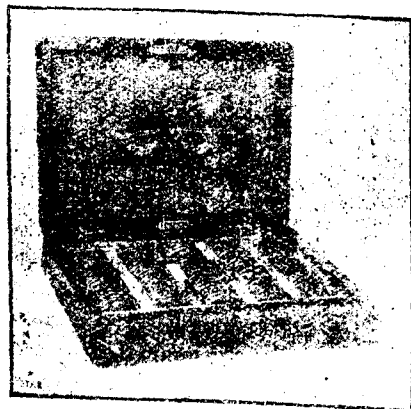
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is the persistence of height of the pressure that counts. Though the height of systolic pressure is given the primary importance in cases of hyperpiesia I think the diastolic pressure is of no less importance and it is of greater value specially in estimating the prognosis of a case, in addition to diagnosis, the reason being that it is less influenced by emotional states than systolic pressure. Most cases of hyperpiesia show a diastolic pressure above 100 mm. of mercury.

It is claimed that excessive strain of life in modern days of hurry and worry has led to an increase in this disease. This statement may have a trace of truth in it but there is no comparative figures relating to races who had a more leisurely existence for basing this assumption. Dr. GEORGE C. BASIL, spent some years in Chungking and is of the opinion that high blood pressure, heart disease and gall bladder troubles, all of which take a terrific toll in American life, are seldom seen among the Chinese; perhaps because when they relax, they do it completely and when they eat they do it slowly and with enjoyment.

Among the pre-disposing factors, contrary to the popular opinion, I don't think alcohol when used in moderation, has anything to do with blood pressure, and tobacco has very rarely any effect. Heredity is important, as shown by the family histories of these cases. The heavily-built, thick-set of individuals show a slight preponderance in this disease. In my 250 cases that I have kept record, the number of female patients was slightly more than that of males, perhaps due to the fact that many women at menopause show signs of hypertension. Recent findings attribute arteriolar spasm as the real cause of high blood pressure, as evident by the "considerable spontaneous fluctuations" of blood pressure specially in the beginning of the disease. It has been observed that diathermy can lower blood pressure for some weeks. Research work is being carried on regarding the effect of unilateral kidney trouble on the production of a picture of hyperpiesia, and timely nephrectomy may cure the patient. When the blood supply of kidneys is partially cut off, the blood pressure is raised by production of a *pressor substance* by the ischaemic kidneys. This piece of work may, in future, help a good deal to investigate into the aetiology of hyperpiesia. Hence, I think, every patient of this disease, should undergo a thorough renal examination, specially when he is of young age. In diagnosing, after a minute enquiry into the present and past history of the case, and a most careful physical examination, one should proceed as follows: (a) Blood urea clearance test should be done for the functional state of the kidneys. (b) A thorough examination of the fundus, and the kidney damage will be most evident by the presence of a definite exudate with haemorrhage. (c) Arteries should be examined for degeneration and sclerosis, for an idea of superficial arteries will enable one to estimate the damage in deeper arteries. (d) Examination of heart by physical methods and skiagraphy. The size of the heart will help in prognosis of the case. (e) Urine examination is one of the most important items.

A few granular casts, a slight trace of albumin in the urine associated with a fairly high specific gravity do not mean anything very bad, but on the other hand same casts and albumin associated with loss of specific gravity mean advanced kidney disease.

In asymptomatic cases, treatment should be almost entirely limited to advise about the general management of patient's life. I have found

that a good many cases require no special treatment, least of all, drug treatment, and some times they are much better off without it. Many persons with a symptomatic hypertension would have been more fortunate if they had never learned of their high pressure. General guidance, preceded by a clear and reassuring explanation of the position, is only effective in these cases. In educated patients it is a wise method to explain that arterial tension is preceded and accompanied by nerve tension, and this will relieve the patient's mind, and hence will act beneficially upon the hypertension.

Treatment of psychological factors plays a great part indeed. Perhaps the most important treatment is to refuse to tell the patient the height of his blood pressure. It requires tact and authority on the part of the doctor to keep the patient's attitude helpful, when the condition has got on the patient's nerves. "Emotional states should be brought under control: Anxiety concerning the patient's domestic and financial affairs, ambition in regard to his career, remorse for the past and taking too much thought for the morrow." The patient should free himself from all irksome and unnecessary responsibilities and an effort should be made to get him to take things more easily and face unpleasantness with equanimity. Excessive concentration on business should be stopped, and he should be induced to devote a sufficient part of his time to play and amusement; but I have found that the latter part of the advice is very rarely followed by a busy Indian. One should avoid strenuous exercise, hurry, exposure to severe cold and excessive smoking. It must be noted that the regulation of the mode of life should not be too stringent, and moderation in all things should be the watchword. Getting rich quickly is another psychological factor.

Focal sepsis has been placed as an important factor in the aetiology of hyperpiesia, and so, if present, it must be dealt with.

Adequate rest is very important, 8 hours rest in bed being minimum in night time, the sleep being undisturbed. The patient should at least rest for an hour after midday meal and the more strenuous part of business should be done in the first part of the day. Vacations are also necessary. Exercises should not be of concentrated sort. Brisk walking along the level may be recommended. Good ventilation of the sleeping room is important. Before ordering a vacation the physician should always be sure of the economic status of the patient, specially in this country, for the advice may not be followed by him, if he cannot afford it. The particular place where vacation should be spent is not of great importance provided the climate is pleasant and the environment restful. High altitude should be avoided generally. The exercise advised should be carefully adjusted to the functional state of the heart.

A number of people who eat much habitually are liable to suffer from hyperpiesia. It is the quantity which should be severely cut down in these cases, rather than depriving them some special type of food. Another indication to use dietetic measure, is to reduce obesity in these cases for relieving the heart of unnecessary work. In cases of flatulent dyspepsias, root vegetables, salads, and fatty food should be avoided. The two most important factors in diet are protein and salt restrictions. I don't think there is any justification for a rigid restriction of protein in the diet of a hyperpietic patient as long as his kidney function is

adequate. This was based on the fact that primary kidney disease was thought to be the origin of hypertension. Moderation should be exercised in the amount of proteins used, and there is no objection to meat in one of the daily meals. It is better to use meat, in these cases, three days a week and fish on other days. There seems no point in disallowing alcohol, provided it is taken in moderate quantities and provided it does not cause unpleasant flushing of the head and face, which may be pronounced in such a case. Tea may be allowed twice a day, freshly made and poured at once. It is better to avoid articles of food containing purin bodies such as sweet bread. Foods like chutney, sauces, kidneys, liver, meat extract, coffee may also be discarded from the menu of the daily diet. Salt should be reduced, both as table salt and in cooking, and patients should avoid salty foods, but there is no objection in using salt in cooking just to make the food palatable. Lemon juice is a good substitute with vegetables and fish, etc. In cases of renal or cardiac inefficiency, more rigid salt restriction should be done.

Fluid restriction forms one of the items of treatment. Opinions differ about the amount of liquid that may be taken in the form of plain water, mineral waters, barley water, etc. Any way this is useful in all forms of myocardial insufficiency. It is better to take large quantities of water and other fluids, according to some, "to flush out" (hypothetical) "poisons" from the body. Others advise moderation so as to save the circulation and renal function. I think the patient should be allowed to drink as much as can quench his thirst. Most of the fluid should be taken between and not at meals. When the quantity taken is excessive, the full stomach may prove to be an embarrassment to the heart, thus the diet used should be of small quantity, nutritious and palatable. Fresh fruit, lettuce, milk and honey should be recommended. As a matter of fact, diet has rarely any special favourable effect on hyperpiesia. The idea is, in the absence of any renal or cardiac failure, to eat as much as is necessary to maintain one's strength, as goes the saying "eat to live and not live to eat."

Many patients of hyperpiesia are obese and overweight and this is a handicap for an already overworked heart. Hence the heart must be relieved by diminishing the load, by cutting down the quantity of food taken, till the patient begins to lose weight. The main aim should be to lose about a pound per week and to continue on such diet. In cutting down, fats, butter, cream, starches, bread, cakes puddings, etc., should come first. I think keeping a fast twice a month is a good procedure. It must be noted that the restriction of diet should not weaken the heart which is already damaged and thus induce general asthenia. It is the relief of heart which is rendered by the loss of weight and not the diminution of blood pressure. Thyroid may be used with beneficial effect in obese persons but should be avoided in females at the menopausal age.

In European countries *Spa* treatment is recommended but it is a combination of mild climate with eliminating measures, an ordered regime and freedom from "business cares" which does good in these cases.

Regarding drugs used in the treatment of hyperpiesia it might be said that we possess no drug, a careful test of which has proved it to possess specific value in this malady. They are numerous and are based upon theory or tradition. In my opinion the less they are used, the better

for the patients specially in asymptomatic cases. The drugs which have been known to diminish blood pressure do more harm than good. Iodine and Iodides are often used, but the actual reason is not known. They should be taken over long periods and in fairly big doses to be useful. Nitrites are vaso dilators, but are disappointing in practice apart from their great value in aginoid states. The slower acting preparations are to be preferred such as Sodium Nitrate gr. $\frac{1}{2}$ as tablet twice or thrice a day; Nitro Glycerine (1/100 gr.), tablet Erythrol Tetranitrate $\frac{1}{2}$ gr. T.D.S.; Amyl Nitrite (2 to 5 minim by inhalation), Amyl Nitrite is used in only acute episodes, notably in angina pectoris, but others are used for protracted periods. I have used Sodium Iodide with beneficial effect for periods of 6 weeks and a month's intermission. An organic preparation of Iodine such as Lipiodine has been found useful; in form of tablets (5 gr.) one on one day and two on the next day. I have used Guipsine pills ($\frac{2}{3}$ gr.) thrice a day with very good results. It is a vegetable medicament for the relief of hypertension. Each pill contains 5 centigrammes of useful principles of Mistletoe. It is manufactured by M. LEPRINCE, Paris, France. I prescribed this pill in many of my patients. For the first three days I gave 4 tablets a day, and the next three days three tablets a day and finally when the hypertension is slightly relieved one tablet twice a day is sufficient. I have used this tablet in a case of crisis of one of the hypertension patients along with venesection. In some cases good results are obtained with Bismuth Subnitrates gr. 10 T.D.S. in capsules. Theobromine Sodium Salicylate, Calcium salts, Sulphocyanates are also used but are considered valueless. Liver extract (the equivalent of 8 oz. of liver daily) has been tried. Good results have been obtained with a substance in water melon seed that lowers the blood pressure by peripheral vasodilatation. Among the patent medicines of Indian make Serpina tablets prepared from root bark of Rauwolfia Serpentina, has given satisfactory results. It is manufactured by the Himalayan Drug Co., Bombay. The dosage I have been using is 2 tablets three times a day for the first 3 days, and then one tablet every 6 hours and finally when the blood pressure is controlled one tablet T.D.S. The choline group of drugs has had an extensive trial, again with poor results, specially because they depress the heart and therefore the patient, if continued. The two preparations most commonly used are Acetyl Choline (gr. $\frac{1}{2}$) given intramuscularly, once a day increasing to 1½ gr. a day for a fortnight in each month over a period of 3 months, and Doryl (choline urethane) which can be administered orally, the dosage being 1.60 grain thrice a day. Sodium and Potassium Thiocyanide have been used but they also depress the heart.

Sedatives are often of great help to these patients, particularly in cases where they are anxious, worried and irritable, and form valuable adjuncts to the rest treatment. Among the most useful sedative drugs in hyperpietic patients are the Bromides, Chloral Hydrate, and Barbituric Acid derivatives notably Veronal and Luminol. Unfortunately the latter are being out of use because of their unavailability. Extracts of the pancreas, liver and other tissues, as mentioned above, are vaunted as having good effects and a variety of other drugs, notably extract of Mistletoe (of whose preparation is Guipsine pills mentioned above). But though we can do little or nothing by way of drug to reduce arterial tension directly, we can do some very useful things by means of them indirectly. In the matter of portal depletion, when indicated, Pillula Hydræ gr. 3 or blue pill after the evening meal is the best, once or twice a week

followed by a simple Saline next morning. For the nervous system, when indicated, there are two useful preparations Theominal or Theo-gardinal as a single evening dose or twice daily if necessary. The best sedative among bromides is the ammonium salt gr. 20 doses once in the evening and twice daily. Thyroid extract has been recommended by some on purely empirical grounds but recently a suggestion has been put forward that essential hypertension may be in nature of hyperthyroidism. If this view is correct it is obvious that thyroid extract is likely to do harm than good. It should be used with great care at the time of menopause and never when there is any suspicion of an associated hyperthyroidism. Hypertension during menopause is a well recognised group of cases. It is essential to reassure the patient, explaining the epochal factor to her and how this favours the prognosis. Full activity may be restricted whenever possible, though it may be a temporary restriction only. Ovarian hormones in these cases are worth trying.

Venesection has been a valuable therapeutic measure in addition to other treatment, in many instances of cardiac failure in hyperpiesia—particularly with the symptom of cardiac asthenia as well as in some of the extremely violent headaches of hyperpiesia that resist all other measures. It must be noted that the blood pressure is not notably lowered by abstraction of such volumes of blood as can be removed for therapeutic purposes. I have found this measure very useful in many cases of crises in hypertensive patients, which will be mentioned later. During all the above measures, it must be remembered that there should be no attempt to lower the pressure excessively as in many cases it is a compensatory mechanism and a certain increase in tension is requisite for the well-being of the patient.

In cases of hyperpiesia associated with severe headaches and vomiting a course of auto-hæmotherapy has been found beneficial. 5 to 10 c.c. of blood are removed from the vein of the patient's arm and immediately injected into the gluteal muscles. This is repeated once a week for 6 weeks.

In the cases of these patients, I have found the use of simple enema at night followed by a colonic injection of about 4 ounces of 25% Magsulph solution next morning beneficial. This lowers the pressure 10 to 20 mm. of mercury and should be repeated at intervals of 5 to 7 days according to the permanency of result obtained.

I think all my fellow practitioners are well familiar with crises in hypertensive patients. They may be mysterious with regard to their etiology but at the same time they are definite. These hypertensive cerebral attacks require active and prompt measures, of which rest in bed, starvation diet, portal depletion, as mentioned above, and venesection are the main elements. I have seen very striking results by using Guipsine pills combined with venesection in a few of such cases. In the cases where the patient can swallow, Magsulph drachm one in water may be given by mouth every hour. An intravenous injection of 40 c.c. of 30% Sodium Chloride solution should then be administered intravenously. If necessary, the patient may be quitted by chloroform during the injection. Lumbar puncture is often of great help in these cases to lower the intracranial tension and the blood pressure. Among depletion methods, an enema at night followed by rectal injection next

morning of 8 ounces of water containing 2 ounces of Magsulph run in slowly as a colonic wash out.

Lastly, we must not forget about the physiotherapy which consists of inductothermy. Both high frequency and diathermy are credited with good effects though temporary. The current is passed through the trunk and each sitting lasts for about half an hour, followed by an hour's rest. It should be given two to three times a week or at least in eight occasions. Vasodilatation and slight pyrexia are produced thereby. The benefit lasts seldom more than 2 to 3 months.

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Cod-Liver Oil in the Treatment of Tuberculosis*

BY

CAPTAIN KHUSHDEVA SINGH, M.B., B.S., P.M.S.,
Secretary, Central Anti-tuberculosis Association, Patiala.

COD-LIVER oil (*Oleum Morrhuae*) is a fixed oil, found in the intracellular fat of the fresh liver of *Gadus Morrhuae* or Cod fish. It is expressed by a heat not exceeding 85 degrees centigrade, and separated from the solid fats by filtration at a temperature below 0 degree centigrade.

It is pale yellow or dark in colour, depending on its freedom from the foreign matter, has got a bland fishy taste, and a specific gravity of 0.92 to 0.93.

There are several species of Cod fish from which the oil is obtained, other than the one named, but that gives the chief source of supply. Lofoten Cod-liver oil, obtained from Cod caught near Lofoten islands is generally considered better than many others.

Besides Cod there are other kinds of fish, namely Halibut, Shark, and Chond from which the oil is obtained and used for the same purpose as Cod-liver oil. Halibut-liver oil is, as a matter of fact, one of the richest natural source of vitamin A (containing sometimes 80 times the vitamin content of the finest Cod-liver oil). "B.M.G." Shark oil is expressed in India in the Government Oil Factory, Calicut, and is guaranteed to contain when packed, not less than 1500 International Units of vitamin A per gramme, and not less than 25 International Units of vitamin D per gramme. Chond-liver oil is expressed by a private firm in Bombay, and is vitaminised with synthetic vitamins to a great extent.

Composition.—Composition of different kinds of Cod-liver oil varies considerably, depending on the source of supply, the fishing season, and

* Specially contributed to 'The Antiseptic.'

the method of expression. The chief constituents however of an oil of B.P. standard are:—

1. Olein (80—85%); it is a fluid fixed oil and is oleate of glyceryl;
2. Palmitin, Myristin, and Stearin about 10%;
3. Free fatty acids as oleic, palmitic, and stearic;
4. Trimethylamine;
5. Bile salts and cholesterin;
6. Many alkaloids;
7. Traces of Iodine and Bromine;
8. Traces of phosphoric and sulphuric acid; and
9. Fat soluble vitamins i.e. vitamins A and D.

Active Constituents.—Up to the discovery of vitamins by SIR GOWLAND HOPKIN in 1912, and the establishment of vitamin A in the Cod-liver oil a few years later, the various components like bromine, phosphorus, and iodine present in traces in this oil, were wrongly accepted to be the active constituents of this medicine. Now it is established beyond doubt that it is the vitamins A and D which are really the active constituents, and make this oil of such an important therapeutic value.

The original source of the fat soluble vitamin A in Cod-liver oil is the chlorophyll containing seaweeds in which it is present in the form of Carotone (which is biologically related to and is a precursor of Vitamin A). These seaweeds are eaten by plankton organisms, and these in their turn, by the small fishes, which form the food of Cod.

Physiological Action.—*External:*—Cod-liver oil is a bland, non-irritating oil, and is certainly absorbed when rubbed in the skin.

Internal—Gastro intestinal:—Like all other oils it is absorbed and digested in the small intestine. It is proved experimentally that Cod-liver oil passes through the animal membrane quickly, and is so readily absorbed than any other oil. The facility with which it is absorbed is also shown by the fact, that it often cannot be recognised in the faeces, although equal quantities of other oils taken by mouth are passed unaltered, and this superior absorbability may be due to its containing more unsaturated fatty acids and bile salts than other oils, and also to its emulsifying more easily.

Tissues:—Not only is Cod-liver oil more readily absorbed than other oils, but it has been proved beyond doubt, that within the tissues it is more readily oxidised (to maintain the body temperature and supply the energy needs of the cells) and the final products of the metabolism are the same, as when fat is burnt outside the body, namely carbon dioxide and water. If it be eaten and absorbed in excess of the actual metabolic processes of the body, the excess is stored in the tissues as fat.

Effect on Calcium Metabolism.—Cod-liver oil in the gut facilitates the absorption of calcium and phosphorus, and thus exercises a regulating effect on their metabolism. This appears to be due to its high content of vitamin A and D, especially the latter.

Action on Bacteria.—MILLER in 1916 found out that the addition of perm oil to media, on which tubercle bacilli were grown, caused rapid regenerative changes in the bacilli. FONTES a little later showed that the addition of 1% Cod-liver oil to ordinary broth culture medium, prevented the development of culture of tubercle bacilli, while CAMPBELL and LIEFER in 1922 demonstrated a definite inhibitory action of this oil, upon the tubercle bacillus.

Now it is established that Cod-liver oil exerts a striking inhibitory effect on bacterial flora of wounds, not only when they are tuberculous in origin, but bacteria ordinarily encountered in infected wounds such as streptococci, staphylococci, and bacillus coli perish when introduced into this oil. This has not only supported the feasibility of the topical application of Cod-liver oil, but has made it of a great practical importance in the treatment of a number of diseases of tuberculous origin.

Food Value.—As is clear from the table shown below, it is a very good fuel food on account of its being very rich in fat. Weight for weight it produces twice as much heat as starch or sugar. Its action as a fuel is more prolonged, so it is a better "stand by." For its high fat content it has long been used as a fattening agent, and a concentrated nutrient. It is also highly anti-infective on account of its being one of the most potent of the vitamin rich foods.

Table showing fat in grammes per ounce caloric value, and vitamin contents of common fatty foods used in India.—

Name of article	Fat	Calories	Vitamin A	Vitamin D
1. Cod-liver oil	28	252	+++	+++
2. Butter	23.10	216	+++	+
3. Ghee (clarified butter)	24.0	223	++	+
4. Fat (mutton)	26.40	239	++	+
5. Lard	26.80	241	V. L.	O
6. Mustard oil	28.0	252	O	O
7. Coconut oil	28.0	252	+	V. L.
8. Cotton seed oil	28.0	252	V. L.	O
9. Olive oil	28.0	252	V. L.	V. L.
10. Gergem	28.0	252	O	O

+++ means rich in ; ++ means moderately rich in ; + means some or poor in ; O means none ; and V. L. means very little.

Dosage—Pharmacopoeial dose is one to four drachms, but as it is more a food than a drug so it is not necessary that the dosage should be strictly adhered to. Individuals vary a good deal in their capacity to digest this oil, some are extremely intolerant while others have a marked tolerance for this preparation, and can assimilate large quantities like one to two ounces per day. Generally speaking, it is largely a question of racial habit and climate, thus races inhabiting cold climates can consume larger quantities than those living in the tropics. Even in the tropics a patient can take and assimilate more of this oil in the cold season than in the hot season. A man can generally take more than a woman.

Therapeutics :—(A) *Internal administration.*—Physicians of past generations bestowed great confidence in the therapeutic values of this drug in tuberculosis, and many modern practitioners still consider it an excellent therapeutic agent. It has been proved that this oil has got no deleterious effect on tubercle bacillus, when taken internally, but even then there is no doubt, that it is an important remedy in early tuberculosis, and pre-tuberculous stage, when the disease is not active and the fever is not high, and in post-tuberculous stage when the activity of the process has subsided, and temperature has come down, be it only for the fact, that it contains a considerable proportion of easily assimilable fat, and a high content of vitamin A and D. Its use is indicated in :—

1. It is useful in persons who have got no tuberculous focus in the lung or other tissues, but have mucous membranes which are very susceptible to disease, and suffer frequently from catarrh of the upper respiratory passage.

2. Children with tendencies to scrofula, with enlarged tuberculous glands, especially tracheo-bronchial adenopathy, and who are underfed and anæmic often derive great benefit from Cod-liver oil, and many times the improvement is very striking. I know of a number of my cases, who not only gained in weight, but made a definite and a lasting improvement in their general health.

3. It is indicated in all forms of afebrile phthisis, and patients often improve in every way under its influence, especially in chronic fibroid phthisis.

4. Intestinal tuberculosis has recently been treated with Cod-liver oil and tomato juice, and all such patients who willingly take it, and digest it well in large doses should be given this oil, without incidentally curtailing their usual amount of other nourishment.

5. It often is of benefit in chronic bronchitis of childhood. My own cousin, an infant of about one year, suffered from whooping cough, and was cured of it in due time with the usual treatment. After some time he developed a sort of chronic bronchitis, which persisted on, inspite of all sorts of treatment. Getting tired I put him on Cod-liver oil, and to my surprise the cough disappeared in about two weeks. However I continued the administration of this oil for over two months much to the benefit of the general health of the child. After that I have always been prescribing this oil for the treatment of chronic bronchitis in children, and the results in a fairly large number of cases are quite satisfactory.

6. It is also useful for any chronic disease, associated with loss of flesh, as it acts as a food peculiarly suited to a wasting malady; thus, in marasmus, when used by inunction, or given internally, if the stomach will stand it, it is one of the best drugs we have.

7. It may be administered with great advantage in rickets, its vitamins rendering it a specific, similarly it is also indicated in diseases which occur due to the deficiency of vitamins A and D, such as nyctopia or night blindness, xerophthalmia etc.

Routes of Administration.—It is generally administered orally, except in those cases who cannot tolerate its taste and get nauseated, even with very small doses, when it may be administered by inunction.

It is also given subcutaneously or intramuscularly as a 3% solution of Sodium Morrhuate.

Some practitioners give it per rectum, a full dose of pancreatin being mixed with it, to increase its assimilability. This method however appears to be useless.

Leaving aside the internal administration, Cod-liver oil is of great use in a number of diseases, of tuberculous origin, when applied locally to the affected part, or injected into the gland or sinus etc. This is by virtue of its bactericidal and inhibitory effect on the growth of the bacilli.

When given by mouth, it is best to administer it in as palatable a form as possible. In former times crude oil was used, and as it has a very

disagreeable odour and taste, so it cannot be taken by an average patient. The light, or amber coloured oil, prepared by the modern methods, is a much better preparation and is acceptable to the patient. It is best that the pure oil should be given and many patients take it easily. With some the odour and the taste have to be masked, and this may be done in one of the following ways.—

It may be given in some orange juice, in some volatile oil, or in syrup of bitter orange peel, the last being a very good mask to its taste. Sometimes a pinch of salt placed in the mouth, before and after the oil is taken, aids in disguising its taste. Many patients take it in coffee or milk, while others use peppermint water or tomato ketchup for this purpose.

The oil may be taken on a full stomach, but as a general rule it is best digested, if taken two or three hours after meals, when gastric contents are about to be passed in the small bowel, where the oil is digested, and if it be immediately followed by a little pancreatin, its digestion will be much aided. It is also readily digested, when given in single nightly dose after supper, or after a light meal just before going to bed.

It should never be given on an empty stomach, as it may not be tolerated and cause biliousness, and when continued for sometime may lead to other serious disturbances of liver, which may give rise to a condition known as acetonæmia.

For patients who cannot tolerate the smell, or taste, and are nauseated even with very small doses, a large number of malted, emulsified, pancreatised creosoted, and purified preparations are on the market. The pancreatised preparations are the best, if oil is present in sufficient quantities to do good. Personally I do not insist to prescribe Cod-liver oil in any form to such patients, and get equally good results, by giving them adequate amount of fresh butter, and some medicinal preparation containing good amount of vitamin A and D.

When administered by inunction, I generally advise that half to one ounce of crude oil may be lightly rubbed in one limb daily by rotation. In this way the turn of the same limb comes on the fourth day, when the said limb is dry, and in a condition to absorb a good quantity of oil. The excess of the oil may be sponged away with warm water and soap. In afebrile cases it can be rubbed in all the limbs at one time, and half an hour later the patient can have a warm water bath. Exposing the body, after the massage, to the rays of the morning sun for about fifteen minutes is beneficial, but is not practicable in the cold season or in cold countries.

Contra-indications.—The administration of Cod-liver oil is contra-indicated:—1. If the fever is active. 2. If there is severe hæmoptysis. 3. If it disturbs digestion, and causes nausea, vomiting or oily taste in the mouth. 4. It is contra-indicated in diarrhoea, whether primary or induced by oil. 5. It is also contra-indicated if it produces a feeling of unpleasant heat in the body or insomnia. 6. And last of all when rapid degenerative changes are occurring in the lungs, as it is not of any service, and may prove distinctly harmful, by disordering the digestion, and destroying the appetite.

Cod-liver oil as Local Treatment.—Locally the use of Cod-liver oil is indicated in—

1. It is used in the treatment of scrofuloderma and lupus vulgaris,

the results are reported to be invariably good in cases of scrofuloderma, though not always so in the case of the latter disease.

2. Suppurating tuberculous lymph nodes, when seen early heal well, on repeated aspirations, followed by injections of Cod-liver oil. The amount of oil to be injected should be one fourth of the pus removed. I have tried this line of treatment in 21 cases out of which 13 have shown very good results, in four cases the skin broke down resulting in ulcers, and the other four left the treatment. Topical application of Cod-liver oil is also of great value in the treatment of tuberculous ulcers. I generally use pure Cod-liver oil, but for patients who do not like the smell of the oil patent preparations like milkan, and Crooke's compound Halibut ointment are available in the market.

3. Cod-liver oil is also of some value in treating cold abscesses, secondary to bone tuberculosis, by aspiration of the pus, and injection of some oil in its place, provided proper attention is paid to the treatment of their source in the bone, by adequate immobilisation with plaster of Paris.

4. The oil when used in the form of eye drops, is of great value in the treatment of phlyctenular conjunctivitis, and keratoconjunctivitis cases. I use it in all my out-door cases suffering from the above-mentioned disease and have found that it gives better and prompter response than other measures. It is instilled into the conjunctival sac three times a day.

5. It is also of great value in the treatment of tuberculous pharyngitis and laryngitis. The treatment consists in spraying the throat with Cod-liver oil by means of an ordinary atomiser three times a day. Ordinary medicinal oil is used and sterilisation is not necessary, as it is free of bacteria. I have tried it in nine cases and six out of these are showing marked improvement. Favourable therapeutic response is seen only in suitable cases in which the disease is not very advanced and the ulceration is not confluent. It generally takes from 4-6 weeks before a marked improvement is seen, though the pain, dysphagia, and other symptoms subside even in much less time.

6. It is also used in the treatment of tuberculous ischio-rectal fistula by daily injections into the fistulous tract. It is recommended that the treatment should be given with the patient in the prone position, and he should keep it for about half an hour after the injection. I personally prefer to open out the fistulous tract, and dress the resulting wound with Cod-liver oil packs, and the wound takes much less time to heal.

7. Tuberculous empyema in the absence of bronchopleural fistula is reported to respond favourably to the aspiration of the pus, and to its replacement by the oil. 50 to 100 c.c. or even more may be injected after the pus has been aspirated. There are some patients who do not tolerate this treatment and may show reaction in the form of dyspnoea and cyanosis, and in these this line of treatment should be given up.

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Nicotinic Acid Therapy*

BY

A. CHANDRA MOWLI, L.M.S., B.S., S.C., L.O.,
Eye Surgeon, Conjeevaram.

IN this short note it is intended to draw attention to the increasing sphere of usefulness of nicotinic acid as a therapeutic agent. It has been largely written about and discussed that among the therapeutic agents of pellagra such as amino acids, histamine, high protein diet and nicotinic acid, the last is far and away the most important. Its effects are, generally speaking, dramatic and certain in many lesions of pellagra. I am not sure that one comes across cases of frank or classical pellagra in children or adults in South India, as largely as one does elsewhere in this country. At any rate during seventeen years of professional work, I have not met with many in my district (Chingleput), but recently I found occasion to appreciate the drug's usefulness in a number of cases of infants, children and adults, in whom one or other of the lesions of pellagra was found and the clinical picture therefore only suggested pellagra but did not definitely point to it. The administration of nicotinic acid was, as a consequence, only empirical. I am inclined to look upon these cases as sub-clinical pellagra or merely nicotinic acid deficiency states. The signs and symptoms which point to nicotinic acid therapy are dermatitis, diarrhoea and dementia.

Dermal Lesions.—These were mainly observed in infants and children. The infants seen were six in number till the date of writing this note and I am sure I shall come across several more. All these were between three and six months old; all showed the rash starting as a sunburn or erythema going on to a dry scaly or weeping eczema; the minimum duration was two weeks and the maximum six. The situations were fairly typical and suggestive. The exposed surfaces and those subject to irritation by clothes were affected in four cases. The other two showed the affection to be universal, i.e., all over the skin of the body. The scalp in all cases was also affected. Three of these infants were reported to be suffering from unprovoked diarrhoea as well. These lesions appearing especially on the limbs and scalp go by the Tamil name spelt in English as "Karapan" and are believed to be chronic, lingering on till the infant grows to be twelve or thirty-six months old when the lesions are said to disappear spontaneously. The patients therefore are brought to the doctor only for temporary or symptomatic relief. As three cases presented the symptom of diarrhoea as well as dermatitis I was led to think of nicotinic acid and so prescribed the same fifty milligrams twice a day. The rest although not suffering from diarrhoea were treated with the same drug empirically. All these infants were fair so that the initial flushing of the skin, if any, that might have occurred as a result of giving the drug by mouth, might have been easy to notice, but no such phenomenon took place. Wherever I thought that the mother's milk was inadequate on account of the mother's poor health or physique, I presented fresh boiled cow's milk in addition. The result of the treatment in five cases was very satisfactory. They were completely cured in two to three weeks. The sixth has shown definite improvement. The reason why these lesions are seen to persist till the child may be twelve to thirty-six

* Specially contributed to 'The Antiseptic.'

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months old is perhaps that the child struggles with the condition till such a time as it is able to absorb the necessary amount of P. P. factor from the variety of food it may get as growth goes forward. In none of these cases was any other medicament internal or external prescribed.

In addition to these as observed above three other children with dermal lesions were observed and treated with Nicotinic Acid. One was the case of the author's boy aged thirty months. This child, a bit anæmic, had just recovered from an attack of infantile hepatic cirrhosis and the liver function was not yet fully restored. In this state the boy contracted scabies and was struggling with it for three or four months. Sulphur ointment, Danish ointment, tonics, etc., did him no good. Meantime the scabetic pustules became chronic ulcers and he was eating and sleeping badly, being all the time dull and irritable. I thought of giving him small doses of Sulphathiazole (Cibazol-Ciba) internally and externally as well, in the form of a dusting powder combined with some suitable demulcent (Amylum), but withheld the administration, in preference to Nicotinic Acid which suggested itself to me on account of the anæmia, the mental symptoms such as irritability and peevishness added on to the skin condition. Nicotinic Acid was given 50 mgm. twice a day. No other drug internal or external was applied. In ten days the skin trouble entirely disappeared, the anæmia has improved and the child is very cheerful and playful. Apparently the child could not shake off the skin trouble in the presence of a B₂ deficiency.

The second case is that of a boy aged four years, just recovered from an attack of infantile hepatic cirrhosis, slightly anæmic, mentally dull, no response to questions and if pressed for an answer the boy cried. Skin was covered all over with chronic ulcers resulting from scabies. The boy was brought to me for some minor eye trouble and the father informed that the skin trouble was being attended to with Sulphur ointment and tonics. I stopped this treatment and got the skin painted many times a day with a solution of Sulphanilamide in Cod liver oil (10%). No improvement followed after a week. Then I supplemented this treatment with Nicotinic Acid internally 50 mgm. B.D. After a fortnight during which time the boy was in a village with his father, the latter came to report complete cure of the skin trouble.

The third is a similar chronic case of scabies, of a boy aged six years. He had scabetic ulcers all over the body, more especially in the usual sites for scabies, e.g., gluteal folds, webs of fingers and toes, medial sides of the thigh, etc. He was also running a temperature of 101°F. The boy was first put on Cibazol one tablet B.D., with the same as dusting powder combined with Zinc Oxide, and Amylum, the usual Sulphur ointment having done him no good for a fortnight. The response was poor after ten days. To this treatment of Cibazol I then added Nicotinic Acid 50 mgm. B.D. On the fifth day after the addition of Nicotinic Acid all the skin lesions completely dried up. Temperature normal.

Mental Symptoms.—The last is the case of a female aged thirty-eight years, who came to me for treatment of trachoma. She was an old patient of mine who was generally in a run down condition. The last time I saw her was in February 1944 after an interval of four years when she was more run down, anæmic, very silent, morose and irritable, and as the husband told me very peevish and boisterous at

times on the slightest provocation. She would shoot off at a tangent into a volley of abuse and loud hysterical cries with tears and that more often than not for no reason at all. She was therefore considered to be possessed and the magician did his best to wean her away from the devil. No good resulted. The anaemia and mental symptoms suggested Nicotinic Acid. This was given to her for a month at the rate of three hundred milligrams daily. The result has been very satisfactory. I saw her after this period. The anaemia is much improved and she looks quite cheerful. The husband informed me that she has had no attacks or outbursts of abusive language or cries during this period. I hope she will continue to improve. Nicotinic Acid or the P.P. factor has the formula $C_6H_5NO_2$ and was discovered by GOLDBERGER and TANNER in U. S. A. in 1925. The former's administration of this acid to dogs suffering from black tongue and the success following the same led to its use in pellagra in human beings with good results. It forms one of the four components of vitamin B₃ complex; those being Nicotinic Acid or the pellagra preventive factor, Riboflavin or Lactoflavin, the antidermatitis factor B₆ and the last the "filtrate" factor. The influence of the last is still quite incompletely understood but it is believed to influence favourably the rate of growth in rats. Riboflavine is believed to cure the syndrome of superficial keratitis, fissured tongue and angular stomatitis in human beings, the symptoms being caused probably by the deficiency of that factor. The usefulness of the anti-dermatitis factor speaks for itself. The P. P. factor is the one that has been probably well defined. The approximate daily requirement of this factor for a human adult is about 100 mgm.; yeast, liver, wheat germ, salmon and egg yolk contain this factor in fair quantities. I should add fresh milk to this test from my experience. One other use of Nicotinic Acid is the prevention and cure of Sulphanilamide poisoning. A useful way of giving it for prevention is 50 mgm. one fourth hour before each dose of Sulphanilamide. For cure heavier doses may be needed depending upon the degree of poisoning. I should be glad to hear of experience of Nicotinic Acid therapy from brother members of the profession.

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Tubal Rupture*

BY

T. P. TIWARI, L.M.P.,

Asst. Medical Officer, Jail Raipur, C. P.

EXTRA uterine pregnancy is not a very common condition and it is none too easy to recognise it either. A proper diagnosis of the same is a matter of no mean importance nevertheless, since that alone can avert what would otherwise be in many a case a veritable catastrophe. While uterine hæmorrhage in early months of pregnancy is admittedly a manifestation of an impending abortion in a large majority of cases, it would be worth while remembering that such hæmorrhage could also arise from

* Specially contributed to 'The Antiseptic.'

a ruptured ectopic gestation. A careful interpretation of the history coupled with a thorough examination of the patient should therefore invariably be the rule in all such cases.

It is now generally accepted that fertilisation of the ovum takes place in the fallopian tube and the fallopian tube is therefore the commonest site for an extra uterine pregnancy. A pre-existing salpingitis with its concomitant kinks and adhesions constitutes a barrier to the onward march of the fertilised ovum, which at its failure to reach the uterus gets embedded in the submucous tissue of the fallopian tube. There being little connective tissue in the tube for decidual formation the trophoblast soon encroaches upon the surrounding walls of the tube with the result that blood vessels are laid open. The ensuing hæmorrhage to begin with, is confined to the cavity containing the ovum which as a result almost invariably dies and in favourable cases all further developments may altogether cease and the mole may in course of time get completely absorbed. As is however more commonly the case, the walls of the tube, being considerably weakened as a result of the trophoblastic activity, usually give way and the subsequent course of events depends upon the site of the breach. If the rupture involves the inner wall of the cavity, blood collects into the lumen of the fallopian tube resulting in what is known as hæmatosalpinx. Blood gradually escapes through the abdominal ostium of the tube (if not obliterated by inflammatory changes) and ultimately gravitates to the pelvic cavity where it forms a hæmatocele. With a view to get rid of the mole, the tube gets repeated contractions and each contraction is followed by a renewed flow of blood which in its turn results in a progressive increase in the size of the pelvic hæmatocele. This is what is known as a tubal abortion which may or may not be complete according to whether or not the mole is discharged into the pelvic cavity. Hæmorrhage in these cases is not as a rule severe. A more serious and emergent condition develops when the trophoblastic erosion is directed towards the peritonium. The peritoneal lining of the tube sooner or later gets perforated and there is free hæmorrhage in the peritoneal cavity. If a big vessel gets involved in the erosion, the symptoms become positively alarming and the outlook in these cases is definitely grave.

The signs and symptoms of tubal rupture are characteristic. The patient who has generally missed one period is suddenly seized with a severe excruciating pain in the lower abdomen. She becomes pale, presents an anxious appearance and may even lose consciousness. Uterine hæmorrhage on account of separation of decidua soon appears but it is never much in quantity. The pulse is quick and small, the abdomen is tender and full and there are signs of free fluid in the lower abdomen. Vaginal palpation gives rise to severe pain on the side of the rupture. The symptoms in a case of tubal abortion are comparatively less severe. After missing a period, the patient suddenly complains of severe pain in the lower abdominal region, followed by a small amount of uterine hæmorrhage. The paroxysms of pain are repeated and the patient may even become unconscious. A pelvic swelling is always palpable in all these cases. If the hæmorrhage is confined solely to the tube, a big swelling can easily be made out at the pelvic brim. In cases, however, where a large hæmatocele has formed in the pelvis, the tubal swelling is generally not discernible and a mass is felt in the Douglas' pouch.

The commonest condition with which a tubal rupture is likely to be

confused is uterine abortion. Prominent features of an uterine abortion are less pain and more profuse hæmorrhage, in conspicuous contrast with those of a tubal rupture in which the pain is extremely severe and hæmorrhage rather remarkably negligible. Absence of an uterine enlargement, presence of hæmatosalpinx or pelvic hæmatocele, signs and symptoms of collapse without a commensurate apparent blood loss are outstanding features of a ruptured ectopic gestation which furnish an eloquent diagnostic data to any careful and observant practitioner.

Treatment of tubal rupture is purely surgical. No sooner than the diagnosis is established, abdomen should be laid open, affected tube removed and the pelvic cavity cleared of all blood clots. Drainage should be provided for a case of an infected pelvic hæmatocele through the vagina by performing the operation of posterior colpotomy. Collapse arising out of severe internal hæmorrhage should be dealt with by suitable resuscitative measures.

Cases and Comments

A Case of Purulent Tuberculous Peritonitis

BY

J. N. BHATTACHARJEE, L.M.F., L.T.M.,
Pathologist, Gopalpur T. E. Hospital, (Dooars).

A Paharia female child named Nepmaya, aged 4 years, came to the out-patient department of this hospital on 1st January 1944 with the following complaints:—(1) Rickets with slight œdema of foot; (2) slow fever with slight enlargement of abdomen.

General Condition.—The girl was rickety.

Structure.—Thin and delicate. Signs of wasting present.

Signs and Symptoms.—The girl had been suffering from chilliness since the last 3 months. There was a slow rise of temperature in the evening every day with morning remissions. Range of temperature 99°F. to 100°F. The abdomen increased steadily in size. There was no appetite and no hankering for food. Spleen 3 fingers below the costal margin. Liver not palpable. After a week abdomen much increased in size. The umbilicus became everted due to distension of abdomen and the skin over it became shining in appearance and was so much pointed that it looked like bursting through navel. There was no shifting dullness and no rigidity of abdomen.

Treatment.—Patient was first treated with anti-malarial drugs (Quinine with Arsenic), subsequently the girl was put on the following treatment:—

R Ammon Chlor gr. iii. Liq. Euonymin et Irid ℥ v. Ext. Kalameg ℥ x.
Ext. Gentian ℥ x. Aq-add 5 iii. To be taken thrice daily.

All sorts of treatment showed no improvement in the condition of the child. The abdomen increased steadily in size inspite of such treatment.

Lastly a superficial incision was made on the pointed navel region. After the incision thick green coloured pus came out in jets and about 3

pints of pus were evacuated. Size of abdomen decreased much. A rubber tube was inserted through the opening and the wound dressed aseptically.

Next day oozed bandage was opened and about 2 pints of pus evacuated. By mouth patient was given M & B 693 (Dagenan) 1 tablet 5 gm. T.D.S. for 3 days. After 3 days tube was removed and the wound dressed with Cod-liver oil with Sulphanilamide.

Within a week wound healed up at the first intention and the size of abdomen became normal. There were no other complications. Temperature fell down after the 1st day of opening.

The child was given mist. Cod liver emulsion for a month. By this her health improved much appreciably and the girl is enjoying her normal life. The child was definitely cheerful with all evidence of improved general condition.

Points of interest:—(1) There was no shifting dullness as is present in ascitis; (2) about 5 lbs. of thick greenish pus evacuated from abdomen—I do not find any other cause save the purulent type of tuberculous peritonitis. Pathological examination was not done; hence there may be controversy in diagnosis.

I shall be much grateful if any criticism appears from my colleagues regarding the true diagnosis of this case.

I am grateful to Dr. T. M. GHOSE, B.Sc., M.B., for his valuable suggestions and kind permission to publish this note.

A Case of Acute Obstruction by Fæcal Mass, Mistaken for Strangulated Hernia

BY

G. N. SINHA, L.M.F.,

M. O. Barwadih D. B. Dispensary, Palaman. (B. & O.)

I WAS called in to treat one Babu B. Das, guard, E. I. Ry., at about 2 a.m. in the night.

Complaint:—Pain in the abdomen of sudden onset and vomiting. On examination I found: (1) Abdomen greatly distended. Pain in the right iliac fossa; (2) constipation absolute i.e., not even the gas could pass; (3) external oblique ring on the right side of very great dimension; (4) pulse 60 per minute, regular but feeble; (5) pain very agonizing; and (6) temperature 96°F. in the axilla.

History.—(1) Habitual constipation; (2) aged about 50 years, of sedentary habits and irregular in diet and sleeping; (3) has inguinal hernia on the right side and wears a truss; (4) had a hearty meal consisting of meat, fish and bread and also other delicacies at about 7 p.m. the night, and the pain suddenly started at 9 p.m.; and (5) tried some *churan* for the relief of pain but of no avail.

Treatment.—(1) R Sodi bicarb gr. xv, Spt. Ammon aromate ℥ xx, Spt. Chloroform ℥ xx, Tr. Card Co. 5 i, Tr. Belladonna ℥ v, Aqua menth pip ad 5 i. Mft. 6 doses one every hour.

(2) Soap water enema given thrice but it did not respond.

(3) Hot water bag over the abdomen.

I attended the patient for the whole night, but the pain did not subside, on the other hand it was increasing. The abdomen also further distended, the vomiting also increased.

I suspected internal strangulation of the hernia or some other cause for acute obstruction, and as such I asked for removal to the Sadar Hospital in the morning but no means of transport was available for the next 18 hours. It was also not desirable to leave the patient unattended. I took the opportunity to consult one Dr. Ghosh, a fellow practitioner. Both of us differed in our diagnosis. He attributed these symptoms to mere indigestion and suggested:

- (1) Morphine gr. $\frac{1}{2}$ and Atropine gr. $\frac{1}{100}$ for injection to relieve the pain.
- (2) Ss. Magsulph $\frac{3}{4}$ i to clear the bowels.

I did not agree since Morphine might mask the symptoms and the purgative might increase the peristalsis and do further damage. After a good deal of discussion we decided to inject Morphine and give a purgative since no other course was left to us. I tried enema twice again but to no purpose. The patient also objected to the enema as five of them proved fruitless.

Morphine was injected and the Ss Magsulph $\frac{3}{4}$ i was given. In about half an hour the pain subsided and the patient slept. At about 3 p.m. the patient passed a motion in which a big, hard faecal mass came out. Subsequently he passed three more motions and a lot of gas. After passing the first motion his pain was relieved. He felt much better and demanded food. He was given plain barley water at 9 p.m.

Points of interest in the case:—(1) Hard faecal mass is often a cause of obstruction.

(2) Morphine and also purgatives are not generally chosen and used in cases like this. But fortunately for the patient it saved him an unnecessary journey and probable laparotomy.

(3) The presence of hernia on the same side of the pain misguides a good deal.

(4) What else a rural medical practitioner can do in such cases where expert aid is not available? Perhaps some reader may be pleased to suggest something.

Severe Pregnancy Anaemia

A Case Report

BY

J. S. BHATNAGAR, M.B., B.S., (Lucknow),

Chief Medical Officer, Jhabua State, C.I.

PATIENT aged 38 years, female, Hindu, housewife, was seen by me at Udaigarh B.B.C.I. about 20 miles from Jhabua where I was summoned to see her, with a history of fever with rigor since a week and vomiting (even water was not being retained) on the morning of the 11th April 1944. On examination I found her in bed with marked swelling over

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legs, face, lids and hands and 102°F. temperature. The patient was severely anaemic and was running the seventh month of her sixth pregnancy. Respirations were hurried and look anxious. Immediately 50 c.c. 25% Glucose solution and 10 c.c. 10% Calcium Gluconate solution I.V., Quinacrine 15 gm. and liver extract Lilly 2 USP unit power 5 c.c. I.M. were injected. Quinacrine was again repeated at 4-30 p.m. and she was brought with me to Jhabua main hospital to be admitted. At about 10 p.m., 4 gr. Morphine was injected. Glucose was repeated in the night.

12th April:—Blood examination. Total R.B.C. 5,30,000 (5 million) Hb percentage 21%, M.T. rings found, urine examination traces of albumin only. Quinacrine 15 gm. and 5 c.c. whole blood were injected I.M. Alkalies were given by mouth. Vomiting persisted but no temperature. Blood transfusion was not possible as no suitable donor was made available. At 5 p.m. Quinacrine 1 gm. was again repeated with Glucose and Calcium and 20 units of Insulin. The pulse still remained fast and there was no improvement in the general condition. At 10 p.m. about 4 oz. of sterile glycerine was injected in the uterus and cervix was plugged. Previously at 8 p.m. Prognon B. Oleosum Forte was given I.M.

13th April:—The patient started pains at about 1 a.m. hot saline enema, Ergometrine B.D.H. 1 c.c. and Pituitrin P.D. 1 c.c. were given. The woman delivered at 2 p.m. Pulse became fast and imperceptible and respirations rapid. Immediately Crooke's Glucose-saline 560 c.c. with Lactoflavine Roche were given by drip method finishing in about 1½ hours, followed by 50 c.c. 50% Glucose solution and 20 units of Insulin. About ½ oz. of Glucose was given by mouth. Atropine gr. 1/100 was injected to avoid basal congestion, and Neo-Gynergen 1 c.c. Sandoz for post-partum haemorrhage. Pulse slowly slowed down with respirations. In the morning following the delivery 10 c.c. whole blood was injected with Quinacrine 1 gm. General condition started getting better but vomiting still persisted.

14th April:—Vomiting was very much less. Lilly liver extract 5 c.c. I.M. was given, 3 tablets of Quinacrine were given, blood was checked, R.B.C. 10,30,000 Hb percentage 30%, and urine N.A.D.

15th April:—For 3 days daily 50 c.c. Glucose with 20 units of Insulin were given in the morning. From this day the patient was put on the following treatment: Quinacrine 1 tablet and 1/2 tablet Plasmoquin in capsules for 5 days only tds. Liver extract 2 c.c. daily I.M. for 1 week and then bi-weekly for 3 weeks. Esdavite pearls 1 tds. for 5 days and then 1 daily. Fersolate tablet 2 tds. for 15 days and then 1 tablet tds. Irradiated Calcinol granules 1 teaspoonful tds. in milk, 5 c.c. whole blood (auto-blood) I.M. bi-weekly. Acid Hydroch. dil xi with pepsin enzyme bd. pc. Haematinic Plastules with liver extract 4 cap. daily. Redoxon 6 tab. daily for 5 days.

16th April:—The same as above.

Blood was checked on—

17th April:—R.B.C. 13,20,000 W.B.C. 10,600 Hb percentage 34%.

25th April:—R.B.C. 24,90,000 Hb percentage 42%.

30th April:—R.B.C. 35,20,000 Hb percentage 48%.

Patient was discharged cured on 2nd May 1944, with the instruction

to continue Fersolate, Hæmatinic Plastules, Esdavite pearls, Acid Mixt., and 1 pill bd. pc. of the following prescription:—

R Quinine Hydroch gr. ii. Ext. Nux. Vom. gr. i. Ext. Belladonna gr. i.
Aloinum gr. i. Ferri Arsenas gr. 1/16. Ext. Gentian Co. Q. 6. To be
taken every alternate week for 2 months.

The patient was again examined by me on 10th June 1944 and I found her to be in normal health.

*Points of interest are:—*1. Induction of labour, not a safe procedure in such a severely anæmic patient which saved the life. To wait for natural premature delivery was not considered safe by me in view of the fact that blood transfusion was not possible.

2. Rapid increase of R.B.C. and hæmoglobin with liver extract and whole blood injections. Liver extract alone could not help much.

3. Utilisation of Glucose by Insulin injections.

4. No puerperal sepsis.

I should thank my assistant Dr. L. N. SAKSENA, L.M.P., and the nursing staff who carried out the instructions skilfully.

A Case of Obstructed Labour

BY

HARI CHAND, M.B., B.S.,

R.M.O. Asafabad, the Nizam's State.

DURING my duty as an R.M.O. I came across an unusual case which I send for publication as it may prove interesting to the readers.

A female patient X, aged 18 years, was brought to the hospital on 10-11-1943 at 4 p.m. for obstructed labour since two days. She was full term and was a primipara. After reporting to the hospital a P.V. examination was done.

The results were as follows:—(1) the membranes had ruptured; (2) cervix was fully dilated and the head was presenting at the cervix which was tightly gripping it on all sides. It was impossible to pass a finger along the head inside.

Pelvimetry was done and the measurements were:—(1) Intuspinous measurement 9½"; (2) Intercristal 11"; (3) Ext. conjugate 7½".

The condition of the patient was—pulse 140 p.m., feeble; temperature 101°F.; foetal heart sounds audible.

A soap water enema was immediately given and the urine was drawn off by a rubber catheter. ½ c.c. of Pituitary Extract was given intramuscularly, but to no effect. The pains did not start.

It was then decided to apply forceps.

The patient was brought under chloroform, but it was impossible to introduce the blades of the forceps due to the large size of the caput succedenum which had formed. Perforation of the head was the next resort. The head was perforated by Denman's Perforator, but even then it was found impossible to extract the child. The whole hand was introduced and it was found that the child had rotated and has occupied an R.O.P. position with incomplete flexion of the head. The head was well flexed and with the hands inside manual rotation to an R.O.A. position was done. Forceps were then applied and the child drawn out. The cord

was tied, cut, and the child separated. The placenta separated completely after half an hour. There was much bleeding and the patient was in a very collapsed stage.

*Post-operative treatment:—*After the delivery of placenta Ergotimine Citras 1/100 gr. was injected intramuscularly. Normal Saline about one pint was given by submammary route. As there was much handling, there was great danger of peritonitis setting up. An injection of anti-streptococcal serum (puerperal) 10 c.c. was given intragluteally. A prophylactic dose of anti-tetanic serum 3,000 I. units was also given. The head of the bed was raised three inches and the patient was given hot water bags. The patient slept comfortably that night. From the next day, patient was kept for a week on the following mixture:

R Rum 3 ss. Ext. Ergot Liq. 3 ss. Aqua ad 3 i. T.D.S.

Sulphanilamide tablets were started. She was given 3 gm. Sulphanilamide daily for a week. Plenty of glucose water was also given along with it. The diet consisted mainly of milk, sago, fruit juice and glucose water.

The only complication that occurred in that lady was retention of urine for a day. She made an uneventful recovery. At first there was some small amount of discharge for a few days, but it disappeared on the 4th day. Though she was kept on such high doses of Sulphanilamide, she did not show any signs of toxicity. The patient was discharged completely cured on 18-11-1943.

The points of interest in the case are:—(1) The unusualness of the case as met with in general practice; and (2) rapidity of action and low toxicity of the Sulphanilamide group of drugs.

A Case Report

BY

DR. PANNALALL, L.S.M.F.

H/o. Behrampur Dispensary, Behrampur (via Dinanagar), Dt. Gurdaspur, the Punjab.

ON 14-4-'43 a female aged 20 was brought in a dholi. She complained of severe attacks of pain in the thighs, calves and feet, especially in the soles. There was some swelling of tarso metatarso joints. The patient gave history of syphilis. She had faded marks on the skin of the whole body suggestive of syphilitic rash. The epitrocular glands were also enlarged.

From the slight swelling on the joints I concluded that the whole trouble was due to secondary syphilis. I prescribed on the 1st day Liq. Hydrag. per chlor orally and Ung Hydrag Co massage on the feet. I also asked the husband to procure Sulpharsenol injection.

On 13-4-'43 I gave her an intravenous injection of 12 c.c. of Sulpharsenol. At night she again complained of severe attack of pain.

In the morning, while studying her case it occurred to me that the trouble may not be of peripheral neuritis but due to arsenic poisoning which she may have taken for the treatment of syphilis. I made minute enquiries and was told that she did have a course of arsenic given by a Hakim. She further told that on the completion of the course she got slight constriction of throat (this she had even now), vomiting, little purging. The urine decreased. Pain in the calves. Falling of hair from the scalp (not marked), ulcers on tongue.

My doubt was confirmed. I stopped the injection of Sulpharsenol and started Sodium Thiosulphate injection and Berin. Alkaline mixture by mouth. Saline purges and later on Sodii Thy. Sulph. gr. 15 t. d. s. orally along with the injection. The patient was advised to take liver and plenty of milk and curd.

The pain gradually subsided and the patient began to sleep at night. Previously she could not sleep even with hypnotics. On the 15th day of admission the patient was seen walking erect. She had been bed ridden for the last one month.

By writing this note I just wish to bring to the notice of my colleagues a case of arsenical poisoning in which the trouble would have greatly increased, had the Sulpharsenol injections been continued.

Moreover it will be seen that it is very essential for the doctor to take note of every minute detail of the case and for the patient to relate every minute detail.

Treatment of Snake-Bites

BY

G. VYDIANATHAN, L.M.P.,

Medical Officer, Arumbatur, Trichy Di.

I HAVE gone through carefully with great interest the article written by Dr. BALAKRISHNA MENON on "Treatment of Snake-Bites" which appeared in the March (1943) issue of your esteemed journal and also the subsequent letter of Dr. SHIVAPRASAD K. TRIVEDI published in your April (1944) issue.

The effect of "Tiriyag" is quite gratifying at my hands in a few cases of snake-bite here. My trial of the drug was only very limited and in no case I was let down. This has made me consider "Tiriyag" a specific for snake-bite though the makers of the drug had not chosen to let the formula known.

CASE 1. A scavenger woman, aged 35, was bitten by a full grown cobra within 100 yards of my house. She came running to me with a bleeding wound, extreme restlessness and rapid setting of unconsciousness and she lost consciousness completely in a few minutes. I was able to revive her with the help of "Tiriyag" in about two hours.

CASE 2. A female child, aged 8, bitten by a Russel viper, was brought to me in semi-comatose condition with a drooping head. Under the above treatment the poison symptoms cleared in about 45 minutes and the child came round to normal in an hour thereafter.

CASE 3. A male, aged 40, bitten by snake (variety not known). The patient came to me with a gradual creeping giddiness and feeling of restlessness. Tiriyag was immediately used and he was completely all right in about an hour.

CASE 4. To another male bitten by some snake (variety not known) immediate treatment was given and he did not develop any poison symptom. I can confidently assure the medical practitioners working in snake-infested rural areas to give a trial to this in cases of snake-bite and they will not be sorry for it.

To condemn or to think low of the medicine because it had not gone through a Research Institute, to be labelled, is quite unjust and unfair.

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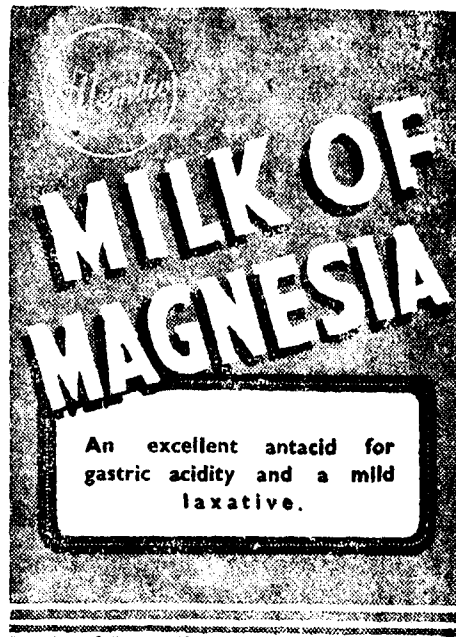
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An Unwise Step

THE Madras Government have issued an order prohibiting medical men in their service from either becoming members of, or accepting office in, the Indian Medical Association and its various branches. The political activities of the main body are assigned to be the reason for this. The order issued is under the Civil Service Regulations by which Government servants are debarred from being members of political bodies whose activities are considered to be detrimental to Government interest.

This order thus presumes, firstly, that the Indian Medical Association is a political organisation, and secondly, that its activities are detrimental to Government interest. With regard to the first, it can be said without fear of contradiction, that the activities of the Indian Medical Association are not mainly political. Year after year, in its annual conferences, the main item of interest is the meetings of the scientific sections, in which medical men from all over the country meet and discuss the improvements in various branches of medicine, surgery, midwifery, etc. A scientific exhibition also forms an important feature of these conferences. The President-elect of the conference and the Chairman of the Reception Committee may, however, in their speeches refer to important aspects of medical science and medical politics. The Indian Medical Association itself meets during these conferences and discusses resolutions only given notice of by the various branches. Most of the subjects discussed are in relation to important problems of the country, not necessarily political though, of course, they may be allied to it. The Indian Medical Association has branches in every Province all over India. Its membership is open to all medical men irrespective of their qualification, or their status or occupation. Private medical practitioners, graduates, or licentiates, members of the Provincial Service, and members of the I.M.S. are all its members. It is true that apart from the Indian Medical Association, there are associations limited to particular groups of medical men. But the Indian Medical Association is the parent organisation and it has no such limitations. Its doors are wide open for any medical man practising his art. It is the only Association which is competent to be

called the Indian Medical Association and therefore to represent the views of medical men of India. It is similar to the British Medical Association of Great Britain, and the American Medical Association of the United States and like them also publishes its own journal which is widely read.

With reference to the second point, that its activities are detrimental to Government interests, it is very difficult to lay this charge against the Indian Medical Association. Its activities have never been anti-government. Many of its members are occupying high positions in government service. Its annual conferences are attended by distinguished men, both medical and others. When the war broke out, its assistance was eagerly sought for the purpose of recruiting medical men to the Indian Medical Service or the I.A.M.C. When the Government of India recently formed the Bhore Committee, it nominated the President of the I.M.A. of that year to the membership of the Committee. One of its past Presidents is now the President of the Indian Medical Council. To the credit of the I.M.A., however, it must be said that in spite of all these preferences it has valiantly fought for the legitimate rights of the Indian medical man. It has striven for uniform medical education and one medical qualification. It has agitated for better prospects and pay for the Indian members of the I.M.S. and the I.A.M.C. It has brought to the notice of the authorities the differences, if any, in the treatment meted out between the Indian and European members of the I.M.S. and the I.A.M.C. It has brought home to the Indian medical man his rights and privileges. It has never, so far, hindered any war effort in this country. On the contrary, if many of the legitimate concessions it had asked for were granted, it promised to guarantee a very large recruitment to the I.M.S. and the I.A.M.C. Nowhere in its activities could it be said that it was against war effort or against Government interest. If fighting for the just cause of the Indian medical men could be termed a crime, then, of course, it should plead guilty to that charge. It is surprising, therefore, that the Madras Government should have banned its servants from becoming members of the Indian Medical Association. It appears to us, a painful contrast to the treatment meted out to Medical Associations in other countries. In England, and in America, recruitment to the medical personnel of the army is entrusted entirely to their respective medical associations—we refer to the B.M.A. and the A.M.A. These associations form their own committees for the purpose of recruiting, selecting and posting these medical men to the various theatres of war with the full co-operation, guidance and support of the governments of their country. But in our country, the one association which can be considered to be equivalent to these Associations in influence and status is not only not given a place of honour in this important aspect of war work, but is banned. This, in our opinion, is a very unwise action. The Madras Government have distinguished themselves by this prohibitory order, for, we learn that no other government in this country have yet dared to take such a reactionary and unwise step.

So far as the I.M.A. itself is concerned, there need be no cause for any anxiety. This extraordinary order will, we are sure, serve as a stimulant to its members who are not in government service to try and bring within its fold all medical men who have so far held aloof and not joined or taken part in its activities. May be some branches will suffer pinpricks at the hands of local satraps as the D.M.O. of a district in our

province who, we learn, from a local daily, tried first to disaffiliate the branch from the I.M.A., and not succeeding in this move, dissolved the meeting and asked the members to clear out of the premises of the Headquarters Hospital and even refused to take tea with its members. Such cases of denial of even the elementary courtesies will, we trust, be only the exception rather than the rule.

We have no doubt that members of the medical profession will rise to the occasion and join the association in large numbers and make its strength and influence felt. But we still have a lingering hope that the Madras Government will, before long, realise the unwisdom of their action and retrace their steps.

Admissions to the Medical Colleges

ONE more year has passed and we have seen the extraordinary rush and bustle of the week preceding admissions to the medical colleges in this Presidency. The position seems to deteriorate from year to year, and never was the rush so great or the disappointment so keenly felt as in the present one. About 300 were selected from about the 1,500 applicants who sought admission. This year about 45% were selected from Tamil Nadu, another 45% from the Andhra districts, and 10% or so from Malabar and South Kanara, and in this selection, candidates were selected according to the district from which they came—each district being given a certain quota—and on the top of all these, communal proportions were applied in making the selections. The obstacles which a candidate had to surmount before he got admission were indeed very great and probably discouraging even to the stout-hearted. This year, so far as the Brahmin community is concerned, only graduates who had obtained either a first class or a very high rank in second class in the B. Sc., examination were successful in getting admission. All others, however deserving they may otherwise be, were not selected. Even first class Intermediates with Group II had no chance. We do not think the position is so acute or that deserving students are refused admission in any other province.

What then is the remedy for this sorry state of affairs? It is, in our opinion, the duty of the government to open more medical colleges and also to increase the number of seats in every one of the existing colleges. We are sure with a little more economy the present colleges could at least take 10 or 15 more candidates. Want of medical men is keenly felt by the country and by the government. It may be argued that, during the duration of the war, it will not be possible to open new colleges, as it will be very difficult to get the necessary equipment and personnel. Buildings formerly occupied by medical schools still exist in Tanjore and Guntur. The personnel for pre-clinical years are still available for re-distribution and could be utilised. Equipment could be made available from the existing colleges with a little more inconvenience to the present students, or they could perhaps be obtained from America on the 'Lease Lend' basis. The government will be earning the gratitude of the public and the students and helping themselves if they take this bold step even during these difficult days.

A word about the method of selection. Would it not be possible to reserve at least 20 seats in each medical college to be filled in by candidates according to the rank they occupy in their University examinations, irrespective of caste, creed and nationality? The rest may be filled up by communal rotation. It should also be possible to reserve at least 10 seats in each medical college for sons of medical men who have served their government and their country well. There is such a thing as tradition, and no government can deny that. So far as the medical profession is concerned, tradition has a great deal to do in the making of a successful medical man. We hope the Madras government will give due consideration to these suggestions. It is no good banging the doors of medical colleges against students seeking admission and at the same time complaining that there are not sufficient medical men to serve their purpose.

We must however express our admiration of the very strict and impartial manner in which the Principals of the three colleges have made the selections. Our adverse comments relate only to the methods prescribed and we trust the government will come forward to remove what we honestly feel and believe to be a great hardship on the poor students.

The Madras Medical Council

WE congratulate DR. V. RAMA KAMATH, the Editor of our contemporary, *The Medical Practitioner*, on his election as the President of the Madras Medical Council. This is the first time a non-official is elected to that distinguished position, and all honour to DR. RAMA KAMATH as he is also the first Indian to secure the place. Another significant fact that must be remembered is that DR. RAMA KAMATH is only a Licentiate and that he has been elected as the President of a body which contains graduates as well. DR. KAMATH has done very good work as a member of the Medical Council for over 15 years and he has been found to be a great fighter and one who boldly expresses his views. His work in the Council has been well rewarded by his colleagues and we trust he will fully justify it.

Cleanings From Medical Press

MEDICINE and THERAPEUTICS

Intravenous Sodium Salicylate in Acute Rheumatism.—Successful results are claimed by A. Pelle (*Paris Medical*, November 30, 1941, 31, 47, 308; *Abst. Praxis*, December 16, 1943, 32, 882) in the treatment of acute rheumatism by means of intravenous drip administration of sodium salicylate. The solution employed was glucose 4.7 per cent. in which was dissolved 6 to 10 gm. sodium salicylate per litre, the solution being contained in ampoules of 200 c.c.m. with one hooked end for suspension (at about 1 metre) above the injection site and the other straight for attachment to the rubber tube. A drip apparatus is incorporated in the tube; a simple one is a small glass ampoule, either attached directly to the tube or by intermediary connecting link, with a clamp above to regulate the flow. The intravenous needle and tube are autoclaved before use, and great care must be taken to see that the tube is free from air and water. The straight end of the ampoule is filed off and the ampoule attached to the rubber tube; the hooked end is also opened and the ampoule suspended. The patient lies in a comfortable position and the injection, preferably into the vein at the bend of the elbow, is given very slowly, drop by drop, so that the administration of 200 c.c.m. takes at least two hours. The treatment is repeated daily until symptoms disappear. No untoward reactions have been noted by the author in the cases treated. This method of administration is indicated in acute attacks until acute symptoms subside; for those patients in whom large oral doses cause gastric disturbances; cases of acute rheumatism with danger of heart disease; chronic cases with acute "flare-ups," and acute articular rheumatism with clinical abnormalities. Cases of resistant sciatica and persistent rheumatism of infective type are also stated to respond well, and marked success was obtained in a case of severe chorea.—*Practitioner*.

Diagnostic Hints in Heart Disease.—By EDWARD PODOLSKY. (*Extracted from the Medical Record*.)—The outstanding diagnostic sign of aortic stenosis is a small pulse, rising slowly to a delayed summit. The condition should never be diagnosed without this sign.

There is a displacement of the left nipple upward, above a horizontal line drawn through the right nipple in cases of mitral stenosis in youthful patients.

An infected vegetation in the pulmonary artery gives severe substernal pain and may be confused with the pain of coronary thrombosis.

Pain, shock and circulatory failure are the cardinal signs of coronary occlusion.

Auricular fibrillation in Graves' disease is much oftener transient or paroxysmal than that due to other causes.

Organic heart pain, particularly that due to coronary occlusion, occurs much earlier and in greater proportion than is ordinarily supposed.

A sense of substernal oppression, a mild twinge of pain, a paroxysm of dyspnoea, an aching sensation in the arm or sudden feeling of great weakness indicate an obstruction of a coronary twig.

Coronary occlusion is not uncommon in patients with arteriosclerotic disease.

An acute enlargement of the liver suggests coronary thrombosis.

Patients with old coronary closure frequently present a peculiar colour of the face, which has been described as "a leaden tint spread over an earthy hue of skin."

In irregular rhythms, if there is a constant quickening of the heart rate during deep inspiration, auricular fibrillation can safely be said to be present.

Systolic retraction of the left posterior aspect of the chest at the level of the diaphragm is a sign of pericardial adhesions.

A double heart murmur with the stethoscope exerting gentle pressure over the femoral artery is a sign of aortic insufficiency.

Rhythmic jerking movements of the head synchronous with the heart beats are seen in patient with aortic regurgitation.

Rupture of the heart is moderately frequent following thrombosis.

Fatigue is an early sign of the late stages of cardiac failure.

The characteristic murmur of aortic regurgitation begins abruptly in early diastole and fades away. It is usually a long, smooth, loud, blowing and high pitched murmur.

The diastolic murmur in cases of suspected aortic regurgitation should be sought for along both borders of the sternum, especially along the left border, where it is particularly likely to appear in cases of slight regurgitation that did not affect the pulse.

Anginal pain occurs in many cases showing aortic regurgitation.

A diastolic murmur is sometimes heard over the main artery of a limb in cases of conspicuous aortic incompetence.

thorax and abdomen, have been treated successfully according to these principles.—*J.A.M.A.*

Sulfonamides in Peritonitis and Peritonsillar Abscess.—Capus used sulfonamide compounds in 33 unselected cases of peritonitis and peritonsillar abscess. In the first 10 cases sulfanilamide was employed, in the following 19 cases sulfathiazole, and in the last 4 cases sulfadiazine. Analysis of the results of this study lead the author to believe that peritonitis and peritonsillar abscess are due principally to a mixed infection rather than to the beta hemolytic streptococcus. The commonest type of mixed infection was that due to combination of the beta hemolytic streptococcus and staphylococcus aureus alone or with other organisms. Sulfanilamide has a beneficial action, since it causes spontaneous regression in some cases and tends to prevent complications. However, recurrences are common with its use. Sulfathiazole is far superior to sulfanilamide, causing resorption in a greater number of cases, and recurrences are not common with its use. Sulfathiazole has a decided masking action on the symptoms without necessarily preventing progression to abscess formation and so enables one to avoid incision and drainage in most cases of abscess formation, or at least to wait for clearcut evidence of fluctuation before carrying out this procedure. Sulfadiazine is probably the drug of choice, but further study is needed to confirm this conclusion. The dreaded complications of peritonsillar abscess are uncommon when therapy with one of the sulfonamide compounds is employed. Conservative treatment supplemented by use of a sulfonamide compound is far safer and more satisfactory than tonsillectomy.—*J.A.M.A.*

Mycotic Ear Infections.—According to Davis, fungous infection of the external auditory canal is a fairly prevalent condition in tropical regions. He reports observations on 22 patients who were examined and treated during June and July 1943. Three stages of this disorder can be observed. In the first stage the ear feels sore and tender to the touch; chewing may be painful. The auditory canal often contains soft semifluid wax and debris of flecks of a white foamy substance. This stage was not seen in this series of cases, since the soldier usually presents himself in stage 2 or stage 3. In the second stage, which was seen in 16 of the patients, the ear is tender and chewing is painful. The canal is coated and sometimes completely blocked with soft, moist, sebaceous-like detritus, often with a greenish tinge due to a secondary infection with bacillus pyocyaneus. After the ear has been syringed the canal wall is found to be red, with excoriation of the epithelial lining. The drum is not commonly affected. In the third stage the canal is swollen, often obliterated and very painful. The pain is worse at night. Sometimes when the swelling subsides an otitis media is revealed. All patients had a moderate pyrexia. In the

initial stages the ear was syringed and swabbed with alcohol to dry out the canal. Glycerin and ichthammol (10 per cent) tampons were inserted. Subsequently the canals were swabbed with alcohol once a day and painted with carbol fuchsin. When the canals were still "moist" after treatment with carbol fuchsin, sulfathiazole powder was insufflated and the condition rapidly dried up. The six patients in the stage 3 category were admitted to the hospital. Apart from routine treatment, analgesics were given and heat was constantly applied by hot water bags. The average duration of treatment was seven days for cure. In eleven of the specimens submitted for pathologic examination, a fine mycelium was identified (trichophyton). A number of the patients had coexistent mycotic skin disease. Transmission of the infection to the ear may have occurred by towels.—*J.A.M.A.*

Epidermoid Carcinoma of Tibia.—Kraft saw 2 instances of epidermoid carcinoma of the tibia within three months. Etiologically there are two types of invasion carcinoma of the tibia: (1) ulcer carcinoma, which arises within a chronic ulcer of the leg, and (2) fistula carcinoma, which originates in a fistulous tract or osseous sinus. These two predisposing conditions may persist for years without becoming malignant. His first patient gave a history of recurrent ulcers dating back more than thirty years. In the second case there was a chronic osteomyelitis. In both instances the malignant growth developed rather suddenly and progressed rapidly. The first patient came to the hospital after a pathologic fracture had occurred. The second patient lost 20 pounds (9 Kg.) during a period of six months and noticed progressive oozing of the leg and progressive weakness. The correct diagnosis was obvious in the first case because of the far advanced osteolytic process. The second case was somewhat complicated because of a typical osteomyelitis. The x-ray appearance was similar to that reported by Hellner. The prognosis of the condition is favorable even in advanced cases, since distant metastatic lesions have been rarely observed. Amputation of the diseased leg is usually indicated unless an early diagnosis allows more conservative procedures. In chronic osteomyelitis efforts should be made to cure residual active foci. With an increase in secretions and development of pain and hemorrhage periodic roentgenologic and biopsy studies are advisable to determine the presence of an epidermoid carcinoma. Dockerty and Meyerding recently reported 2 cases of adamantinoma of the tibia. They concluded that tibial adamantinomas are merely modified squamous cells which vary considerably in their differentiation into ameloblasts. The fact that adamantinoma is closely related to epidermoid carcinoma finds further corroboration in the following common characteristics: (1) a long history of chronic irritation with episodes of either initial or aggravating trauma and (2) sites of predilection in the tibia, jaw bones and skull.—*J. A. M. A.*

JULY '44]

CLEANINGS FROM MEDICAL PRESS

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Etiology and Treatment of Convulsions during Anesthesia.—Convulsions occurring during the administration of general or local anesthetic drugs are primarily due to interference with or inhibition of cerebral cell respiration. The interference or inhibition is usually caused by (i) frank anoxemia (ii) occult tissue anoxia due to alkalemia and acapnia; or, as when local anesthetic drugs are used, by (iii) the toxic action of the drugs inhibiting the oxidation-reduction enzymes of the tissues. The principal treatment is prophylactic and consists in avoiding open and semi-open administration of ether, particularly when any of the secondary convulsion-producing factors are present, and in avoiding large doses of local anesthetic drugs. Before any operation the patient's urine is usually examined with particular attention to the question of albumin, casts, and white or red blood cells. This urinary investigation should also include a reasonably accurate report on the pH of the urine so that the anesthetist would be warned of an incipient tendency to alkalemia. In non-emergency the NH₃ excretion could also be reported with advantage, and for the same reason.

The actual treatment of convulsions *per se* includes—

1. Immediate inhalation of 5% CO₂ and 95% O₂ by means of a face mask and rebreathing bag.
2. If this does not bring about cessation of convulsive movements within a few minutes, intravenous sodium pentothal in 25% or 5% solution (or hemobarbitone or sodium amytal, should be administered in addition to the inhalation therapy described above, and in a dosage adequate to control the convulsion.
3. As soon as the convulsion is brought under control an intravenous infusion of glucose saline (10% glucose in normal saline) should be started for the purpose of promoting renal function and restoring the normal kidney mechanism of H₂O adjustment (that is, the elimination of excessive basic phosphate and bicarbonate via the urine) one litre of glucose saline might well be given to an adult and 500 to 700 c.c. to a child. Once the convulsion is under control the anesthetic and operation may be continued, with the early termination of the latter always in mind.
4. If facilities are available the patient's blood pH and/or the pH of the urine should be checked within an hour after the convulsion ceases, and CO₂ and O₂ inhalations should be given to the point of hyperpnea or for 5 minutes every half hour until such time as the blood pH is below 7.5 or an acid urine is being secreted. The reaction of the urine is not as accurate an indication of alkalemia as the blood pH.
5. Before the patient is returned to bed 2 to 3 dr. of ammonium chloride in six times that volume of water should be instilled by gastric tube in the stomach.
6. As soon as the patient is able to

swallow he should be given cool drinks freely, but in small quantities at one time. The basis of the drinks should be physiological saline. Prior to this rectal saline is indicated.

7. The patient must be kept cool—if possible, in an air conditioned room; or, failing that, an electric fan may be played on face and torso.

8. If his temperature remains higher than 102°F. the atmosphere is warm and possibly humid, a wet sheet may be placed on the patient's thorax and abdomen and a stream of air directed thereon from an electric fan.

9. If his temperature is persistent higher than 102°F. and respiration continues to be rapid, cold, 5% glucose saline enema may be administered in addition to the measures mentioned above.

10. Inhalations of 5% CO₂ and 95% O₂ must be instituted on the first sign of any facial or other twitching.—*P.M.J.*

Enuresis.—A meeting of the Section of Urology of the Royal Society of Medicine was held on Feb. 24, with Mr. E. W. Riches in the chair, to discuss Enuresis.

Mr. T. Twistington Higgins said that enuresis was a symptom, not a disease. It was defined by Meredith Campbell as "the involuntary and usually unconscious voiding of large amounts of urine which occurs predominantly during sleep in the absence of organic disease of the nervous or urological system." The word "unconscious" seemed to exclude children who were deliberately dirty in their habits; these could not be excluded. The definition made no mention of age, but enuresis should not be spoken of until the child was 3½ or 4 years old. The ordinary healthy infant properly trained might be expected to be dry at 18 months, and if between that age and 3 the child was perpetually flooding, something was likely to be wrong. Spina bifida occulta must be considered. If the spine of all children were examined by x-rays a proportion—estimated by some authorities to be 35%—would be found to have fusion defects, and, particularly if these were in the sacral area, spina bifida occulta might be the cause of enuresis. Approximately one case in ten would be found to have some organic disease of the urinary tract. The organic disease to be expected were urinary tract lesions in the tract or almost all due to obstruction in the tract or infection, or a combination of the two. Careful examination should also be made of the external genitalia. In girls vulvovaginitis was the commonest lesion. In boys the state of the external urinary meatus should be noted to ascertain whether it was contracted, ulcerated, or misplaced. One of the many traditions about the prepuce was that it must be amputated to stop bed-wetting, but he had never seen a prepuce, however tight, concerning which he was satisfied

that it was a cause of urinary obstruction. Circumcision had no place in the treatment of enuresis. The condition of the bladder and the question of residual urine should be taken into account; a complete urine analysis should also be made. If this preliminary examination indicated a urological basis for the enuresis the child should come under treatment by the urologist, but if nothing abnormal was revealed the enuresis might be assumed to be functional. For functional enuresis institutional treatment of the proper type was invaluable, but not all children with bed-wetting could or should be treated in an institution. Sociological implications could not be ignored. The remedy was in better and surer homes and more intelligent, wiser, and less harassed parents.

Mr. H. P. Winsbury White said that during wartime a great increase in this condition was to be found in males, especially adolescent and adult males. He mentioned the value, after excluding extra-urinary conditions, of dilating the contracted urethra. As such dilatations would often have to be repeated it was wise to make this perfectly clear at the outset. The minimum period between dilatations should be one month. Prolongation of the individual treatment by the use of the indwelling catheter for half an hour, and its removal before the patient awakened from the anaesthetic, could be carried out in certain cases. Chronic frequency of micturition when present usually improved quickly from dilatation.

A classification of cases:—Dr. W. Shelton, as practising paediatrician, said that enuresis was by no means confined to the poorer section of the community; it was found in all social grades. In young children it was equally divided between the sexes; in the second half of childhood it was more common in boys and girls. Among 5,000 children aged between 5 and 19, and living in L.G.C. institutions, the incidence of enuresis was 5%. The classification he had found useful was: (1) cases associated with organic disease of urinary tract; (2) cases associated with disorder of health in some other system of the body; (3) cases associated with mental retardation; (4) functional cases, persisting from infancy or reappearing in later years. In the first group there was usually diurnal as well as nocturnal enuresis of very great frequency of micturition. He had yet to see a boy whose case he could justly be attributed to phimosis. In the second group many conditions might act as contributory factors, including disorder of the alimentary tract. Adenoid enlargement causing snoring and disturbance of sleep should be considered. He would reserve ephedrine for the treatment of enuresis whose condition was associated with allergic rhinitis. The use of drugs in the third group was universally disappointing. Something might be done with children in this group by bribery in return for a dry bed. In the fourth group it was useful to withhold fluid after tea and

forego any meal at bed time; also to see that the child made a real effort to empty the bladder before going to bed, and was roused again after four hours to pass water. The attitude of the home must be encouraging. Praise for a dry night was better than scolding for a wet one. The raising of the bed so that the hips were above the level of the shoulders was sometimes useful. There was another group consisting of sensitive and intelligent children, who stood particularly in need of sympathy and encouragement; and lastly, there was a small group of enuresis whom the war had brought into notice—those who intentionally wetted the bed to gain some advantage, such as transference to a fresh billet.

Dr. E. B. Strauss considered that enuresis belonged to psychiatry more than to urology. There was an atavistic pull-back to the original watery environment of the race. Another important factor was the extreme sensitiveness of the kidneys and bladder to emotional stimuli. He doubted whether spina bifida occulta was found more often among enuretics than among those who had developed normal bladder control. When pills and lotions effected a cure they worked through their suggestive rather than their pharmacological properties. He had treated a group of children with a belladonna and potassium citrate mixture and a control group with burnt sugar only, and had got the same proportion of cures in both. Secondary enuresis might respond well to hypnotic suggestion. Mr. E. W. Riches said that the finding of spinal fusion defects in a child with enuresis was not to be taken as an assumption that the defect was the cause of the enuresis. It was an indication for further investigation, which should be carried out with the cystogram and the cystometrogram. By these means, with cystoscopy, of course, it was possible to discover which bladders showed signs of disturbance of nerve function. If these cases were left he was sure that they would go in progressive stages to the complete incontinence of senility.

Wing Chair, J. C. Ainsworth Davies found that indigestion was an important exciting cause. He had seen enuresis associated with gastric and duodenal ulcer, and when the ulcer improved the enuresis cleared up. One way to distinguish the malingering from the true incontinent was, on applying the urethroscope and fixing on applying the internal meatus, to watch the floor of the internal meatus, to watch the roof, which ordinarily could not be seen unless the urethroscope was pulled right down to an angle of more than 45 degrees. If however, the roof simply flapped into view he thought the case was likely to be one of true incontinence—that is to say, a relaxed internal sphincter. He also raised the question of the possibility of plastic operations. Mr. F. McG. Loughnane said that he had no experience of operating for enuresis. In examining these children with the cystoscope he had never found an abnormality in the bladder or neck of the bladder. If the children were of the age of

about 12 he used the cystoscope without giving any local anaesthetic and in a large number of these cases whether as the result of suggestion or not, they did get better quickly. One of the most disappointing patients was a boy who at the age of about 14 was at a public school, where the matron was always holding him up as a bad boy on

account of this habit. He had no abnormality. The speaker saw the boy when he was about 18 and sent him to a psychiatrist, who said that he had a fear complex which would take a very long time to resolve. He believed psychiatry to be indicated more than surgery in the treatment of these cases.—B.M.J.

OBSTETRICS and GYNÆCOLOGY

Leucorrhœa.—By Herbert E. Schmitz, M.D. (Condensed from the "Medical Clinics of North America," Jan. 1943).

Leucorrhœa is an expression of some underlying disorder, either functional or organic, and may occur at any age. The diagnosis and treatment of various types will be discussed.

Diagnosis.—A complete history is essential to arrive at a correct diagnosis. Information as to exposure, habits, home surroundings, marital history, personal hygiene, and contraceptive practices may lead one directly to the underlying cause. Systemic diseases, such as diabetes, nutritional disorders and cardiovascular renal disease, must be eliminated, since these general conditions may cause an irritating vaginal discharge.

When the patient is being prepared for the pelvic examination, care must be exercised not to disturb the vulva until the examiner has noted the type and quantity of secretion bathing the genitalia. A smear and drop of this secretion should be taken for study. The character of the discharge is of importance; for example, a blood-tinged discharge in a woman beyond 40 years of age may be a symptom of carcinoma somewhere in the genital tract. A mucosanguineous discharge is associated with cervical polyp, submucous myomas and hyperplasia of the endometrium. Clear mucous, viscid or thin, milky and non-irritating, is found with erosions of the cervix, pelvic congestion and retro-displacement of the uterus. Frothy, bubbling or thick creamy fluid is found associated with infection by the trichomonas, yeast organisms causing crusts on the vaginal walls. Gonococcal infections, infections by non-specific organisms, pyometra and septic vaginitis produce a yellowish discharge. A foul odour associated with vaginal secretion usually means death of tissue, as in carcinoma or sloughing myomas. Foreign bodies such as neglected tampons, pessaries and other objects inserted through ignorance or in attempts at masturbation, may also be the cause.

The diagnosis depends on a careful history, digital examination, inspection with adequate illumination and magnification if necessary, smears, cultures, hanging drop examination, and a determination of the hydrogen ion concentration of the vaginal secretion. The latter determination may be made quickly by means of nitrozone paper.

Leucorrhœa of vaginal origin.—Tricho-

monad infection: This is the most common cause of an irritating leucorrhœa. By mixing with warm normal saline solution a drop of the secretion removed from a speculum or the examining finger, and placing a drop of this mixture under a cover glass on a glass slide, the trichomonads can be identified by their characteristic flagellar movement. It must be remembered that lubricants and antiseptics stop this activity of the protozoan and therefore should not be used when making such an examination.

During treatment sexual contact must be prohibited for trichomonads occur in the urethra and prostate of 5-10% of men. Thorough cleansing of the vagina with diluted tincture of green soap, followed by drying with warm air, tends to alleviate symptoms rapidly. This must be followed by some form of treatment aimed at reducing the normal hydrogen ion concentration of the vaginal secretion from 6.5 to 7.5 as found when trichomonads are present, to the normal of 4. Although many expensive preparations are available, applications of milk sugar followed by daily lactic acid douches, 1 dram to 2 quarts of water, has given us the best results. If trichomonads are present in the stools, viovorm or stovarsol should be administered by mouth. Treatment should be carried on through 3 menstrual periods with applications to the vagina being made daily at first and at least 3 times a week after subsidence of symptoms. The douches should be used daily and the patient must be instructed to elevate the hips and close the entrance of the vagina about the douche tip so as to distend the folds of the vagina and thus dislodge all the organisms.

Vaginal Monilia:—This occurs more often in pregnant women because of the increase in glycogen and vaginal acidity which accompanies gestation. Treatment should be intense, to relieve symptoms and prevent infection of the fetus. The vagina is thoroughly cleaned, then painted with 1% aqueous gentian violet, Lugol's solution or 50% tincture of iodine if tolerated.

Vaginal infection due to Vincent's organisms or various types of spirilla and spirochaetes is usually associated with and favoured by uncleanness. In cases of fusospirochælosis the discharge is profuse, thick, yellow and purulent.

Treatment consists of thorough cleansing of the vagina with a saturated solution of sodium perborate. The patient is instructed to use a sodium perborate douche.

Neo-arsphenamine, 10% in glycerin, may be used as a daily topical application.

Treatment of acute gonorrhoea should be absolute rest without local therapy, as the latter only tends to hasten the upward extension of the organism through the cervix. Sulphathiazole in a dose of 2 gm. followed by 1 gm. every 4 hours day and night has given the best results. Such therapy must be continued for from 5 to 8 days, with constant vigilance being maintained for toxic manifestations of the drug. External cleansing of the vulva, with careful instruction to prevent self-contamination or spread of the disease, should be part of the routine. When the acute symptoms have subsided, intravaginal douches with antiseptic solutions will tend to lessen the discharge and cleanse the vagina of cellular debris.

Leucorrhoea in children occurs commonly but is often times neglected because mothers hesitate to permit their children to submit to examination and some physicians hesitate to make adequate examinations. The cause may be a specific or non-specific infection, or as Allen found in 5 per cent. of his clinic cases, a foreign body in the vagina. In specific vulvo-vaginitis the sulphonamide compounds are replacing other therapy. Although children tolerate the drugs well, great care must be exercised not to carry this therapy too far. In persistent cases or in those patients not tolerating the sulphonamide, the use of oestrogenic suppositories brings good results. If re-infection occurs, the cervix should be examined through a vaginoscope or urethroscope to determine whether the cervix may be harbouring the organism. In non-specific vaginitis, good results are again obtained with the use of the female sex hormone in the form of small vaginal suppositories.

Senile vaginitis due to hypo-oestrogenism is often accompanied by a purulent discharge and severe pruritis. If it is untreated, ulcerations and adhesions of the vaginal membranes may result. Oestrogen is a specific cure for this condition and may be given orally or in suppositories.

Leucorrhoea of cervical origin.—The more common lesions of the cervix include erosion, laceration, polyps, strictures and stenoses, and less frequently leucoplakia. In 85 per cent. of patients presenting themselves for one of the above lesions, leucorrhoea is the major complaint. Erosions must always be differentiated from carcinoma. For this cervical biopsy is essential. Tuberculosis of the cervix is rare. Syphilis of the cervix may be diagnosed by finding a sharply punched-out ulcer with a grey base. Darkfield examination must be made.

Treatment of cervical lesions involves prophylaxis. Improved obstetrical techniques decrease injury to the cervix. Routine postpartum examinations should be made, 50 per cent. of cervixes being pathologic 8 weeks postpartum. Treatment at this time prevents serious complications at a later date.

The purpose of treatment is (1) relief of

abnormal discharge, (2) eradication of cervical infection, and (3) anatomic restoration of the cervix. Topical applications are of little value except in acute erosion or mild endocervicitis. Postpartum erosions and ectropion respond well to Negadol or mild silver preparations, the more severe cases demanding cautery. The use of chemicals in treating the diseased cervix is to be condemned because of the high incidence of stenosis and strictures following their use. Surgical procedures are rarely necessary. If the lacerations are deep, trachelorrhaphy is indicated. If the erosion, ectropion, or endocervicitis is severe, then amputation, preferably by the Strumderf technique, is warranted. Polyps of the cervix are removed by twisting them off after cauterizing the base. All such specimens must be microscopically examined, because occasionally one finds early malignant change.

Cauterization with the actual cautery has proved most satisfactory in our hands for the treatment of erosions, ectropion and mild endocervicitis. Too deep cauterization must be guarded against, but proper technique requires deeper scarring within the canal than at its portal, so that during the process of healing the everted cervical lips are drawn inward, thus restoring the normal appearance of the cervix. If frequent dilatation is practised for several weeks following the use of the cautery, stenosis or stricture need not be feared. Following cauterization the cervix should be inspected on about the 8th day, since the slough is loosening at this time and some bleeding may result. It is our practice to have the patients take douches of normal salt solution twice a day until healing is complete. Healing normally requires from 6 to 8 weeks. If the cervix does not heal within this time, or if it heals and the scar breaks down without apparent reason, biopsy or amputation should be performed.—G.P.

Treatment of Essential Dysmenorrhoea with Ethinyl Oestradiol.—The results obtained in a series of twelve patients with essential dysmenorrhoea treated by oral administration of ethinyl oestradiol, a synthetic oestrogen of high potency, are recorded by R. A. Lyon (*Surgery, Gynaecology and Obstetrics*, December, 1943, 77, 657). The ages of the patients ranged from nineteen to thirty-five, and in no case was an additional gynaecological disorder present. Ethinyl oestradiol was given orally in dosage of 0.05 mgm. once daily, beginning at least twenty-one days before the menstrual period and continuing for twenty-four days. The general plan of treatment was oestrogen for two successive cycles, and then one menstrual period without treatment. Some alteration in the length of the cycles was noted during oestrogen treatment, i.e. the duration of bleeding was 1.8 days longer and the flow was less profuse. In consequence the succeeding cycles, particularly when untreated, were usually shortened to 25 or 26 days. Forty-four cycles were treated; in six there was

no bleeding and either an additional 24 tablets of ethinyl oestradiol were administered after a free interval of one week or a normal menstrual period without treatment was allowed. In the latter event dysmenorrhoeic cramps invariably occurred. Toxic reactions in the treated series were nil, and the treatment was well tolerated and liked by the patients. As the basic principle of the treatment is the temporary suppression of ovulation the importance of instituting treatment at least three weeks before the menstrual period should not be overlooked. None of the patients treated failed to ovulate when the treatment was withheld. Prolonged ovarian rest or suppression of the

follicle-stimulating hormone has been shown generally to be undesirable: the method therefore is advocated not as a cure but as providing a temporary relief in intractable cases of dysmenorrhoea, and also, as these patients usually show immature development of personality, the method was found to inspire the necessary confidence which is frequently lacking. The patients in the reported series were under observation for from six to fifteen months. In view of the potency of ethinyl oestradiol results can be obtained with low dosage. The method of two treated cycles and one untreated is advocated in order to preclude persistent oestrinism.—Practitioner.

Correspondence

Peri-Articular Arthritis of the Shoulder-joint

To The Editors, THE ANTISEPTIC.

Sirs,

My article on Peri-Articular Arthritis of the shoulder-joint was published in Jan. '43 issue of the Antiseptic. A recent article on Humero-Scapular-Peri-Arthritis in the March '44 issue of the Indian Medical Gazette describes the same disease with an interesting anatomical and pathological explanation. The disease has received attention in Europe at the hands of Duplay in 1872 and of Codeman in 1906 in America. Since then other physicians and surgeons have worked on the subject from therapeutic and pathological points of view.

Restriction of movements in abduction, extension and circumduction, pain over the greater tuberosity of humerus extending down the arm which has to be kept in the adducted position, peculiarity of age incidence invariably above 40, no occurrence of complaint in any other joint and

ultimate recovery after some months are all points which make up an entity of the disease. The anatomical and pathological explanation of Dr. Politzer is an additional feature to treat this arthritis as a separate disease and deserves a special mention in text books.

It is very often mistaken by medical attendants for a common chronic arthritis and not much attention is paid to it by them. From its great inconvenience and discomfort to patients this peri-articular arthritis deserves wide recognition by the profession. My experience tells me that it is a much more common complaint than is supposed by doctors in India and abroad.

I would, therefore, request you to write a special article on the subject to bring it to the notice of the medical profession and to put up a strong plea to include it in text books.

26th April, 1944 }
Agra Road. }
Nasik.

Yours truly,

Capt. P.S. GUPTA, M.B., B.S.,
Nasik.

News and Notes

Penicillin—A Reply to Critics

A statement "to correct a great deal of inaccurate information which appears to be current on the subject" of the development and production of the new drug penicillin has been issued by the Therapeutic Research Corporation of Great Britain, embracing five big drug houses and two other firms. The statement says:

Recent Parliamentary questions regarding penicillin suggest that the time has arrived for the companies engaged in the development of penicillin production to direct attention to certain facts.

The discovery of penicillin and its development for therapeutic use were both due to British scientists—the first to Professor Fleming and the second to Professor Florey and his collaborators at Oxford. Up to the present, however, penicillin can only be manufactured by biological, as distinct from chemical, methods and procedures are highly

technical and complicated. Penicillin itself is of an unstable nature. Thus, although its manufacture both in Great Britain and America is now developing on a commercial scale, there is as yet no absolute certainty that each batch will be of the same standard activity.

Professor Florey's results stimulated research work by a number of scientific workers in both the industrial and academic fields. Indeed the research leading up to the large-scale production of penicillin may well be claimed as an instance of collaboration without parallel in the pharmaceutical industry. For example, the Therapeutic Research Corporation of Great Britain (which was formed in 1941) had already established a pooling of research effort between Boots Pure Drug Company Limited, the British Drug Houses, Limited, May & Baker, Limited, Glaxo Laboratories, Ltd., and the Wellcome Foundation, Limited.

Imperial Chemical (Pharmaceuticals), Ltd., who had been researching into these problems, and Kemball Bishop & Co., Ltd., who had long experience in the manufacture of products by mould fermentation, completed the industrial pool of skill and experience directed towards the problem of the manufacture in Great Britain of penicillin in quantities adequate eventually for the nation's need.

Meanwhile, other eminent academic workers, notably Sir Robert Robinson, Professor Heilbron, and Professor Raistrick, with their associates, have collaborated in full measure.

Exchange of Information:—Subsequently, the Medical Research Council in this country and the Committee on Medical Research in the United States arranged for the regular exchange of information between teams of workers in the universities and industrial laboratories and other institutions on both sides of the Atlantic.

Finally, the activities of the institutions and undertakings concerned are co-ordinated with the various interested Ministries and Service departments in the General Penicillin Committee of the Ministry of Supply. Anything less like a monopoly has never been seen. There is full mobilization of the appropriate skill and talent possessed by both Britain and the United States for speeding the solution of the problem of manufacture of the supplies of penicillin which are vitally needed.

We trust that this will serve to correct a great deal of inaccurate information which appears to be current on the subject.

Signatories to the statement are: Therapeutic Research Corporation of Great Britain Limited (Boots Pure Drug Company Limited, The British Drug Houses Limited, May & Baker Limited, Glaxo Laboratories Limited, The Wellcome Foundation Limited), Imperial Chemical (Pharmaceuticals) Ltd., and Kemball, Bishop & Co. Limited.

Errata

In Chapter V of the article on "Indian Village Obstetric Emergencies" published in the June issue, please delete the words "which will necessitate a second anaesthetic" after the words "Saxtroph's manœuvre" in page 362, II (i) (b) lines 7 and 8.

21st All India Medical Conference, Cawnpore: The Secretary, Scientific Section, writes:—

The next session of the Indian Medical Association will be held at Cawnpore during the next Christmas week. The members of the Cawnpore Medical Association have taken upon themselves the responsibility of making this Conference a success, counting on the full co-operation of the medical profession in India. The most important side of the Conference is its Scientific Section and the task of organising this Section has been entrusted to me and I have accepted this difficult task in the hope of getting your full support and co-operation.

For the present we have selected the following subjects for which papers and discussions are invited:—

Surgery, Medicine, Ophthalmology and Ear, Nose & Throat, Obstetrics & Gynaecology, Radiology, Pathology and Bacteriology, Tuberculosis, Nutrition and Pediatrics. If you are interested in any other subject the suggestions would be welcome.

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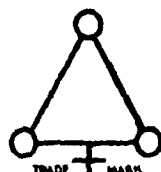
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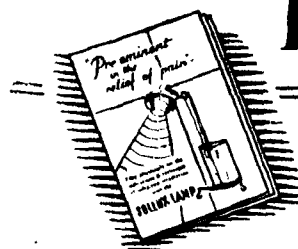
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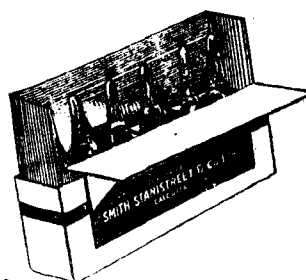
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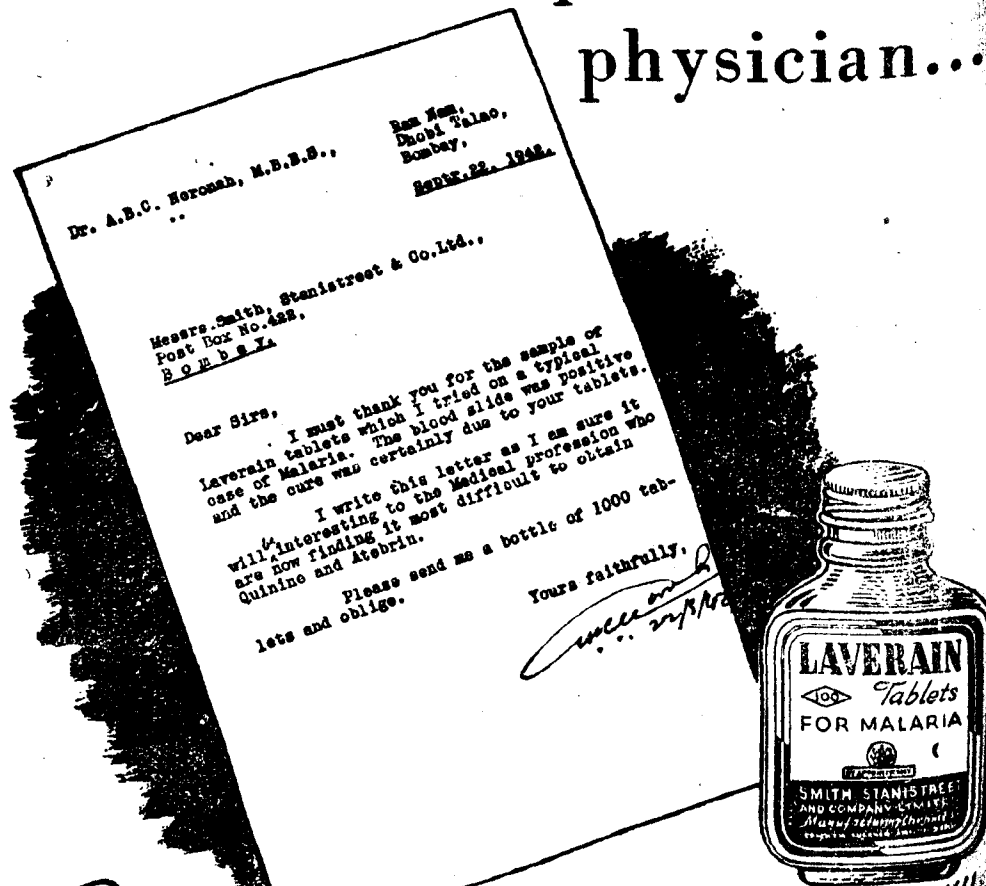
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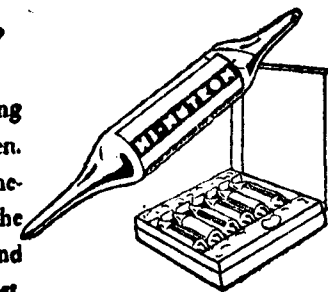
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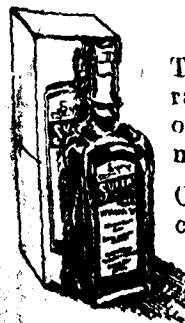
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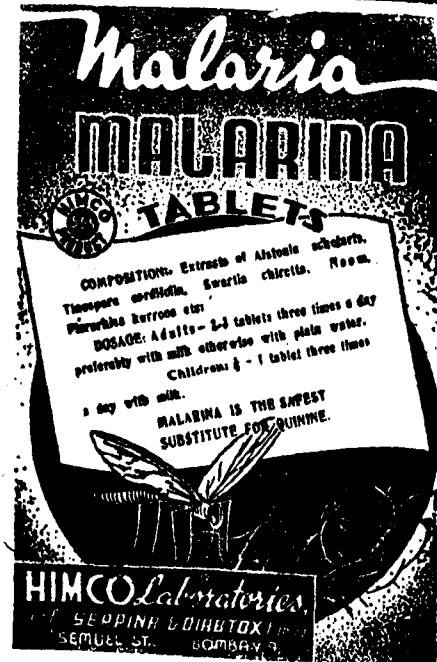
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The following are the minor medical indications for therapeutic abortion, according to Whitehouse (Whitehouse, B. : Abortion : its frequency and importance, British Medical Journal, 2 : 1095 : 1929 : Indications for induction of abortion. British Medical Journal, 2 : 337-341, 1932. Burrell, L. S. T.; Cotton T. S. and Whitehouse, B. : Medical indications for premature termination of pregnancy, Proc. Roy. Soc. Med. 25 : 247, 1931), who enumerates a wide range of conditions that have been claimed as justifying interruption : (1) very recent pregnancy ; (2) general debility with loss of weight ; (3) after suppurative appendicitis that has produced extensive adhesions ; (4) after a previous cesarian operation ; (5) to prevent increasing prolapse of the pelvic organs ; (6) after plastic repair of the pelvic floor to prevent a recurrence ; (7) eugenic reasons such as birth of a defective child or Parental feeble-mindedness ; (8) suicidal tendencies ; (9) economic reasons in women of high fertility ; (10) previous post-partum infection ; (11) co-existing malignant disease ; and (12) necessity for travel to remote regions where pregnancy cannot be properly cared for.

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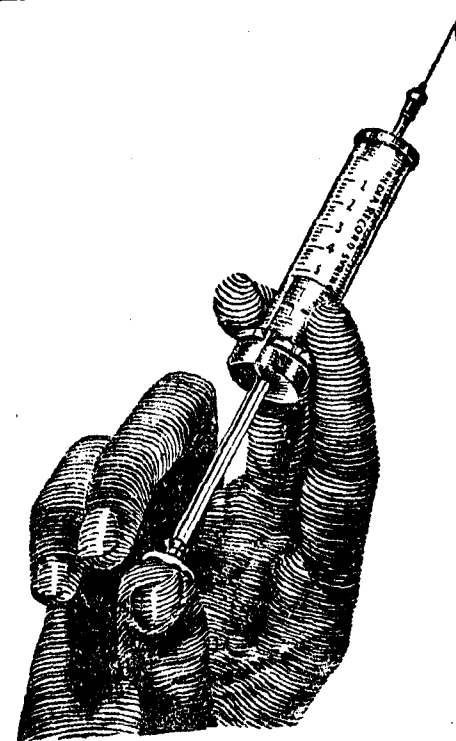
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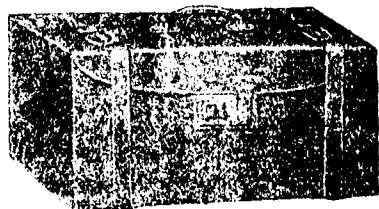
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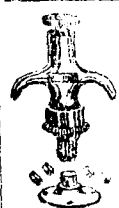
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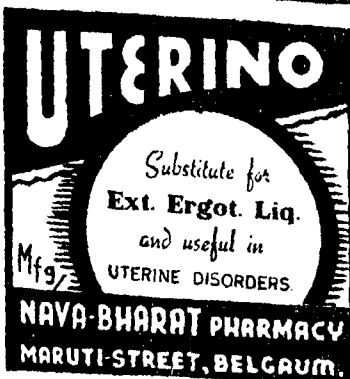
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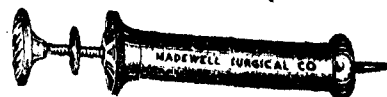
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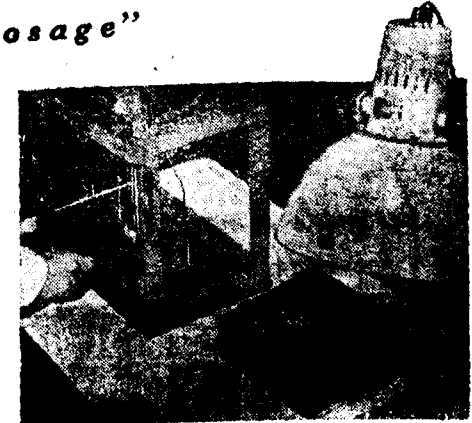
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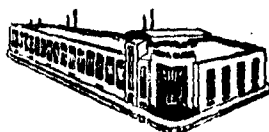
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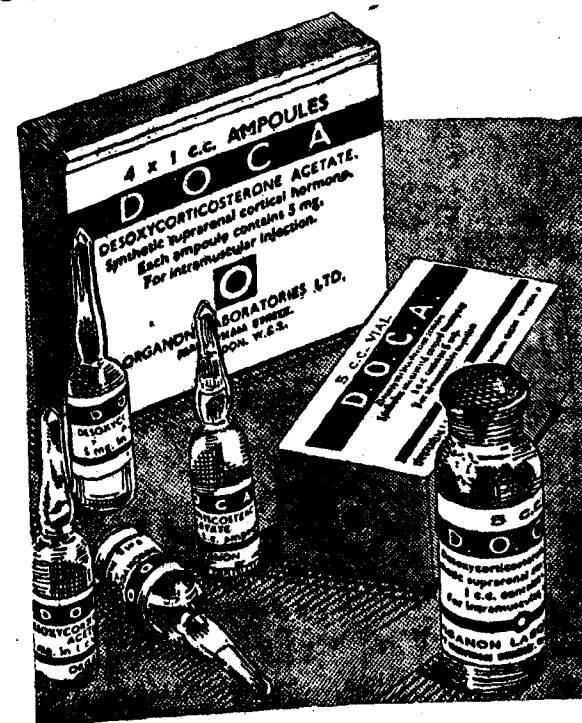
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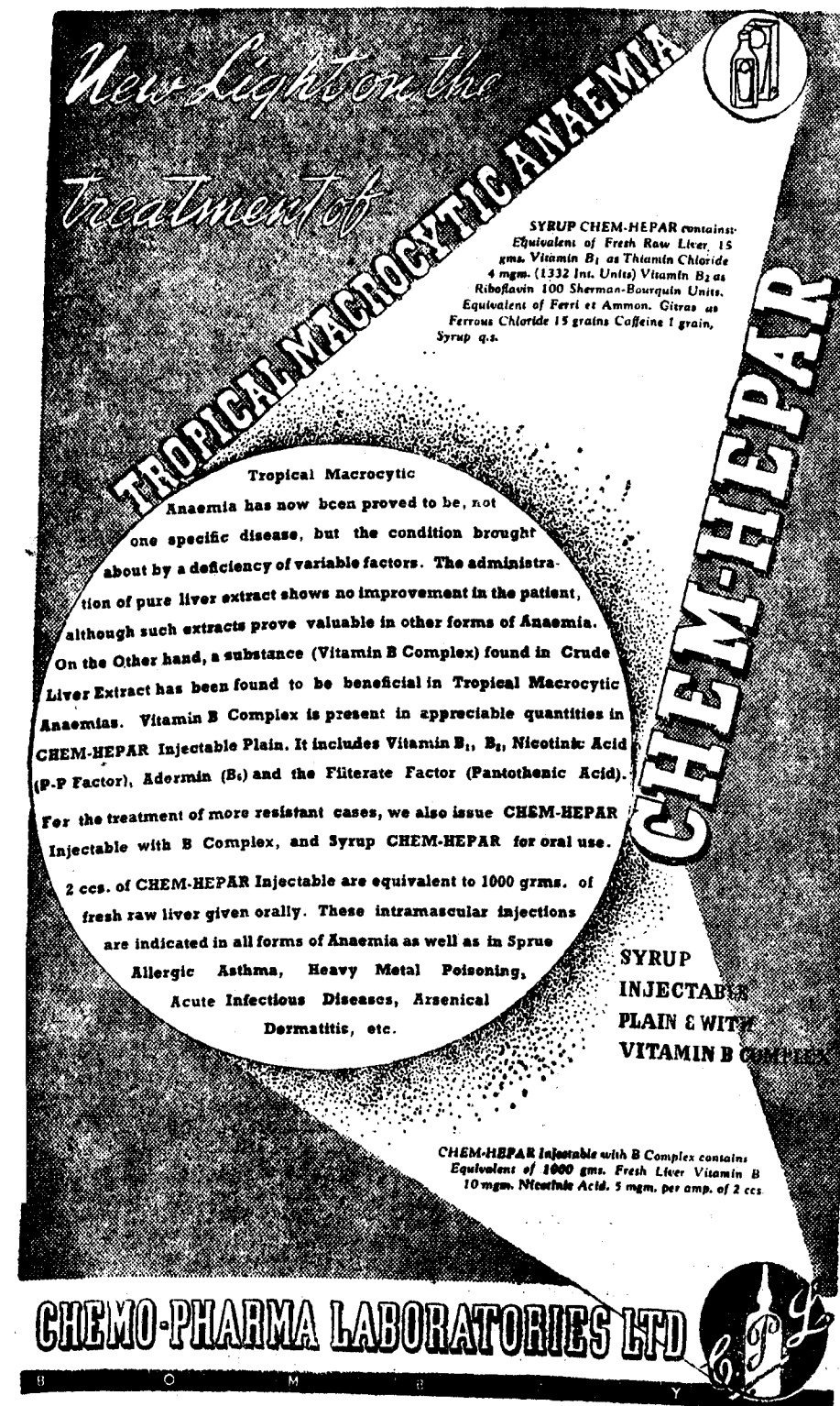
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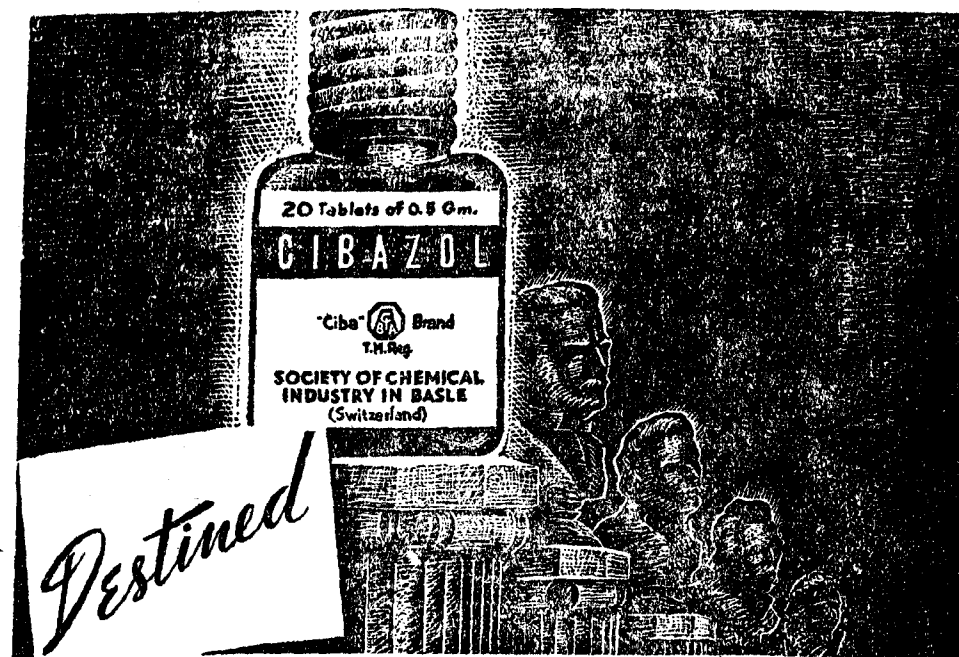
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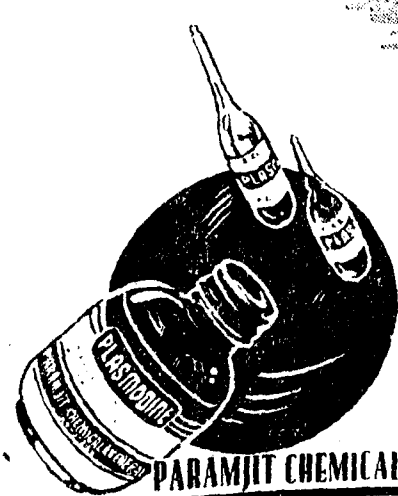
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Original Articles

Piles and Their Treatment*

R. N. RAJU, M.B., B.S. (Andhra), & P. RAMARAO, M.B., B.S. (Andhra),
Qajar Hospital, Podur P. O., West Godavari Dist.

Definition and classification.—Piles or hæmorrhoids can be defined as the varicose anastomosing radicles of the superior, middle and inferior hæmorrhoidal veins in the submucous and subcutaneous tissues of the lower end of the rectum and anal canal. These are mainly of three varieties: (1) Internal, (2) External, (3) Interco-external.

Important points in surgical anatomy.—The anus and lower inch or two of the rectum are supplied by three main arteries: (1) superior hæmorrhoidal, (2) middle hæmorrhoidal, and (3) inferior hæmorrhoidal. In this portion of the gut, unlike in other parts of the alimentary canal, these vessels lie longitudinally along the bowel wall and connected by transverse branches to form a plexus around and just above the anus. Superior hæmorrhoidal artery divides first into two main branches: (1) right, and (2) left. The right branch again divides into two, an anterior and a posterior. The corresponding veins follow the same course. This is the reason why we see three primary internal piles at 3, 7 and 11 o'clock positions, with the patient in lithotomy position. The five secondary internal piles that we see generally are roughly at 12, 1, 4, 8 and 9 o'clock positions and will correspond to the situation at which branches are given off by those three vessels. The superior hæmorrhoidal vein is a tributary of the portal vein and the middle and inferior hæmorrhoidal veins are tributaries of the systemic veins. Thus this venous plexus around the anus and lower end of the rectum forms one of the most important anastomosis between the portal and systemic circulation.

In addition to these facts, the situation of these veins in the loose sub-mucous tissue with inadequate support, the valueless nature of the superior hæmorrhoidal and portal veins, the erect posture of the human being getting always this venous anastomosis into the most dependent position, and lastly the unavoidable exposure of this portion of the gut to

* Specially contributed to 'The Antiseptic.'

sudden variations of pressure before and after defaecation will explain the frequency of this disease occurring in human beings.

Pathology. An external pile is a subcutaneous hæmatoma or subcutaneous thrombotic vein, which, after organisation, may leave a tag of skin containing scar tissue. An internal pile is a distended venous plexus in the loose submucous tissue along with an arteriole. The mucous membrane covering the pile, when it gets prolapsed, is denuded; and as the pile mass prolapses more and more fibrous tissue will grow with the necessary arterial supply and the columnar epithelium covering the pile will turn into stratified epithelium. The bleeding from the internal pile is either from the main arteriole or from the arterioles of the fibrous tissue or from the distended venous plexus, which, because of exposure to constant injury and pressure, changes during and after defaecation yield and bleed through the denuded mucous covering.

Causes of piles. Having considered the anatomical peculiarities, we can classify the causes into the following groups:

- (1) Any cause that produces obstruction to the portal circulation. { Cirrhosis liver, secondary deposits liver, splenic anaemia, etc.
- (2) Any cause that produces obstruction to the normal pelvic circulation. { In the bowel, cancer rectum outside the bowel, pregnant uterus, enlarged prostate, ovarian tumour, etc.

- (3) Any cause that produces obstruction to the systemic circulation. { Heart failure.

(4) *Idiopathic cause*.—This is always associated and aggravated by chronic constipation where the constantly loaded colon produces obstruction to the flow of circulation in the lower part of the rectum and anal canal causing piles. Once piles are formed, a vicious circle develops i.e., constipation producing piles, and dread on the part of the patient to timely emptying of the bowels, due to the associated bleeding and pain, thus causing much more loading of the colon.

Sex and age incidence. More common in males than in females. Age varies between 25—55 years.

Relation to occupation.—Persons who leads to sedentary life are specially predisposed to this disease.

Signs and symptoms.—Frank painless bleeding of fresh blood per rectum either before or after defaecation is the early symptom. Patient may have some discomfort and feeling of heaviness in the rectum. Pain is a late symptom to develop and this is due to gripping of the pile by the external sphincter during the act of defaecation or due to some complication of piles such as abscess or fissure. Pruritis is common in external piles. Anaemia results from chronic bleeding.

Examination and diagnosis. A patient who complains of bleeding along with stools must be subjected to both general and local examination. All the possible causes that produce stasis and varicosity of the hæmorrhoidal venous system must be excluded during general examination. External piles are easily seen and diagnosed. Digital examination alone will not finish local examination because a simple internal pile, not associated with some degree of thrombosis or prolapse, cannot be palpated by a finger. Proctoscopy must be done always to diagnose piles. By this method, piles are visualised as longitudinal, soft compressible, dusky in colour, globular masses in the lower part of the rectum

anal canal. Sometimes they are rough and granular, particularly in chronic prolapsed condition. Sigmoidoscopy, which is not generally done by the average practitioner is really useful to diagnose piles that occur sometimes, high up 3—4 inches above the internal sphincter and to eliminate any malignant disease in the upper reaches of the rectum and in the sigmoid colon. By careful examination it is easy to diagnose piles from other swellings that occur in the region of the anal canal and rectum such as, prolapse rectum, polypus rectum, condylomata, epithelioma, etc.

Complications of piles.—(1) Pain is more a complication than a mere symptom of piles; (2) strangulation; (3) thrombosis, phlebitis, pyeliphlebitis; (4) peri-anal or ischio-rectal abscess; (5) fistula-in-ano; (6) fissure; (7) pruritis ani; and (8) prolapse. This is of four degrees. *1st degree.*—Pile mass prolapses as far as the external sphincter. *2nd degree.*—Pile prolapses out of the external sphincter and goes inside on its own accord. *3rd degree.*—Pile prolapses outside and requires manual interference for restoration inside. *4th degree.*—The prolapsed pile keeps outside and irreducible even by manipulation. Before embarking on radical treatment of piles all the complications must be looked for and treated, particularly any septic condition.

Treatment.—The success of treatment depends on the proper selection of cases. It is always imperative, before radical treatment of piles is thought of, to eliminate any cause to which the piles are secondary and it is futile and sometimes positively harmful to treat a case of piles such as one secondary to cirrhosis liver or cancer rectum, or pregnant uterus, where palliative measures only are indicated. Palliative treatment of idiopathic piles by patent ointments is advocated, but we have little knowledge nor experience of them.

Injection treatment.—Non-prolapsing piles and piles of the first degree of prolapse are chosen for this method. The principle behind the injection treatment or by diathermy coagulation is to cause fibrosis in the sub-mucous tissue and obliterate the pile by thrombosis and subsequent organisation. Diathermy coagulation was the favourite method of Dr. M. G. KINI at Vizagapatam, but we have not heard about his opinion and experience recently on this method. We have not tried this method. The common injection material is 5% Carbolic acid in olive oil with 2 grains of Menthol to an ounce of the solution. It is sterilised by boiling the solution and adding Menthol when the oily solution boils and the flame put out. 2-3 c.c. are injected and one pile is injected at each sitting which is taken once a week.

Requirements and preparation.—An hour or two before the injection patient is given a pint of soap and water enema. The following are got ready absolutely sterile: (1) 10 c.c. record syringe; pile needle, this has a point and shoulder; (2) pile solution; (3) proctoscope; (4) sponge holder; a few swabs, vaseline; (5) towels, gloves, aprons; and (6) a good torch light.

Technique.—No anaesthesia is necessary. After the patient is kept in lithotomy position, the skin around the anus is touched with spirit and under aseptic methods, the proctoscope is gently introduced, rectum and anal canal are mopped dry under good light. The pile masses are visualised by removing the obturator and gently withdrawing the barrel. The necessary quantity of pile solution is taken into the syringe and is injected observing the following points:

- (1) Inject always at the base of the pile and in the submucous

tissue only. When injection is given too low, *i.e.*, beneath the epithelial lining of the anal canal, severe pain is produced and fissure or abscess is likely to occur in a few days. If the injection is given too superficially, a white spot will be observed as soon as the injection is begun and an ulcer will form in course of time at the site. If the injection is given too deep, *i.e.*, in the muscular coat, patient gets severe pain. In short, a properly given injection should cause least hurt to the patient and is rewarded with good result.

(2) The level of the needle should be directed towards the mucosa on the side of injection, rather than towards the lumen of the rectum. With this procedure, the puncture is easily performed and one can feel by practice that the point is in the sub-mucous layer.

(3) Injection is stopped before the tissues become too tense, *i.e.*, when blanching is usually seen with the capillaries standing out on the oily mass which bulges into the lumen of the rectum as injection proceeds.

(4) A proper dose of the injection is important, because if too large an amount is injected, necrosis and sloughing result and if the quantity is small, the results will be naturally poor.

(5) While injecting at an area which was once previously injected, the dose must be smaller because the already fibrosing tissues are less tolerant to the sclerosing fluid than normal tissues.

After the injection, patient is advised to take rest for the remaining part of the day; advised to take 1 ounce of liquid paraffin at bed time, floated over milk. His usual diet is allowed. A liberal fruit and vegetable diet will relieve the constipation which is necessary to be corrected in the habits and life of the person.

Complication of injection.—Massive thrombosis of the hæmorrhoidal plexus may result after overdosage. If the injection is improperly made, complications like superficial necrosis of the mucous membrane, abscess or fissure, are common. In our experience we had no such complications.

Operative treatment for internal piles.—Cases that are unsuitable for injection treatment are selected for operation. Preparation: On the morning of the day prior to operation, the patient takes light diet. Same evening, he is given one pint of soap and water enema and allowed only milk with plenty of sugar. At bed time, he takes an ounce of Tincture Catechu mixture. On the morning of the operation he is again given one pint of soap and water enema. He is allowed to take a cupful of milk or coffee. The perineum and neighbouring parts are shaved, and after cleaning with turpentine, soap and water, are touched with spirit. The area is covered with sterile gauze and cotton and T bandage applied.

Anæsthesia.—Spinal analgesia is excellent for the purpose and we use it as choice for this operation. Percaine 1 in 1,500 in 0.5% Saline 6 to 7 c.c. is given, with the patient in 7½° Trendelenburg position during injection and operative and post-operative period for at least 8 hours. Caudal analgesia by injecting 2% Procaine 20 c.c. into the sacral canal extra-durally can be used. Due to uncertainty of getting the landmarks particularly in women, and the difficulty of reaching the proper space, this analgesia is not quite reliable, and we had disappointing results. Local analgesia does not give relaxation of the sphincter and is not helpful. General anæsthesia—C₂ E₃ mixture or Ethyl Chloride induction and maintenance by ether—is not convenient nor advisable, because the struggling of the patient during induction and recovery and the post-operative vomiting cause congestion of the stumps and possible bleeding. Withal,

the relaxation is not quite good with the sphincter, unless put to deep surgical anæsthesia.

Technique.—After giving spinal analgesia, the patient is put in the lithotomy position with the head lower than the buttocks, the area of the operation is disinfected with spirit and sterilised towels are laid. One observes perfect relaxation of the sphincters under spinal analgesia. One primary pile is taken in an artery forceps. Slight traction in the axis of the pile is applied to enable a firm grip of the pile by another straight long pile is applied to enable a firm grip of the pile by another straight long artery forceps as far high as its base. The forceps is taken in the left hand and with a curved pair of scissors in the right hand the mucocutaneous junction is snipped and the pile is carefully dissected upwards along the submucous layer, under the grasp of the forceps till its point is reached. If the surgeon finds a tag of skin or external pile corresponding to each or one primary internal pile, a racket-shaped incision is made round the external pile with its handle pointing towards the anal margin, the dissection is commenced at the periphery and continued upwards in the subcutaneous and sub-mucous tissues till the point of the long artery forceps is reached. At that level the pile is transfixed with a curved round bodied needle and No. 2 or 3 cat-gut and ligatured with the knot towards the lumen of the gut. A cheap and equally good ligature material is linen thread; No. 40, four card, double layer is used. The pile is cut below the ligature and the stump allowed to retract. A knot is put about 3 inches on the free ends of the ligature and ends cut. All the piles are dissected one by one, thus. Perfect hæmostasis is necessary. Simple twisting of the bleeding points in the external wound will arrest bleeding, keeping the ligatures in their respective positions outside the anal margin, a fairly stiff rubber tube 3" x ½" with a ribbon paraffin gauze wound over its middle is inserted into the anal canal. The purpose of this tube is to facilitate external signs of internal bleeding. Then a flat piece of sterile gauze moistened in sterile acriflavine, 1 in 1000, is tucked into the anal canal all round the tube. A pad of sterile gauze is put with a slit in the centre through which comes out the rubber tube; a safety pin run through the tube, prevents the tube slipping in. A pad of gauze and cotton and a T bandage will finish the operation.

Post-operative treatment.—Trendelenburg position is maintained during the stage of shifting of the patient to bed and on the bed for at least 12 hours after operation. The following mixture is given three times a day for three days.

R Tannic Acid grs. 30, Tincture Opii ℥ 20, Tr. Catechu ℥ 30,
Spt. Am. Aroma. ℥ 15, Aqua ad 3 ℥. 3 I.T.D.

These subsequent three days he takes liquid diet. Post-operative retention is treated by 6th hourly catheterisation. To relieve pain ½ gr. Morphine can be resorted to. If there is much pain and local discomfort, the tube can be removed after 24 hours. On the 3rd day night, he is given 2 ounces of liquid paraffin floated over milk. On the 4th day morning 4 to 6 ounces of warm gingili oil is injected slowly into the rectum by a catheter and syringe; and ½ hour later one pint of soap and water enema is given. After the patient passes a motion, the wound is irrigated with weak Eusol lotion under vision, and dressed with square Eusol gauze. The ligatures slough off and fall off in 5 to 12 days. From the fourth day patient takes his usual diet and 1 ounce of liquid paraffin at bed time. The dressings are changed every time the patient passes a motion and the

nature of the dressing material varies with progress of the granulating wound. Patient is instructed on the general treatment of his constipation and if stricture rectum is expected, dilatation is started from the early part of the 3rd week of the operation.

Post-operative complications. Haemorrhage from the stumps, suppurative pyelephlebitis, formation of peri-anal or ischio-rectal abscess, fissure etc., may occur. In our practice we had none. The development of post-operative stricture depends on the number and nature of piles dissected and the stricture can be minimised by sparing as much as possible linear strips of healthy anal mucous membrane between the contiguous piles dissected.

Treatment of external piles is essentially excision under an anaesthetic. The preparation is precisely the same. General anaesthesia or caudal or spinal analgesia can be used. Local analgesia is better avoided because an external pile is the seat of recurrent inflammations, and to introduce a foreign solution is not only painful but is also attended with dangers of sepsis. Local nerve block in the ischio-rectal fossa is described but we have not tried it.

Operation.—Under all aseptic and antiseptic measures, putting the patient in the lithotomy position and after holding the pile by an artery forceps, it is excised by a racket-shaped incision with its handle pointing towards the anal margin and including a liberal amount of skin allround. Insufficient excision of skin leads to fistula. Ligatures are avoided as far as possible and bleeding is controlled by a hot pack, and twisting of bleeding points. After complete haemostasis is obtained, the wound is packed from the bottom with Acriflavine gauze and bandaged. Post-operative treatment is precisely the same as for internal piles excision. Complications are few.

Summary. (1) A description in some detail is given of piles and their treatment. (2) The relative value of spinal analgesia for this operation is stressed. (3) Treatment of piles is within the reach of every general practitioner if only he is keen on knowing and doing. It is so simple to diagnose and treat piles with a few instruments and practically with no economic burden on the patient. (4) A cheap and useful substitute for cat-gut, i.e., linen thread, No. 40, four cord, double layer in black colour, is suggested.

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Infective Hepatitis

Dr. A. A. Lisney (*Proceedings of the Royal Society of Medicine*, February, 1944) states that the onset is gradual and for several days there are general malaise, headaches, anorexia and perhaps nausea. More rarely coryza or sore throat is present. There is often a slight rise in temperature, but in some cases, though the face is flushed, the temperature is found to be normal. Bradycardia is frequently present. These prodromata are quickly followed by pain in the upper abdomen, usually accompanied by vomiting. Both last for a short time and are followed by epigastric tenderness, which persists for some days. At this stage there may be enlargement of the liver and even of the spleen also. From one to 14 days after the acute symptoms develop, jaundice appears. It usually starts in the conjunctivae and spreads over face, neck, arms, trunk and lower limbs. The faeces are now pale and the urine dark, but these appearances may precede the jaundice. The jaundice is at its maximum in two or three days and lasts for seven to ten days. Itching may occur. Recovery is usually slow and convalescence takes two to four weeks, or even longer.—*Medical World*.

Indian Village Obstetric Emergencies

K. R. W. KINKEAD ALLEN,

Master in the Art of Obstetrics and Doctor in Medicine of the University of Dublin, Poona.

(Continued from page 427 of July 1944 issue).

Chapter VIII Oedema

I. The earliest sign may be swelling of the feet and ankles followed by a spread to other parts of the body. The woman notices that she is passing less urine than normal. The blood pressure is usually raised and albumin will probably be found when the urine is tested. These are the signs of hypertension due to pre-eclamptic toxæmia, essential hypertension or chronic nephritis. If in addition there is headache, dizziness, dimness of vision, pain or a sense of tightness round the lower ribs, nausea and even vomiting, or the woman becomes restless or dull and even stuporous, your patient is in a condition of impending eclampsia, hypertensive encephalopathy or uræmia.

II. Severe anæmia can cause similar symptoms but without a rise in B. P. and a glance at the woman's mucous membrane will be a guide. Severe anæmia may be due to syphilis, worms, malaria or to a shortage of the vitamin "B" group and protein in the woman's diet. There is also a rare serious hæmolytic type as well as occasionally a true pernicious anæmia. Chronic malaria may also, as a result of anæmia, produce the above symptoms without rise in B.P. or rise in the temperature but a thick blood film will show parasites. In Lederer's anæmia there is a rise in temperature.

III. The immediate treatment for mild pre-eclamptic toxæmia, hypertension or chronic nephritis is rest in bed, sedative drugs, restriction of fluid intake to the amount of urinary output and deprivation of all Sodium Chloride even in cooked articles, i.e., if the woman passes 4 ozs. she may only drink 4 ozs. until she again passes urine. The urinary output and the fluid intake must be measured and must be charted until all danger of a relapse is over. As soon as the 24 hour output is known the intake can be planned for the next 24 hours and as the output increases so may also the intake increase for the subsequent 24 hours. If the symptoms are severe treat as for eclampsia [Chapter IX (D)]. In threatened uræmia alkalies should be given by mouth and in a severe case 2% Sodium Bicarbonate or 3% Sodium Citrate up to 2 pints may be required "I.V."

IV. For anæmia prescribe 30 to 45 grains of freshly prepared Bland's pills daily or the iron mixture mentioned below plus food rich in protein and in the vitamin "B" group, such as liver and raw eggs. Worms, syphilis or malaria require attention as the case may be. A good iron mixture is made as follows: Powder one drachm of Ferrous Sulphate in a mortar. Titrate it well with 1½ ozs. of Liquid Glucose. Allow to stand for 10 minutes and then slowly add 1 drachm of Acid Sulph. dil and water up to 12 ozs. The dose is one ounce two or three times a day after food and the mixture must be prepared freshly every four days. Rice huskings from a mill and milk curd are also valuable.

V. Severe anæmia often and hæmolytic anæmia always requires a series of very slow blood transfusions and so may have to be sent to hospital. Some macrocytic anæmias, if not too bad, may be tided over

by injections of very crude whole liver extracts and vitamin "B" group preparations. Foods rich in protein in the vitamin "B" group and in vitamin "C," such as ground up raw liver flavoured with pepper salt and tomato juice, or raw eggs beaten up and hidden in a mixture of orange juice, milk and sugar, must be given. Give also the iron mixture mentioned above.

VI. If there is achlorhydria or hypochlorhydria it is necessary to give dilute hydrochloric acid 5 to 10 minims after meals to obtain good absorption of the anti-anæmia factors.

Chapter IX Fits

I. Fits in which the woman passes through a toxic stage followed by clonic spasm and unconsciousness, in which there may be dilatation of the pupils and conjugate deviation of the eyes with loss of conjunctival reflex, in which the tongue may be bitten and the woman injure herself (and not the onlookers) are due to:

(1) "*Eclampsia*" which only occurs in the 2nd half of pregnancy, is unusual before the 3rd Trimester and is often preceded by the symptoms of pre-eclamptic toxæmia [See Chapter VIII]. The blood pressure is usually above 150 mm. Hg. Albumin is found in the urine *early* in the fit or *before* the fit commences. Ophthalmoscopic examination may reveal retinitis or papilloedema.

(2) Other symptomatic epilepsies due to bacterial or metabolic poisons, acute yellow atrophy of the liver, infective hepatitis, uræmia, malaria, syphilis, hypoglycæmia, alcohol, antimony, arsenic, aspirin, barbiturates, camphor, canabis indica, cantharides, cocaine, copper salts, corrosive acids, cyanides, digitalis, poisonous fungi, iodine, lead salts, nicotine, opium, oxalates, phosphorus, silver nitrate, strychnine and turpentine can occur any time in pregnancy.

(3) Idiopathic epilepsy usually has a previous history.

II. Other fits may be due to apoplexy (usually a sinus thrombosis), trauma, cerebral tumours, acute encephalitis, subarchnoid hæmorrhage, diabetes, vaso-vagal attacks, hysteria or meningeal irritation, and "hypertensive crises."

III. Tetanus and rabies should not be confused with fits. The victim does not lose consciousness.

IV. *Treatment of 1. Eclampsia*, severe pre-eclamptic toxæmia, hypertensive encephalopathy or uræmic convulsions:

(a) Place the woman in a quiet room which must be sufficiently well lighted for you to see the veins in the woman's arms. Cloth may be wrapped round the handle of a spoon and used as a gag to save the tongue from being bitten.

(b) Give "subcute" either $\frac{1}{2}$ grain Omnopon or $\frac{1}{4}$ grain Morphine or 2 to 3 grains of Sodium Amytal or Luminal. This may have to be repeated every 3 to 6 hours. Paraldehyde is possibly a better sedative, especially for the fœtus, than opium derivatives or barbiturates. The disadvantages of paraldehyde are its abominable smell and taste and, if given by Ryle's tube, stomach tube or rectally, the disturbance entailed for the patient. Paraldehyde decomposes on storage unless kept in amber bottles. It remains sterile if kept closed. In urgent cases 5 c.c. may be given "I.V." or 10 c.c. "I.M." and when the patient is tranquil the

balance of 2 to 5 drachms in oil by rectum. The injections may be repeated in 8 hours if necessary.

If given by mouth the dose is 4 to 5 drachms. If given rectally the dose is 5 to 8 drachms in 5 to 8 ounces of bland oil. The bowel should be emptied by means of a normal saline or plain water wash out. The injection should be run into the rectum by tube and funnel fairly rapidly, say in 4 minutes between contractions. Should convulsions or uterine contractions occur during the injection or for 10 to 15 minutes afterwards a pad of wool must be held over the anus and the buttocks pressed together to prevent the ejection of the paraldehyde. The above mentioned dosage should induce response in about 15 minutes and the effects ought to persist for 5 to 6 hours, by which time the original dose or a slightly smaller one may be repeated, if required. There must be no hesitation in repeating the paraldehyde as soon and as often as necessary because large doses are well tolerated and are frequently essential. When ordering paraldehyde for administration by a nurse or an attendant, write the word "*drachms*" in full or order the dose in cubic centimetres to avoid errors.

(c) Give "I.V." 20 cc.m. of 50% Glucose solution and repeat every couple of hours or better give "I.V." 500 cc.m. of 20% to 50% Glucose solution in Pyrogen-free water taking at least 30 to 40 minutes for the infusion and repeat 2 to 3 times a day. In cases of uræmia the Glucose should be added to 3% Sodium Citrate or 2% Sodium Bicarbonate solution in Pyrogen-free water.

(d) Give "I.M." or "I.V." 10 cc.m. of 20% Mag. Sulph solution in water to be repeated after every fit up to 8 doses (be careful of the sterility especially of the "I.M." injection). It must not be used if the woman is in coma. If in rare cases it causes cyanosis, dyspnœa or feeble pulse give "I.V." 10 c.c. of 20% Calcium Gluconate solution or "I.M." 20 c.c. of a 10% solution.

(e) Give 30 grains each of Chloral Hydrate and Potassium Bromide in an ounce of water every 6 to 8 hours. If the woman cannot swallow give this rectally.

(f) *Do not overtreat* with the above if the fits are under control.

(g) If there is œdema give less fluid by mouth and "I.V." than the urinary output. If for instance 4 ozs. of urine is passed or removed by catheter then not more than 4 ozs. (say 100 cc.m. "I.V." fluid + 1 oz. oral fluid), may be given until urine is again passed. A chart must be kept showing fluid output and intake. As soon as the 24 hour output is known the intake for the subsequent 24 hours can be planned. Then give an ounce less than the output unless the climate is exceedingly hot and dry when just sufficient fluid to allay severe thirst may be allowed.

(h) At a convenient moment puncture the membranes provided the external "os" is a couple of fingers dilated. Otherwise empty the bladder of urine and then by means of a long needle passed through the midline of the abdominal and uterine walls draw off 400 to 500 cc.m. of Liquor Amnii. If you possess axis traction for your forceps, as soon as the cervix is fully dilated and the presenting vertex reaches the level of the ischial spines, apply forceps and deliver slowly and carefully. If you are without axis traction wait until the parietal bosses pass this ischial spines before applying the forceps. Leave a breech alone and only interfere if progress is delayed as explained in Chapters IV and V.

(i) If in spite of the above the fits continue draw off 50 to 100 ccm. of C.B.F. and/or, unless the woman is anæmic, 300 to 400 ccm. of blood from a vein. This is of special value in a case of "hypertensive crisis."

(j) As soon as the fits are under control avoid over-treatment by drugs. Rely on rest, restriction of fluid intake to equal the urinary output, deprivation of Sodium Chloride and mild sedation by Chloral Hydrate and Bromide to avoid a relapse.

2. Other epilepsies or fits should be treated according to their cause i.e., malarial convulsions or coma is treated by the "I.M." injection of 0.3 gramme of Atebrin Musonate (Quinaerine Soluble, Mepacrine Methane-sulphonate) dissolved in 9 ccm. of warm distilled water, this dose is to be repeated on the following day if necessary or an interval of one day between injections may be permitted. It is advisable to follow up the injections by a course of oral Atebrin totalling 1.5 grammes which with the injections brings the grand total to 2.1 grammes. If the woman is very small and light in weight the dose may be reduced slightly.

In apoplexy, which is usually due to a venous thrombosis, enforce absolute rest, and give diffusible stimulants especially Alcohol and Glucose. For restlessness prescribe bromides. Avoid purgatives and rely on enemata. Watch the urinary bladder. If there is long coma nasal feeding may be necessary. To avoid overlooking meningitis and malaria lumbar puncture and blood films are required.

Chapter X. Abdominal Pain

I. Pain in the abdomen followed by first one and then more of the following:—Shock, collapse, fainting, nausea, vomiting, rapid pulse, fever, constipation or diarrhoea may be due to a variety of causes, but unless the cause is very obvious and the case typical it will usually be essential to tabulate information on the following points before being able to reach a diagnosis:—(1) Name, nationality, caste or religious denomination. (2) Age and occupation. (3) Physical and mental condition. (4) Particulars of digestion, defæcation, urination, and menstruation. (5) Exact description of the onset of the present trouble. (6) Temperature, pulse and respiration. (7) Inspection of the chest and abdomen in a good light. Do not forget to look for a scar on the labia. (8) Palpation. The superficial lymph glands everywhere and the hernial orifices must not be forgotten. (9) Percussion of abdomen and chest. (10) Auscultation of abdomen and chest. (11) Test of reflexes. (12) Examine the urine for reaction, smell, colour, sugar, albumen, acetone, deposits, after centrifuging and sodium chloride content. (13) Examine the gums and tongue and notice the smell of the breath. (14) Thick and thin blood films should be taken and searched for malaria parasites and circulating diplococci. (15) The stools should be examined for entamœba histolytica and for ova of worms. (16) Rectal and vaginal examination. (17) Progress of the case, especially the pulse rate.

A. Conditions affecting the uterus:

1. Sudden displacement of the gravid uterus early in pregnancy is said to produce acute pain and collapse following a history of a jump or other violent exercise. On vaginal examination the uterus will be found in an abnormal position and should be replaced.

2. Abruptio placentæ.
3. Abortion or miscarriage.
4. Acute hydramnios.
5. Hydatidiform (vesicular) mole.

} See Chapter VI.

6. Perforation or rupture for the uterus. See B (iii) below and Chapter III, 19, but if the patient is not in labour there will be no cessation of non-existing uterine contraction, otherwise the symptoms are similar.

B. Abdominal cavity emergencies: (Mnemonic *tophi* for torsions, obstructions, perforations, hæmorrhages and inflammations):

1. *Torsion*:—Little or no shock, followed by gradually increasing pulse rate, distension, tenderness and fever. A swelling can often be felt if due to an ovarian cyst or tumour, a pedunculated myoma, the omentum, the gall bladder, a Meckel's diverticulum or a length of intestine.

2. *Obstruction*:—Colicky pain, shock, collapse, constipation, sub-normal temperature, pulse weak and slow at onset, followed later by gradually increasing vomiting and pulse rate. Except in mesenteric thrombosis, increased peristaltic sounds will be heard on auscultation. Obstructions are caused by external and internal herniæ, intussusceptions, new growths, stenoses, foreign bodies.

3. *Perforation* or rupture of a hollow viscus: (a) When due to disease, sudden agonizing pain, and severe collapse—Patient lies very still and pulse rate increases. Rigidity and absence of peristaltic sounds on auscultation are almost immediate signs when the intestinal canal is involved, but are of more gradual onset when other organs only are ruptured. (b) When due to *injuries* there may be a latent period before the above signs commence to develop. This is specially true of rupture of the spleen and in perforation of the uterus early in the pregnancy in which case there may be no signs until *inflammation* sets in. Later in pregnancy rupture of the uterus at the site of an old scar, or as a result of external violence, or during labour is liable to cause immediate and severe symptoms of perforation but without rigidity. Later follow the symptoms of hæmorrhage. There may be some escape of blood "P.V."

4. *Hæmorrhage* due to spontaneous or traumatic rupture or perforation of a viscus or blood vessel. If the bleeding is slow and continuous, the initial attack of pain, etc., is often followed by a deceptive latent period during which symptoms are slight or even absent. Typically the pain is colicky, abdominal tenderness becomes extreme and the pulse rate increases. Peristaltic sounds gradually disappear, while pallor, restlessness, sweating, air hunger, abdominal resonance with dullness in the flanks and possibly tetany and shoulder pain become noticeable. Rigidity is usually absent or very late in appearing.

5. *Ruptured ectopic gestation sac*:—The woman may or may not consider herself pregnant owing to menstrual irregularity. There may have been colicky lower abdominal pain for a few days. Suddenly the woman feels pain in the hypogastrium and possibly on top of the shoulders. She may vomit and faint and develop all the signs and symptoms mentioned above, or if the bleeding takes place into the broad ligament there will be temporary recovery. There may be slight bleeding per vaginam and examination reveals a tender, possibly boggy swelling in one

fornix while the cervix may feel softened and on being moved cause pain. The uterus may be slightly enlarged.

6. *Inflammation of the appendix, gall bladder, fallopian tube, pancreas, intestine and peritonium*—The initial pain and vomiting may pass off for a few hours but the pulse rate commences to *increase*. A stabbing or aching pain grows accompanied by vomiting. Owing to collapse the temperature does not rise much. Tenderness and rigidity develop followed by distention. Peristaltic sounds disappear.

C. *General diseases* :—Malaria, syphilis, early pneumonia, or diaphragmatic pleurisy, pneumothorax, coronary thrombosis, pericarditis, angina pectoris, acute gastro-enteritis, bacillary dysentery, abdominal influenza, enteric fever, amœbic typhilitis and hepatitis, constipation, intestinal colic, worms, biliary colic, renal and ureteric colic, Dietl's crisis, pyelitis, uræmia, diabetes, pancreatic colic, acute adrenalitis, Addison's disease, mesenteric adenitis, splenic or liver embolism, heat cramps, visceral neuralgia (due to herpes zoster, migraine, spondylitis, angina pectoris and fibrositis), hysteria, retro-peritoneal hæmorrhage (due to scurvy, trauma, hæmophilia or sarcoma), contusions of the abdominal wall, thyrotoxicosis, hypoparathyroidism and Waterhouse-Friderichsen syndrome in meningococcal meningitis.

II. Treatment.—A. Conditions affecting the uterus :

1. Sudden displacement of the gravid uterus should be replaced bi-manually. Sometimes this can best be done in the knee chest position.
2. Abruptio placenta See Chapter VII (III).
3. Abortion or miscarriage do (I).
4. Acute hydramnios do (III) (b).
5. Hydatidiform mole do (II).
6. (a) *Perforation of the uterus* early in pregnancy is nearly always due to attempts at abortion. If the patient cannot be sent to hospital a prophylactic course of Sulphathiazol should be given as well as tetanus anti-toxin and gasgangrene anti-toxin (Chapter I, 19). The perforation will heal if she survives the sepsis. The pregnancy terminates or goes to term. (b) *Rupture of the uterus* later in pregnancy or during labour requires hospital treatment. (See Chapter III, 19).

B. *Abdominal cavity emergencies* call for removal to hospital. To ward off shock "I.V." Glucose and, if it is going to last more than a couple of hours, Hypodermic Morphine should be given before the journey commences. The effect of the Morphine will then have worn off by the time the surgeon sees the patient. In any case no competent surgeon will operate upon a patient who is in a state of shock. If you are certain the case is one of salpingitis conservative treatment at home is permissible. Some cases of appendicitis and many of cholecystitis will resolve at home but there is always the risk of perforation and the patient will stand the journey better before she perforates than after she has perforated.

C. *For the treatment of general diseases* you must consult a text-book of medicine and see Chapter XII.

(To be continued).

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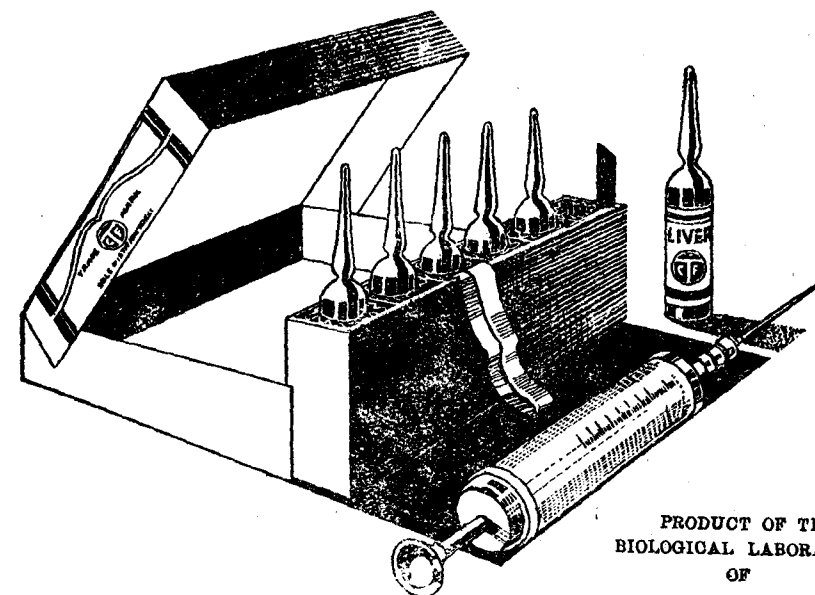
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Some Pelvic Conditions that Cause Acute Abdominal Pain and Their Diagnosis*

DR. KRISHNABAI PATIL,

Lady Resident Medical Officer, Shree Sayaji General Hospital, Baroda.

Introduction.—Acute pain in the abdomen is caused by many pelvic conditions and one should know how to diagnose them correctly.

Pain in the lower abdomen that is in the pelvis is associated with tension in the pelvic organs. Usually it is the result of overfilled vessels, or in other words of congestion. It may be due to actual inflammation that, is congestion due to infection. It is acute and very severe and at times it will be referred to all over the abdomen; or it may be of dull aching character. It is elicited by pressure which makes it worse. In its worst form, it is of peritoneal origin. It may be due to simple congestion of uterus, tubes or ovaries without infection or with evidence of actual inflammation.

Superficial pain on the skin of the abdomen.—The pain is sometimes felt on the skin in the ovarian region which is supplied by the tenth dorsal nerve. It may radiate all over the abdomen, groin, crests of ileum down the thigh. The pain may come not only from the ovaries, tubes or uterus but also from the kidneys, ureter, gall-bladder and some parts of the intestine. Diagnosis of the pelvic conditions can be made by P. V. examination.

At times the referred pain in the ovarian region is found specially on the left side between the umbilicus and the anterior superior spine of the ileum which cannot be explained when the actual examination is made. It may be due to severe hysteria. There is extreme hyperesthesia in this region accompanied with anaesthesia of the skin of the legs and feet upto the knee joints with brisk knee-jerks and absence of the palate-reflex.

Actual pelvic conditions are divided into: (1) Obstetric; (2) gynaecological; and (3) other conditions.

Obstetric conditions.—1. *Abortion*:—Threatened abortion—When the abortion is to take place in (1) acutely ante-flexed under developed uterus; (2) in the uterus with defective musculature, specially in cases of congenital malformations of the uterus as uterus bicornis, uterus unicornis, uterine septum, uterus didelphys etc.; (3) hypertrophic elongation of cervix, stenosed os-cervix or atresia of cervix or cervical fibroid. There will be acute spasmodic pains in the lower abdomen due to painful uterine contractions which may be mostly rhythmic and sometimes continuous; but they are not as acute as in the case of tubal pregnancy. There may be vomiting and slight rise of temperature and pulse. The extra strain is due to the weakness or incapacity of the uterus to throw out the contents or due to the obstruction of the abnormalities of the cervix that are already mentioned.

CASE REPORT OF CANCER CERVIX WITH PREGNANCY:—Patient Shanta C., aged 31 years, was admitted in the maternity wards, S.S.G.H., Baroda, on 12-1-44 with the history of four months' pregnancy and severe uterine pains since three days. Diagnosis of threatened abortion was made. The patient was put to bed and uterine sedatives were given. Uterine pains continued and bleeding started on the 16th. Spontaneous abortion was expected. Nothing happened till the 20th; so P.V. was made and cervix

* Specially contributed to 'The Antiseptic.'

was found one finger dilated and rather rigid. Uterine pains ++. Again time was given till the 23rd but no progress was noticed. Cervical condition was the same. The uterine fundus was pushed high up and the lower uterine segment was much stretched. The ovum could not be reached through the cervix. Evacuation of uterus was decided and the ovum was removed with great difficulty. When the speculum was put at the operation an irregular growth of the cervix bleeding easily was noted. A piece came out easily from the growth. This may be the cause of non-dilatation of cervix. If this case had been left alone still longer expecting spontaneous abortion, the uterus would have been ruptured. At present she is alright and is under observation.

Diagnosis of abortion can be made: (1) By the history of amenorrhœa; (2) by the subjective and objective symptoms of pregnancy; (3) by P. V. examination when bulky uterus with uterine pulsation is found; and (4) later on by noting the products of abortion.

2. *Ruptured tubal pregnancy*:—Diagnosis—There may be acute pain in the abdomen first on one side and then all over. The patient is shocked and collapsed with the accompanying symptoms due to internal hæmorrhage. The abdomen is distended and tender and the blood pressure is low. There is primary anæmia and leucocytosis upto 30,000. When the rupture is small, the hæmorrhage may occur slowly. There is pain in the abdomen, but not as acute as above. Later the pain becomes dull and of aching type. Blood is collected in the pouch of Douglas. Temperature may be raised, pulse full, abdomen rigid and tender. There may be slight uterine bleeding in both and a decidual cast may come out. On P. V. there will be severe pain in the pelvic region and soft mass will be felt in the pouch of Douglas due to pelvic hæmatocele. Definite diagnosis can be made: (1) By missing of a period or two; (2) by previous history of septic puerperium, pelvic inflammation or tubal pregnancy; (3) by subjective and objective symptoms of pregnancy; (4) by pain in the pelvis on the movement of cervix and blood in the pouch of Douglas; (5) before rupture the spindle shaped pulsating mass in connection with the tube is found on one side; and (6) Aschheim Zondak's and Friedman's tests for pregnancy being positive.

3. *Rupture uterus due to pregnancy in one of the underdeveloped horn of the double uterus or in the uterus bicornis will give the signs and symptoms of acute abdomen.* Uterine bleeding with signs and symptoms of pregnancy will settle the diagnosis.

4. *Threatened rupture of uterus in full term pregnancy cases*:—When in labour will give the symptoms of acute abdomen. *Diagnosis*—(1) The uterus will be enlarged and very tender to touch; (2) the abdominal wall will be reduced; (3) there will be tonic contractions of uterus; (4) RX retraction ring above the pubis will be seen; (5) the left round ligament will be found prominent; and (6) there will be disproportion between foetal head and pelvis or there will be abnormal presentation.

5. *Incarceration of the retro gravid uterus, the fundus of which is incarcerated behind the sacral promontory, may produce the symptoms of acute pain in the abdomen.* Here the symptoms are more pertaining to the bladder and bowel. There is traction on the bladder and the urethra by the upward growth of the anterior uterine wall producing great congestion above the trigone and at the urethral sphincter. There will be pressure

of the cervix also, which is pushed up behind the symphysis pubes. The bladder may reach to the umbilicus and the patient may come for the retention of urine or for incontinence of retention. The pain may be due to the strain of retention. Later, rupture of bladder and peritonitis with extravasation of urine may occur and signs of toxæmia may be added. At times the thinned out anterior wall of the uterus may rupture while attempting to expel the ovum. Due to pressure on bowel the symptoms of partial obstruction may occur. Diagnosis can be made: (1) By signs and symptoms of pregnancy, and (2) by P. V. examination. The cervix is pushed directly behind symphysis pubes while the dense fundus is felt in the pouch of Douglas. The promontory is felt buried in the uterine wall; distention of the bladder will need catheterisation before P. V. examination. Pregnancy after ventral fixation may give such symptoms.

6. *Axial rotation of pregnant uterus* is a very rare condition. Usually uterus is found dextro-rotated in many cases but sometimes exaggeration of the same condition may occur to complete twisting. If gradual, no symptoms will occur, but if sudden symptoms of acute abdomen may come and the case may become fatal. It is supposed to be due to a symmetrical musculature of uterus. The diagnosis is easy and does not need description.

7. *Impaction of pregnant uterus* may occur when there is ovarian tumour or other tumours of pelvis. There will be acute pain in the abdomen and vomiting, tenderness and rigidity in the pelvic region. Abdominal palpitation will show irregular tender fixed mass instead of showing the regular top-shaped uterus of pregnancy. There will be long history of the tumour in the pelvis and the recent history of pregnancy with the tumour on the particular side will settle the diagnosis.

8. *Incarceration of the gravid uterus didelphys*:—A non-pregnant uterus didelphys may be pushed at the back of the pregnant one and incarceration in pelvis may occur. This will give rise to acute abdominal pain, vomiting with frequency of micturition and constipation. Diagnosis can be settled with 1. History of the amenorrhœa; 2. with previous history of uterus didelphys; and 3. P. V. which shows 2 vagina and 2 cervixes; pregnant uterus will be found with its own cervix and the other cervix will be continuous with the tumour at its own back. Gauze packing of the uterus after operation and after pains of delivery also give pain in the abdomen.

Gynaecological conditions.—Pelvic inflammation with pelvic peritonitis will cause acute pain in the abdomen. There will be severe pain in the hypogastric region, swelling and rigidity. The abdomen is very tender to touch. P. V. examination shows tender and doughy masses in the pelvis. Uterus and adnexa cannot be separated from the mass. Sometimes the thing gets localised and abscess formation takes place in the pouch of Douglas. There will be history of puerperal sepsis, septic abortion or gonococcal infection.

1. *Acute salpingitis*:—There will be pain first in the uterine and tubal region, and then it will spread all over. In the acute condition no swelling will occur, the pulse will be fast but not as fast as in appendicitis. The pain will be more on one side than on the other. History of gonorrhœa, puerperal sepsis or septic abortion will be found. Later the condition

may get localised in the pelvis and irregular masses may be felt per abdomen on both sides below the umbilicus. P. R. and P. V. will show irregular tubal ovarian masses on both sides of the pelvis or behind the uterus.

2. *Rupture of hydrosalpinx and pyosalpinx* also may give the same kind of symptoms. Pyosalpinx with threatened or actual rupture will cause extension of inflammation to the peritoneum and give signs of acute abdominal pain. The swelling is found in one or both broad ligaments, or tubal masses will be found adherent at the back of the uterus which then becomes very painful. Purulent discharge. P. V. is present. The history of trauma with P.V. may settle the diagnosis.

3. *Axial rotation of the ovarian cyst* may be missed for retro-gravid uterus. There is acute pain in the abdomen with vomiting, which may or at times gradually. The symptoms appear with pain and tenderness in the abdomen and distension. Temperature first subnormal, may rise a little, pulse fast. If tumour is known to be there and if the patient is examined again, its size is increased due to congestion and hæmorrhage inside, and the diagnosis also will be certain. The rotation is common with moderate sized non-adherent tumour. It is common after painful micturition, defæcation, by fall or muscular exertion, and during pregnancy and puerperium. The pain may subside after treatment. If there is no history P. V. examination will settle the diagnosis. The symptoms of the incarcerated retrogravid uterus are already given.

4. *Rupture of ovarian cyst* may occur by fall or blow on the abdomen or violent straining at the stools or other causes which increase the intrasystic tension. The thin follicular cysts are easily broken while doing P. V. examination. When rupture occurs there is a sharp pain over the affected side and vomiting, and then the pain gets generalised due to peritoneum reaction and involvement. Further complications depend upon the contents of the cyst. If watery it will be absorbed and the pain will subside. If mucous, pseudomyxoma peritoni and if papilliferous, papillary growth in the abdomen may occur. There may be free fluid in the abdomen and the patient may die later on.

5. *Graffian follicle and corpus luteum cyst*:—There will be severe pre-menstrual and inter-menstrual pain on the affected side. Ovarian dyspareunia. Freely mobile semi-elastic lemon or orange-sized tumour pediculated from the uterus may be found by P. V. Rupture may occur with intra-abdominal hæmorrhage which may closely resemble ectopic pregnancy and diagnosis may be difficult and sometimes it will be diagnosed at abdominal operation only. Hæmorrhages are common with corpus luteum cysts.

6. *Ovarian hematoma*:—Sometimes acute effusion of blood occurs in the stroma of ovary which may be due to trauma or endometrioma of ovary. There will be acute pain and swelling of the abdomen and signs of peritoneal irritation. P. V. examination will show that the ovary is enlarged and tender and if hæmorrhage is still continued, it will be found increasing more and more till the bleeding stops. Endometrioma is a disease caused by endometrial implants from the uterus. Small irregular chocolate-coloured cysts with peritoneal irritation are found in the ovary. There is history of pain at every menses as the endometrium in the ovary acts in the same way as in the uterus at menstruation.

7. *Axial rotation of the pediculated sub-peritoneal fibroid of uterus*:—The stalk being very thick, it is rare; but very rarely sudden rotation may occur with acute abdominal pain. Sometimes uterus gets also twisted and supra-vaginal amputation of uterus may occur. Rotation of fibroid is common with pregnant uterus. If the uterus is partially rotated menstrual fluid may be obstructed in the cavity and signs of hæmætometra with acute pain in the abdomen may come on. A diagnosis can be made with previous history of fibroid tumours, with menorrhagia and metrorrhagia and P. V. examination with hard tumour besides the uterus will settle the diagnosis.

8. *Expulsion of fibromyomas from uterus* may cause acute pain in the pelvis and abdomen. Fibromyomas are mostly intramuscular to begin with; then they become either subserous or submucous. Those which come under the endometrium of the uterus leave their capsule and tend to become polypoid and are covered with endometrium, which supplies them with blood. In the uterus they act as foreign bodies. The uterus contracts and tries to throw them out. At the time of menstruation these contractions are spasmodic and more painful than at other times. Diagnosis can be made by the history of metro and menorrhagia. P. V. will show the uterus bulky and at times irregular. The fibroid may be felt through the dilated cervix which may again be missed for abortion, but in case of abortion there will be history of amenorrhœa, there will be other signs of pregnancy, pregnancy tests will be positive and microscopic examination of the tumour will help.

9. *Dysmenorrhœa in women* may cause acute abdominal pain with vomiting with slight rise of temperature and at times diarrhœa. The pain of spasmodic type either begins before and lessens after the free flow of menses as in the acutely ante-flexed uterus with cervical stenosed and with congenital malformations.

In congestive type dysmenorrhœa, as in cases of retroflexed uterus and cases with the history of tubo-ovarian inflammation, the pain begins before and continues till the menses stops and little after.

In membranous type of dysmenorrhœa the pain will stop with the expulsion of the endometrial cast of the uterus. The diagnosis can easily be made by finding the above causative conditions. The pain is mostly in connection with the menses.

10. *Functional neuralgia of pelvis*:—Women suffering from severe hysteria and neurasthenia may complain of severe pain in the lower abdomen.—A young woman once came for the treatment of debility and slight pain in the abdomen in private practice. P.V. was made and nothing abnormal but slightly tender uterus was found. Milk injections and general tonics were advised which she was taking. After a month the patient came with severe pain in the hypogastrium. P.V. was made again and nothing abnormal was found. After going through the history it was found that she had recently lost her child and since then she was getting hysterical fits and pain in the abdomen. It was nothing but a case of functional neuralgia.

11. When the bladder is fully distended and the uterus is already in the first stage of retroversion, sudden complete displacement with flexion may occur as a result of heavy lifting and of unusual exertion, when there will be acute pelvic distress with vomiting and other symptoms

which will subside within a few days, when the uterus is restored to its normal position.

12. Advanced cases of carcinoma of cervix may also give rise to acute abdominal and pelvic pain, which can be diagnosed by: (1) P. V. and speculum examination; (2) by its occurrence mostly in middle aged and old women; (3) by foul discharge, loss of weight, anæmia etc.; and (4) by its short history.

Other conditions.—1. Appendicitis:—Appendicitis is at times found to be overhanging on the pelvic brim and to lie in close contact with the tube and ovary. Sometimes the right tube and ovary are inflamed due to spread of infection from the appendix and then it is difficult to diagnose between the morbid conditions of these organs. The attack of acute appendicitis may be mistaken for tubal pregnancy. Appendicitis can be diagnosed by: (1) the history of previous attacks if any; (2) in addition to the signs of acute abdomen there are toxic symptoms also; (3) on careful examination there is rigidity of rectus muscle in its lower half and tenderness at the Mackburney's point; (4) if the condition does not become very acute to become gangrenous or perforative and if there is time for protective adhesions, the swelling in the right iliac region will be seen; (5) while appendicular abscess develops specially in the pelvic type of appendix cases, the pus may gravitate into the pouch of Douglas which condition may simulate the tubal rupture with hematoma. But in the tubal pregnancy there will be uterine bleeding and history of amenorrhœa with pregnancy tests positive. Still the diagnosis may be confusing and laparotomy only settles the diagnosis.

2. *Spastic constipation* will give rise to acute pain in the abdomen specially in the pelvic and perineal regions. In this there will be a long history of attacks off and on, no occult blood and the hard stools will be felt through the rectum and in pelvic colon. Spastic constipation is sometimes found in the neurotic women and the pain is diagnosed from the other conditions by the shreds and coagulated mucous which will be passed along with the stools.

3. *Stricture of the rectum* due to malignant disease is found in the middle aged old women after giving strong purgatives as Mag-sulph. There will be sudden congestion and blockade at the sight of the stricture which will give the signs of acute abdominal pain. But the short history of blood and mucous per rectum, loss of weight, anæmia and P. R. examination and X-ray with barium enema will settle the diagnosis.

4. *Pelvic kidney*:—The kidney is sometimes misplaced in the pelvis and will give rise to pain in pelvis and abdomen, specially at the time of menses, pregnancy and parturition. Diagnosis can be made by finding a kidney-shaped tumour on the sacral promontory or on one side of pelvis. There will be absence of kidney on the same side. If the same kidney gets diseased there will be symptoms in the urine. Tumours of the pelvic organs as ovarian cysts can be excluded by P.V. examination. X-ray may help. By air inflation the rectum may be seen to pursue an abnormal course. Very short ureter is found on ureteral catheterisation. Psychic disturbances are common. Sometimes the diagnosis is so difficult that it can be settled only after opening the abdomen.

5. *Acute cystitis* will cause acute abdominal pain specially in the

hypogastric region. There will be high fever, frequent and painful micturition. There may be blood, pus and epithelial cells in the urine. The blood count is normal. There is no leucocytosis. History of previous catheterisation, puerperal sepsis and gonorrhœa may be found. Microscopic and cultural examination of urine will help. On P.V. there will be definite tenderness on the bladder when other organs in the pelvis may be found normal.

My thanks are due to my Assistant, Dr. Miss. Randive, L.M.P., who helped me to write this article.

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Abdominal Migraine*

PARMA NAND AHUJA, M.B., B.S.,
Karachi.

THIS short note is intended to draw attention to a clinical condition which I have ventured to name 'abdominal migraine.' It is a summary of observations made on few cases two of which are detailed. I have been unable to find much on the subject in the literature available to me and will be interested to know if such observations have been made by others.

General.—Migraine has been defined as paroxysmal attacks of pain frequently recurring at varying intervals of few weeks or months. The term was originally applied to pain in the cranium, hence the name 'hemispheres,' but its scope should be considerably widened to be applied to attacks of pain generally but essentially paroxysmal in character. The periodical and paroxysmal attacks of pain occurring at times in the abdominal region resembling attacks of migraine may simulate many pathological phenomena generally comprising an 'acute abdomen' misleading the unwary, who may open the abdomen to find us offending viscera responsible for the painful condition to the great distress of the patient and discomfiture of the surgeon.

Illustrative Cases.—As illustrative cases the writer cites the following:

CASE NO. 1. Mrs. H, a middle aged Hindu woman, complained of regularly recurring attacks of excruciating pain in the right iliac fossa radiating towards the groin. Previous to and during the attacks she was constipated. The pain usually started in the early morning hours lasted till the mid-day and then gradually lessened in intensity. During the attack physical examination showed rather tense condition of the abdominal muscles in the iliac region. Deep palpation did not reveal any swelling or tumour in the region. She would lie on her right side with her legs drawn up. There was no rise in temperature. Urine was freely passed and analysis did not reveal any abnormality except phosphates on few occasions. Menstruation was rather defective in quantity and painful in the beginning but otherwise quite regular. No history of V. D. She

* Specially contributed to 'The Antiseptic.'

was a mother of four grown-up children, eldest being 18 years old. She was advised operation at times for appendicitis, other times for inflammation of the right tube and ovary by different surgeons at different times. But she stubbornly refused surgical interference.

A thorough examination by the present writer and observation of the patient for months revealed her to be a highly nervous woman, anæmic and debilitated. The attack was ushered in by pricking sensations in hands and fingers etc. At the beginning of the attack her face was rather pale and haggard but later on it became flushed and 'rosy.' Blood picture was one of microcytic anæmia. The attack continued for a week or ten days, then subsided recurring again after a month. The tentative view that the condition was due to vasomotor disturbance in the abdominal vessels caused by some toxic state of the blood was confirmed by the subsequent course of events. An eliminatory line of treatment was adopted. Paraffin emulsion with Phenolphthelene was given for a fortnight and later paraffin alone. A mixture of Cannabis Indica, Hyoscyamus and Valerian was administered and actual attacks were controlled by Pyramidon. Diet regime was regulated. Later her anæmic condition was improved with iron, arsenic etc., and course of vitamin B₁ prescribed. She has been free from the malady for the last 3 years or so now.

CASE No. 2. Mrs. F., a European lady aged 42 years, complained of paroxysmal attacks of pain in the epigastric region and the right hypochondriac region for the last year or so. The pain lasted for few hours and then disappeared. There is nausea or at times vomiting. It bears no relation to food, generally comes late hours in the evening. There is no inclination to take anything during the attack. Physical examination did not reveal any abnormality in the abdominal viscera except slight enlargement of the liver, two fingers breadth below the costal margin. Screening did not reveal any departure from the normal. There is history of dyspepsia extending over years. She had been advised exploratory incision which was refused.

Careful analysis of her case history showed that she was neurotic in temperament and easily excitable. The attack is heralded by sensation of tingling in the feet or legs. It regularly occurs every day lasting for a week or so and then disappears under some laxatives or partial fasting. The pain is described as agonising in nature. Palpation did not reveal any swelling or tumour but the abdominal parietes in the epigastric and hypochondriac regions felt tense and tender to touch. Expression of the face is flushed and anxious, the arteries on the temples beating visibly during the attack. Sphygmomanometer records no variation from the normal. She feels more comfortable to lie on the back. Blood condition is normal. Urine is loaded with phosphates. There is recurrence of the attack almost every month preceding the menstrual flow. Menstruation itself was quite free and painless and regular.

The present writer tentatively diagnosed her to be a case of abdominal neuralgia, the toxins in the blood acting on a hypersensitive nervous system near the menstrual period leading to spastic condition of the vessels in the abdominal region. The attacks were controlled with analgesic drugs and during the intervals vitamin B₁ and Calcium parenterally were pushed. Bromides were required to give her sleep at night. Emunctories were kept fully active and free. Rest was enjoined

Small doses of Bromides and Arsenic were continued later on, when the lessened frequency and intensity of pain warranted such a procedure.

Ætiology.—The above two cases are a pointer in the direction of this rather obscure malady which should be borne in mind when investigating a case of acute abdomen. The literature on the subject is rather meagre. The writer is however inclined to the view of a neuro-vascular spasm in the particular region caused by some toxic condition of the blood. The toxins may be due to dietary faults, gastro-intestinal intoxication, menstrual disturbances (endocrine), metabolic, etc. It may be reflex stimulation of the sympathetic nerves through mental weariness or psychological causes. If this short note succeeds in evoking interest among my worthy colleagues to throw more light on the subject, the attempt will not be in vain.

Differential Diagnosis.—The condition may masquerade any of the grave abdominal conditions comprising acute abdomen and lead to undesirable consequences.

Pneumonia and other chest conditions may be referred to abdomen, but high temperature, disturbed pulse-respiration ratio etc., may put one on the guard.

Perforation of a typhoid ulcer leading to acute pain, distension of the abdomen, vomiting etc. But the history of the case will be distinct and the temperature generally shoots high.

Acute appendicitis may be masked in the right iliac region. It is characterised by "the sudden onset of a severe colicky general abdominal pain, sometimes doubling up its victims and causing them to roll about." But sub-acute and chronic appendicular trouble may be difficult to differentiate. In the acute condition, rise of temperature and in case of actual abscess formation the blood picture will put one on the right track.

Acute pancreatitis—The condition is much more severe with prostration and shock and rapid pulse.

Colics—Renal and biliary colics may present some difficulty in diagnosis, but careful attention to the site of origin and distribution of pain will point in the right direction. In the case of renal colic there may be some disturbance in the passage of urine. Intestinal colic may tax the skill and ingenuity of the surgeon to arrive at correct diagnosis.

Obstruction—Rigidity is absent and tenderness is diffused all over the abdomen. Vomiting is persistent and particular signs of volvulus and intussusception will have to be kept in view.

Intra-abdominal hæmorrhage due to injury or ectopic gestation will cause sudden and severe shock and collapse of the patient. There is air hunger, restlessness and rapidly rising pulse.

The above are some of the important emergencies which 'abdominal migraine' may simulate in any of the areas affected leading to wrong conclusions and graver interference.

Conclusion and Summary.—The line of treatment adopted by the writer in case of genuine neuralgic conditions of the abdominal region grouped under the term of "abdominal migraine" has been suggested above. The writer would welcome any suggestions or views to elucidate and illuminate many of the dark lacuna in this obscure condition. The chief points brought out being the disturbance of sensation, pricks and

needles' in hands, feet etc., at the start; periodical and paroxysmal nature of the pain lasting for few hours every day for a week, with complete intermission for a month or so; the neurotic constitution of the patient and favourable response to sedative and antineuralgic remedies and toning up of the nervous system and removal of the underlying cause, dietetic, allergic, endocrinic etc.

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Naga Sore*

SUSHIL BOSE, L.M.F.,

M.O., Hulmari T.E., P.O., Khawang, Upper Assam.

THIS disease is most prevalent among the coolies of the tea-gardens, especially in Upper Assam. During the rainy season, too many cases attend the hospital. The treatment is very irksome and the choice of medicine is the main difficulty. The disease may run years together, if neglected.

Site.—At first a small nodule just like a pea appears over the lower part of the tibia, on the outer surface of the ankle joint, over the foot, on the knee joint, or rarely on the fingers of the hand.

Cause.—The patient, when he attends the hospital, narrates that a few days back, either there was an injury or abrasion or a leechbite on the part. Some authors describe it to be due to fusiform bacilli and others are of opinion that it is due to spirochaetes.

Symptoms.—(1) At first a small nodule—gradually developing into a small ulcer like a copper coin or a rupee, but in neglected cases, it becomes much more bigger than that. (2) Then it forms into deep ulcer with granules and indurated margins. (3) Slough constant. (4) Muscular tissues are destroyed near the area. (5) There is burning sensation and pain. (6) Sometimes there is watery discharge from the ulcer.

Differential diagnosis—The disease will be differentiated from: *Simple*:—(1) Acute ulcer; (2) Healing ulcer; *Specific*:—(3) Syphilitic (primary sore); (4) Gummatous ulcer; (5) Tubercular ulcer; *Malignant*:—(6) Carcinomatous ulcer; (7) Rodent ulcer.

Treatment.—I tried host of drugs externally *e.g.*, Calamine Preparata, Zinc Oxide, Boric Acid, E.C. in warm (luke) water, M & B '693' powder, Copper Sulph. solution, normal Saline wash, Urea Stibamine powder in unguentum and internally *e.g.*, M & B '693,' Calcium, Mist Iron Tonic, but I found Magsulph externally and Calcium Gluconate internally very useful. I treated 92 cases of Naga Sore during the last two years and all were cured.

I gave the following treatment:—

External:—Excess of pus and crusts is cleared gently from the ulcer with a piece of gauze and forceps and thick crusts are gently lifted with the forceps. Firstly, absolutely dry Mag Sulph powder is poured liberally directly on the ulcer so as to cover it completely. Secondly, over this a light layer of gauze and wool is placed and bandaged firmly with

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a small many tailed bandage. The second bandage will be done after 12 hours, when excess of wet salt is lifted off gently with a spatula and fresh dry Mag Sulph applied. Fresh gauze and wool are bandaged as before. After 4 dressings there is usually one change in 24 hours. If there is excessive pain or burning sensation add to it Boric Acid powder.

Internally.—Calcium Gluconate—gr. xv B. D. till recovery. This treatment takes 10 days to 3 weeks for a complete cure. After recovery there is always scarring on the affected part.

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Ear in its Relation to General Practice*

C. V. C. RAO, Z.L.O. (VIENNA),

Eye, Ear, Nose and Throat Surgeon, Hyderabad (Deccan).

EVERY general practitioner will have occasions to treat cases of ear trouble, at one stage or other. As a matter of fact, even the cases that come to specialists, will have consulted a general practitioner in their early stage. Hence it is imperative that a general practitioner should have some knowledge of the ear and its diseases, so that he could successfully treat the minor troubles himself and be able to recognise major troubles, early enough. This is the object of this paper and hence the readers will pardon me if I don't enter into greater details, which are of no interest to the general practitioner.

The most important symptom that the patient seeks relief and which every practitioner is called upon to treat sooner or later, is pain in the ear (otalgia). Although it is a symptom, as it is so important, I have taken it first and propose to deal with it a little in detail. Pain in the ear may be an occasional twinge coming on at intervals or may be severe paroxysm of sudden onset preventing sleep and demanding prompt relief. Unless the cause be discovered, it is evident it can't be adequately treated.

Pain in the Ear (Otalgia).—May be due to local causes or it may be only reflex, referred to ear from affections of neighbouring or distant organs.

Local causes should be looked for in the auricle external meatus, tympanic membrane and the surrounding parts of the ear. It is necessary that one should be provided at least with a good focussing light and an aural speculum, if a head mirror is not available. The majority of cases are inflammatory, streptococcal infections, ulcerations and growths are also possible. Perichondritis of auricle is a fruitful cause. A boil in the ear is not always easy to discover. Pain on pulling the auricle in different directions will help or a probe may discover a tender spot in the ear. Sepsis of scalp can spread to the ear. A tender gland in front of tragus points to a furuncle. The tympanic membrane must be examined for its normal pearly colour. Even slight redness along the handle of malleus may indicate starting otitis media. Look for hæmorrhagic blebs on meatal wall or membrane. In case of acute or chronic otitis media, tenderness over mastoid region suggests that pain is due to mastoiditis.

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Briefly the local causes are:—*Affections of auricle* as trauma, erysipelas, perichondritis, new growths. *In the meatus*: wax, foreign body, furunculosis, hæmorrhagic blebs. *Middle ear*: myringitis, acute and chronic inflammations, new growths (though rare), gland enlargement due to scalp infections, lymphangitis and mastoiditis.

Reflex causes:—When one excludes these causes, one has to look in other organs for possible reflex causes. A deep study of the nerve supply and anatomy of ear shows that it is connected with a number of nerves motor or sensory, in some way or other. Briefly V, VII, IX, and X cranial nerves supply sensations to a wide area and to the ear. Hence the whole upper respiratory tract must be examined for the cause of reflex pain. The examination must include teeth, tongue, tonsils, palate, nasopharynx, pharynx, epiglottis and larynx. Carious tooth and maleruption of teeth is a common cause of pain in ear. Ulcerations and new growths of tongue (trigeminal or glossopharyngeal), tonsillar affections and growths on tonsils and palate (V nerve) can cause otalgia—(I had a case of intermittent pain in the ear of nine months' duration. When examined during acute attack, a cystic growth is noticed in one tonsil. It was incised and considerable plug of debris and cheesy material was expressed from the deeper parts of a crypt and the pain is relieved). Pressure on tonsil by a long styloid process can produce pain in ear. Inflammations and œdema of soft palate are another source. (Glossopharyngeal) growths in the nasopharynx, affections of pharynx, epiglottis and larynx can produce only ear-ache.

Symptoms.—Sinusitis or congested nose can cause also ear-ache. (glossopharyngeal).

After examining internal parts, a search is to be made in the external structures. Enlarged glands in the neck, parotid gland and temporomandibular joint, may reveal the cause of pain. Finally examination for high blood pressure, some general nervous disorders as tabes and gout is to be made. Neuralgias from posterior root inflammations of the nerves which send branches to ear, cause severe ear-ache. These are chiefly trigeminal, glossopharyngeal or 2nd or 3rd cervical. These may be accompanied by herpetic eruptions of the skin, though it takes three or more days after appearance of the pain. Severe afebrile pain in and around ear with normal hearing and normal meatus must arouse the suspicion of neuralgia.

Treatment of otalgia.—It is needless to say that the treatment depends on removal of cause as far as possible. In cases of referred pain treatment of exciting causes is indicated. In neuralgias with or without herpetic eruptions, treatment is symptomatic. Aspirin hypnotics and even Morphia in severe cases are required. For post-herpetic neuralgia, a mixture containing Quinine Hydrochlor gr. 1; Phenacetin grs. 5, t. d. s. is often successful. For ear-ache after tonsillectomy, insufflation of finely powdered aspirin or orthoform on to the tonsil beds, gives relief. As to the treatment of local causes, I shall deal with them in due course.

In dealing with the causes of pain, I have practically exhausted the names of the diseases of ear. It now remains for me to deal briefly with the important diseases that are likely to be of interest to the general practitioner.

External auditory meatus.—*Impacted cerumen*:—This is very common in general practice. This can give rise to variable degrees of deafness

and pain due to pressure. Although it is easy of diagnosis, some cases tax the ingenuity of the doctor, to completely remove it. When it is very hard, better to use Soda Bicarb solution for a couple of days, to soften the matter before syringing. In this connection, a few words on syringing, I hope, will not be out of place. For a successful syringing a little space between the wax and the walls of the canal is necessary, because the object of syringing is to manage to get the water behind the cerumen, so that it is pushed out by it. Hence it is advisable always to direct the flow of syringe either along the floor or roof of the canal and not straight on the cerumen. Much force should not be used as it may cause damage to the drum. If the canal is tightly plugged with wax, it is necessary to disturb the thing with the probe, inserting it between the canal wall and wax and thus create some space for water to get behind (provided the wax is soft). Blind and forcible syringing against the wax might drive it further back against the drum and cause more trouble. Although unskilled instrumentation is better avoided, as abrasions may be caused to canal walls, still, in some cases, judicious use of probe or scoop will help a great deal. When complete view is not possible, better to rely on softening with Sodium Bicarb cum Glycerine solution and gentle syringing. Syringing by itself should not cause much pain and, when it does, it means that something is wrong with your method and is a signal to stop it and further explore with light.

Foreign bodies.—Here again judicious use of syringing is the safest and best method. Forceps should not be used, for fear of driving the body further down. Cases are very frequent, where by such manipulations by inexperienced doctors, patients are put to unnecessary sufferings and complications, necessitating drastic steps for its removal. An apparently harmless and easy looking thing can be sometimes converted into a difficult and dangerous problem by our injudicious acts. If the foreign body is a vegetable substance, a few drops of rect. spirit might help to shrink it. If canal is swollen Cocaine-Adrenaline drops will reduce it and give more space. If the F.B. is a hard one and if there is space between it and canal walls, a hook can be tried. If manipulations are painful and not easy, especially in children, light general anesthesia is necessary.

Boils and furuncles of external ear are very common. There is pain in the ear, which gets worse when swallowing or masticating. It has to be distinguished from middle ear trouble. If the trouble develops after a cold, it points to middle ear. Pain on moving tragus, presence of tender spots in canal, clear and transparent drum are some of the points for diagnosis. In external ear trouble, pre-auricular sulcus is obliterated, while in middle ear trouble, it is not affected. Pressure on mastoid causes pain in middle ear trouble. In most cases, diagnosis is easy, especially when furuncle or inflammation is present. But in some severe cases, external meatus is so much swollen, that the drum is not visible. In these cases it is not easy to exclude middle ear inflammation and it is also possible that both may exist together. In doubtful cases better to treat it as for the more serious trouble of middle ear inflammations. As for treatment of external furunculosis tampons dipped in saturated solution of Mag sulph, Argyrol 10%, Lead Acetate solution or Itchylol Glycerine 10%. It is necessary to keep the parts in contact with the medicine. Hence the ear is lightly plugged with thin gauze soaked in any of these solutions and later the gauze is kept moist by dropping the medicine two hourly over it. Once a day it may be removed

and fresh one replaced. If pus is clearly visible, boils may be incised, if pain is too much. Dry fomentations to the ear and analgesics are also indicated and are very helpful. After the acute stage is over some antiseptic drops are needed for a couple of months. Along with this, general treatment also may be carried. Vaccines, manganese, and yeast are advocated by some. In recurrent cases, search for underlying infective focus should be made especially in teeth, tonsils and sinuses. A most useful prescription in troubles of external ear is, Ung Hydrar Oxy Rubra 80 grains, Ol. Cinnamon 1 minim and Ol. Amygdale one ounce. These drops to be instilled once a day. Ionisation with Stannoxyd in recurrent cases helps to sterilize the canal.

Other common troubles of external ear are chronic dermatitis and oto-mycosis. These are very persistent. Syringing should be avoided in these cases, except when too much debris gathers in the canal. Dry treatment with insufflations of Calamine or Zinc Oxide or moist treatment like Mercury ointment or above mentioned Mercury drops can be tried. The canal should be kept clean. In the healing stages itching is the most trying symptom. Calamine or Camphor ointment (190) is good for this. Ionisation is also worth trying.

Tympanic membrane.—Inflammation of drum otherwise known as myringitis—the drum takes part in inflammations of external and middle ear, or it may alone be inflamed. Very severe pain in ear, which the patient points as deep inside, is its chief symptom. It is usually the result of exposure to extreme cold or nasal catarrh. Syringing the ear can produce myringitis. There is no fever. Hearing is very little affected. In fact, this is a very helpful sign to distinguish it from inflammation of middle ear. The drum is red and congested and the outlines are not visible. If there is considerable exudation in the layers it is bulging, otherwise bulging is not much noticeable. The condition may generally last two or three days. Sedative drops as Acid Carbolic grs. xv, liquor opii m. 20, Menthol gr. x, Glycerine 3 i, may be dropped in ear every 2 hours. Dry fomentations and analgesic tablets will generally relieve the condition. If it persists in intensity after 24 hours, in spite of treatment, an incision of the drum, whether bulging or not, will give quick relief. This early incision of the drum, especially if drum is bulging, is very important and helpful in preventing middle ear complications. Of course many a case may be cured without, in spite of indications, but what happens in these cases is the patient's agony prolonged, with increasing risks of middle ear complications. If pain is not relieved but increases after 24 to 48 hours the best thing is incision (paracentesis).

Middle ear troubles.—The most common disease that a G. P. is called upon to treat is discharge from middle ear, commonly known as "running ear." It can be chronic or acute. A chronic discharge is always the result of an acute attack and an acute attack has its origin in infections of upper respiratory tracts. Hence while the discharge is being treated, it is also necessary to pay attention to the causes like nasal catarrh, septic tonsils and adenoids. These troubles are very common in children and if not promptly and adequately treated, are liable to leave behind their effects like impaired hearing, apart from the possibility of intra-cranial complications. An acute middle ear suppuration starts with severe pain and fever. Drum is inflamed and bulging with some impairment of hearing. If the drum is burst and discharge starts, pain may be relieved and hearing restored with cessation of other symptoms. In se

cases, the discharge does not come out although all inflammatory signs are subsided. In these cases hearing is affected and requires catheterisation. The chief causes of otorrhoea are as already mentioned nasal colds, acute infections, septic tonsils and adenoids, sinus infections and dental caries. It is important that attention should be paid to these factors also, if a recurrence or chronicity is to be avoided. Above all the chief factor that contributes to the success of the treatment is the resistance of the child, depending upon general health and environments. If attention is paid to all the above factors, there is no reason why an acute attack should become chronic. Chronic otorrhoea is more a case for aural specialist. Hence I will deal here chiefly with acute attacks, as to how they can be prevented from becoming chronic.

I have already given a resume of the signs and symptoms of acute otitis media. I shall mention just one more point before I go to the treatment. In all cases of acute attack, a certain amount of mastoid inflammation is bound to take place, which is revealed as haziness of mastoid cells by X-ray and by tenderness to manual pressure. This need not give rise to any alarm of mastoid infection. In most cases this subsides with the proper treatment of middle ear, without necessitating mastoid opening.

Treatment of acute middle ear.—In the early stages, before discharge starts, the drum can be seen inflamed and bulging. In every case of bulging drum, with signs of acute inflammation, an early incision of the drum goes a long way in the subsequent course of the case, than waiting for normal bursting of the drum, to discharge the pus. In the waiting period not only patient has to suffer agonising pain, but the pus under pressure is likely to run to antrum and thence to other mastoid cells and thus spread the infection. On the other hand, an early paracentesis relieves the pain immediately, provides an outlet to pus and then reduces the dangers of mastoid infection. In any case an early paracentesis does no harm even in cases that started discharging. This may have to be done if the perforation is not big enough to allow free flow of pus (a potent cause for chronicity).

The most important thing in the treatment of acute suppuration, is cleaning the ear as often as necessary and thus facilitate the flow of pus. Some advocate slow syringing but it is widely condemned. The best method is cleaning with dry cotton swabs. Any medicine into the ear is of doubtful benefit. Routine use of H_2O_2 does more harm than good and therefore to be discouraged. Weak solution of Acriflavine 1 in 500 may be tried. Any throat or nasal sepsis should be attended to—gargles if necessary. Menthol nasal drops in the nose to keep down congestion in the nose. An ordinary case, free of mastoid trouble, should be alright in 4 weeks. If discharge persists after that, without other complications, an infection of the upper respiratory tract should be suspected and more vigorous treatment of causes is indicated. Finally, as already pointed out, the general health of the patient must receive attention with a view to increase the resisting power. Fresh air, avoidance of overcrowding, general hygiene, good food and vitamin A are the pointers.

This, in brief, is the treatment of A.S.O.M. The majority of cases get well after rupture of membrane and discharge of exudation. Pain and hyperpyrexia disappear within a few days. The discharge, at first blood-stained, becomes thicker and purulent, then in a few days thinner and finally dries up and the hearing is restored. This fortunate termination

of A.S.O.M. attacking a previously healthy ear, especially if properly handled, is the commonest event. But one cannot always expect a complete recovery, even when it is carefully treated. It may drift into chronic suppuration, or one of the graver complications may set in. It is not necessary to go into the details here. I will stop with giving some of the danger signals, which must put the practitioner on his guard: (1) If the discharge continues for more than 4 weeks, even if pyrexia and pain subside; (2) if pain persists in spite of free flow of pus; (3) if that pain is accompanied by pyrexia; (4) persistent headaches frontal or occipital; (5) tenderness and swelling of mastoid region; (6) persistent vertigo; (7) sudden cessation of discharge, with pain and headache and possibly temperature; and (8) swelling and bulging of roof of external canal.

The treatment of complications and chronic middle ear suppuration belong to the sphere of specialists—also hearing defects.

Appendicitis*

SUDHIR CHANDRA ROY, B.Sc., M.B., D.T.M.,
Hong. Physician, Sadar Hospital, Nawabganj, Rangpur, Bengal.

BEFORE I actually proceed to write something about the line of treatment to be adopted in acute appendicitis. I think it will not be out of place to mention a few salient points about the diagnosis of this disease as laparotomy has often been performed on a mistaken diagnosis.

The first symptom is malaise, some nausea, loss of appetite and a vague pain over the abdomen. At times there is slight colicky pain accompanied by nausea and often vomiting and slight rise of temperature. On examination the patient looks rather flushed but not very ill, tongue furred and the pulse and temperature above normal. There is some restriction of movement of the abdomen, particularly in the right lower quadrant. The right rectus muscle is rigid and there is definite tenderness on pressure. The location of maximum tenderness varies according to the position of the appendix. If it is lying just below cæcum in the right iliac fossa the tenderness will be at McBurney's point, but when it is retrocaecal the tenderness is higher up and more medial. Rigidity of psoas from spasm is common and this causes flexion of the thigh on the affected side. When the appendix is lying over pelvic brim the abdominal tenderness is very slight. A rectal examination will elicit definite tenderness on the right side. In some cases the local signs are vague and there is no muscle guard, and rectal examination yields a negative result. In such cases a leucocyte count will help to make a diagnosis. A count over 10,000 will point towards appendix. A low leucocyte count, however, does not exclude appendicitis. C.H. WATSON and T. R. SARJEANT (observation on 160 cases) concluded from the total leucocyte count and the polymorphonuclear count in acute appendicitis that a low leucocyte count indicated not a failure on the part of the bone-marrow in leucopoiesis, but the withdrawal of a large number of leucocytes from the blood stream to a suppurating focus. In ordinary acute appendicitis one of the earliest signs of infection was a rise of leucocyte count and the curve of the total leucocyte count and the polymorphonuclear count running parallel, when the peritonium was involved, there was a further rise of polymorphonuclear count, with an increasing shift to the left, due to the entry of young

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leucocytes into the blood, but there was an abrupt fall in the total leucocyte count, the extent of the fall being determined by the size of the area involved in the suppurative process. In fatal cases the curves representing the total leucocyte and the polymorphonuclear counts diverged continuously, whereas when the infection was successfully resisted, the curves converged and fell together to the normal.

In obstructive variety the temperature may be normal and the pulse slow and regular during the first few hours, the tongue is not also dirty but there is generally anxious look, intermittent colicky pain, vomiting and some tenderness in the right side of lower abdomen. Leucocyte count also does not help in these cases in the early stage. The symptoms are also very deceptive in old age as there is little reaction, the natural power of local defence being poor. Intra-peritoneal infection is less likely to be circumscribed in the child, the reason being that the omentum is relatively short and the leucocyte reaction relatively slight.

Treatment.—With these preliminaries I proceed on to the method of treatment and myself being a physician I shall not deal with the operative technique, the best method of performing it etc., but shall deal only with the expectant method of treatment.

There exists still now controversy as to the right method of treatment of acute appendicitis and it is now generally accepted that wise and safe method of treatment for acute appendicitis, if diagnosed early i.e., within 24 to 48 hours, is immediate operation. The expectant treatment is better not to be adopted in cases of children or the aged. In old age operation is perhaps the best but the death rate may be as high as 30 to 35 per cent. The expectant treatment is more suitable for cases seen four or five days after onset of attack and who is not seriously ill and presents a firm mass in the right iliac fossa. In this method three events might follow: (a) subsidence of local peritonitis and recovery; (b) the formation of an abscess; and (c) increasing local rigidity with rise of temperature and pulse rate. Only in (a) group of cases the treatment may be continued with safety and surgeon's help should be taken for (b) and (c) groups when ever occasion arises. Under expectant treatment many cases of most serious type will slowly recover with or without the necessity for the evacuation of an abscess.

In the expectant treatment the patient must be under constant supervision and pulse rate must be recorded every hour and the temperature four hourly, and everything must be kept ready for operation should the necessity arise. The patient is put in Fowler's position and no fomentations are applied to the abdomen. Nothing except water or Glucose is allowed per mouth until the temperature and pulse are satisfactory. Intravenous glucose and saline preferably by the continuous drip method is given if the starvation is prolonged. Purgatives must not be given on any account and glycerine enema may be tried for flatulence and abdominal distension. Sulphanilamides may be given and it is a good drug for peritonitis. Along this treatment I am particularly fond of giving Emetine Hydrochlor gr. i. in 1 c.c. injections every day or every other day up to a total of six or nine in less severe cases because I am at present rather convinced that many of the cases of appendicitis are due to amoebic origin particularly in this area. Immediate operation will be necessary if—(1) there is an increase of pulse rate; (2) the patient vomits repeatedly; (3) any exacerbations of abdominal pain; and (4) occurrence of rigor, as all these show that inflammation is spreading.

* Specially contributed to 'The Antiseptic.'

Doubts have been expressed about chronic appendicitis as a clinical entity but it is agreed by most clinicians of experience that a chronic inflammation of the appendix does occur. But ill-defined pains and tenderness in right iliac fossa, for a long period associated with some ill-health are more likely due to constipation, neurosis, chronic salphingitis etc., than to appendicitis and so careful investigation should be undertaken before operative procedure is adopted.

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The Diagnosis of Renal Pain*

BON BEHARI DAS, L.M.F.,
Chief Medical and Health Officer and Superintendent of Jail,
Chingbhakar State, E.S.A., C.P., via Shadol, B.N. Ry.

THE pain in the loin may be due to two fundamental causes: (1) Renal, or (2) arising in organs other than those of the urinary tract and simulating more or less closely true renal pain.

The true renal pain is of two varieties: (1) A dull aching or dragging pain or renal aching. (2) Renal (and ureteric) colic.

Renal aching.—This is generally associated with swelling of the kidneys e.g. inflammation, hydronephrosis, growth or large calculus. Unlike the renal pelvis, the parenchyma is totally insensitive to pain, and the cause of the aching is probably due to the stretching of the renal capsule, or the drag upon its pedicle from an over-weighted organ. Its side is comparatively circumscribed and may be either in front or behind. In front, the tip of the 9th rib corresponds fairly accurately with its distribution, behind, the angle between the last rib and the area of choice.

Renal colic.—The pain of renal and ureteric colic is of an intensely agonising character and in its severest form is associated with sweating, vomiting and a thready pulse, and on occasions hæmaturia. Repeated attacks may originate at a progressively lower level, and the pain radiates downwards along the ureter to the iliac fossæ, testicles, labium and upper thigh. Severe ureteric colic is associated with the passage of relatively small calculi down the ureter.

The emptying of a pyo or hydronephrosis is associated with pain far more chronic and far less agonising than that of calculus. It resembles the pain due to the inability to pass winds.

In Dietl's crisis, where a kinked ureter is associated with torsion at the kidney pedicle, pain, sweating and vomiting occur with occasional passage of blood in the urine. Rigidity is present and the kidney, if palpable, is tender.

Diagnosis.—It is better to consider it under four heads:

- I. Back-ache.
- II. True renal conditions.
- III. Colic.
- IV. Intraperitoneal conditions.

I. Back-ache.—The main causes of back-ache are spondylitis, fibrositis, Pott's disease, perinephric abscess, cervicitis and more rarely prostatitis. The commonest cause of "back-ache" confused with chronic renal pain is that of *spondylitis*. It occurs usually in men above the age of 55, gives rise to bilateral back-ache and radiates round the flanks to the iliac fossæ. It is associated with rather rigid poker back, and tender

* Specially contributed to The 'Antiseptic.'

spots are not present. Spinal movements are limited and may provoke pain. An X-ray shows lipping of the vertebræ and other changes in the spine.

Fibrositis can hardly be confused with true renal pain, but the therapeutic test of injection of 1% Novocain 10-20 c.c. will clear up temporarily at any rate lumbar pain of this nature.

Cervicitis:—It is a prolific cause of back-ache. The appropriate removal of a septic mucous membrane by the electric cutting loop is followed by a spectacular cure in most cases.

Perinephric abscess:—It is a condition sometimes overlooked in its early stage. The high swinging temperature, pain and rigidity, the bulging flank and a fixed tumour that does not move on respiration are its main characteristics.

II. True renal pain.—This is caused by pyelitis, pyelonephritis, pyonephrosis, hydronephrosis, tubercle, renal calculus, growth and Dietl's crisis.

Pyelitis and pyelonephritis are evidenced by a tender, painful and palpable kidney, a high swinging temperature and pus in the urine, to which may be added blood and casts as the renal parenchyma become more involved. Rigor is common and the condition is acute. There may be some difficulty in distinguishing pyelitis from appendicitis and cholecystitis, but the tender kidney of pyelitis, the higher temperature, the rigor and the absence of pain in the iliac fossa or hypochondrium are generally sufficient to differentiate the renal from intraperitoneal causes. Pus in the urine, when found, is of course, a most valuable aid. Pyelitis is common in pregnancy and in children.

Tuberculous kidney:—There is chronic ache, and is as likely to be present on the sound side as on the diseased side owing to the compensatory swelling of the normal kidney. It is rarely an early sign, and generally frequency and dysuria will have drawn the attention of the doctor to the possibility of urinary tubercle before renal has become marked.

Renal calculus:—The pain is due to the associated infections or to renal enlargement. The more chronic conditions of pyonephrosis, hydronephrosis, growth, large renal calculus and mobile kidney require investigation by X-ray pyelogram and cystoscope.

Some cases where pain is localised to the kidney region have no organic basis at all and their case is obscure.

III. Colics.—Urinary, biliary and intestinal have as their common cause the violent contraction of plain muscle.

COLICS

INTESTINAL	BILIARY	URINARY
Onset of pain is sudden or gradual but varies in severity. It is central and subumbilical. Nature paroxysmal and twisting, relieved by pressure, constipation or diarrhoea present.	Sudden constant or paroxysmal aching and agonising pain in the right hypogastrium, radiates backwards and upwards to the region of scapula specially right, or in all directions over the abdomen and lower part of thorax first relieved by pressure, then unbearable over the gall-bladder. Jaundice soon supervenes, other hepatic symptoms may be present. Generally attacks the female at or after middle life 45.	Pain begins suddenly in loin, radiates downwards over the anterior aspect of abdomen on affected side, shoots down to testicle, ovary to the inner side of the thigh. Crystal or other urinary changes, hæmaturia, frequent micturition or stranguary. Subsequent passage of calculi in urine. At any age usually male.

It is sometimes difficult to distinguish between a ureteric colic and appendicular colic. As a rule ureteric colic is more violent, more intermittent, associated with sweating and a tender loin, whilst rigidity if present during spasms is absent between. The right iliac fossa is free from tenderness. Blood may be present in the urine, but rarely appears at the beginning of the attack.

IV. Intraperitoneal conditions.—Sometimes intraperitoneal catatrophe as acute appendicitis, cholecystitis and perforation of the gastrointestinal canal are confused with renal pain. The most likely confusions are between a low ureteric and an appendicular colic, between a cholecystitis, appendicitis and a pyelitis and in an accident between a ruptured kidney and a rupture of spleen, liver or even intestine.

Reference: The Medical Press and Circular, 8-1-41, pages, 34 to 36.

Psychological Importance of Skin*

K. D. LAHIRI, M.B., B.S. (Pat.),

From Skin and Venereal Diseases Dept., Patna Medical College Hospital.

THERE is no water-tight compartment between the science of medicine and the science of psychology, rather one merges into the other, and all the elucidation demands a psychological view-point is now attracting attention of the specialists in skin diseases.

The skin presents a very complicated and manifold relationship to the individual and there is no other system of the body which is psychologically and physiologically so intimately related to the body. The developments in the psychological importance of the skin promise to produce surprises. DR. O'DONOVAN of the London Hospital thinks it to be an outstanding feature in the practice of dermatology that additions to knowledge and experience are reflected with great rapidity in the field that would discuss the effect of psychological injuries and emotional strain in producing cutaneous diseases. DR. DUFKE in Derm. Wochen, May, 1926—reported a case of psychical urticaria in a female patient, aged 28, who complained that for the last two years contact with silk material caused a rash. Six minutes after placing a silk on the patient's neck typical urticarial wheals appeared, not only on the part of the skin covered with silk but also on the face. On another occasion a piece of linen was used; no skin change followed, the patient probably detecting the change of material. When, however, the patient was told that it was silk, urticarial wheals appeared in the usual time. I had a blind patient who had eczema of the feet. This would develop soon after putting on new shoes if it was of goat's skin. He tried all sorts of possible prescriptions for his skin condition. At the time I saw him he had a new pair of shoes on, which he bought only a week back and hardly put on a day or two. I was tempted to try psychotherapy and took him in my car and both of us drove to the "Boots Store" and I with the co-operation of the dealer managed to give him the same pair of shoes after trying two, three pairs and putting in a separate box and with lace of different type saying at the same time that it was of calf skin having the same price. My patient seemed to be satisfied. I gave him few injections of distilled water daily for four days and on the fourth day I could hardly see on the skin of his feet any of those eczema. The more psychological factors are looked for, the more frequent appears to be their incidence. It is common

* Specially contributed to 'The Antiseptic.'

for a skin specialist to examine a patient for signs and symptoms of functional disorder, in order to aid in interpretation of his physical signs.

One might think that I would be prejudiced in favour of a high incidence of psychological factors in diseases ordinarily regarded as mainly physical in origin but to support my conviction I would like to quote a few well-known doctors' observations. DR. W. J. O'DONOVAN, O.B.E., M.D., M.R.C.P., Physician of the Skin Department of the London Hospital, has observed that the very resistant type of seborrhœic dermatitis is due to mental shock, and is cured by appropriate measures. DR. ROGERSON, a skin specialist of the Guy's Hospital, London, has observed that the psychological factor in 21 cases of the asthma-prurigo complex in children at Guy's Hospital was cured by psychotherapy. DR. STRAUSS, another skin specialist, has found in over 50% cases the psychological factor in adult asthmatics. DR. THOMAS, an American skin specialist, at the New York Medical Centre, found the psychological factor present in 31 cases of rheumatoid arthritis.

Psychological conditions are among the most chronic and are also amongst the most curable diseases once their real nature is recognised. When a doctor starts investigation of a physical disease from psychological aspect one is sure to find some very important, hitherto unexpected and unsuspected psychological stresses. When any disease is treated without any regard to the psychological factor that disease is sure to become chronic and resistant to all treatment. As disability and insanity may disappear marvellously after court settlement, so, too, a doctor's advice can equally work wonders in some chronic types of skin diseases.

The psychological importance of the skin may be classified as:—(1) Skin is an important organ of emotional expression and reacts to the feelings of fear, anger and shame. Excessive sweating and excessive blushing give rise to disturbances of the skin function resulting from emotional causes.

The continued emotional stress or conflict as with psychoneurotic conditions of contemporary origin and getting rid of the symptoms may depend either on the possibility of acquiring a different attitude to the situation itself. These psychogenic symptoms continue simply on the basis of misunderstanding of their nature, explanation and reassurances will clear them up.

A lady was cured of excessive sweating when she started liking her husband whom she always hated and was told of the social inconvenience of excessive sweating and that it was not a constitutional weakness of any kind.

There is a fairly complicated variety of emotional factors behind excessive blushing and may have developed from œdipus complex or narcissism.

(2) Skin as a medium for exhibitionism—the pleasure one gets by displaying. DR. GILLESPIE has recorded two very interesting cases; a hysterical woman came under his observation who exhibited numerous bruises especially on the skin of her breasts which she never hesitated to exhibit, and another case of a young woman who had a small ulcer on her right buttock which she never allowed to heal and scratched to remove the scab to keep it a wound to exhibit.

(3) Skin as an important organ of excitation—according to FREUDIAN theory—the entire body surface is regarded as an erogenous arch in infancy with areas of special erotic sensibility round the mouth, the anus and the

genitals, each reaching its maximum relative importance in that order in time of development. Ultimately in the normal course of events the genital area predominates. SADGER added the hypothesis that in later life, regressive processes may lead to reanimation of interest in the anal region and accounts for the tendency to pruritus ani in later life.

(4) Skin is an organ of appreciation of pain. In some individuals skin becomes of special importance in relationship to deep-seated emotional trends such as feelings of guilt.

(5) Skin at a vehicle of hypochondriacal tendencies—hypochondriacal preoccupation with the skin. It is very commonly seen amongst women who never feel clean until they wash many times either their hands or feet or plates. DR. GILLESPIE saw once an interesting family group, which illustrates a variety of the ways in which the skin may become the focus of hypochondriacal trends in the personality. Father, mother and daughter all believed themselves to suffer from a parasitic affection of the skin, and continually washed and disinfected with lysol.

Cases and Comments

Lathyrism

BY

S. K. AHMAD,

Medical Officer, Safdar Memorial Hospital, P.O. Milaky (Gaya), Bihar

LATHYRISM is a deficiency disease but fortunately it is not so common as rickets or beri-beri. Its aetiology is not definitely known. Some believe that it is a chronic food poisoning caused by the ingestion of maize, and others claim that it is due to the too frequent use of khesari dal in diet. Maize and khesari may be the causative agents but the disease is also found in places where maize and khesari are not used by the people at all. Some authorities say that it is caused by the deficiency of vitamin B and it affects only those whose diet is deficient in vitamin B. Khesari dal is a very common food in this locality but so far I have come across very few cases of lathyrism.

Lately, I have come across six cases of this disease, three from one village and the rest from three different villages. All gave a history of having taken khesari dal (not maize) for months together. All cases were from the poorer classes and they were males below the age of 20 years. None gave any history of venereal diseases. The patients are very poor, their food lacking in most vitamins. They could not afford to take milk or fresh fruit.

The main signs and symptoms resembled those of lateral sclerosis of the spinal cord. The patients first feel weakness of the legs. There is pain in the girdle and difficulty in walking and getting up from bed. The foot feels heavy on walking. In a full developed case there is acute lumbar pain, the lower extremity is rigid, the gait spastic, the knee-jerk exaggerated and the patient cannot stand erect. Later he is unable to stand up or walk about without the help of others. The upper extremity was found normal in all cases. Sphincters were not involved in any case. There was general debility in all cases, and complaint of insomnia due to acute pain. No other abnormality was detected except irregularity of the bowels and loss of appetite.

As regards treatment the patients were advised complete rest. Khesari dal was stopped in the diet. Vitamin B was administered parenterally as well as orally. Fresh fruits and green vegetables were given. Calcium and Bromide were also given. After the administration of vitamin B, it was found that the lumbar pain was much decreased and the patients were able gradually to walk about freely without any help, but spastic gait was still present on close observation.

I am grateful to the Civil Surgeon, Gaya, for his kind permission to publish this article.

A Case of Leukæmia in a Baby of 6 Months

MANORANJAN CHANDA,

"Chanda Niketan" Munikganj (Dacca).

IN September last I was called in to see a baby (male) patient 3 months old for bronchitis from which he had been suffering then for the last 3 days. The baby was found to be fairly well developed, well nourished and of mild disposition (not irritable), but slightly anæmic. The throat was congested, one tonsil was slightly enlarged. The spleen was 3", liver $\frac{1}{2}$ " below the costal margin. No abnormality was noticed in the heart, lungs, digestive system or in urinary or central nervous system. The baby was somewhat constipated. Temperature was 98.2° F. Next day of my visit the patient vomited a considerable quantity of yellow liquid in the morning and temperature rose upto 100° F in the evening. I suspected it to be a case of malaria which was prevalent at the time in an epidemic form. I prescribed him Quinine Ethyl-carp, Seroline, Calcium, Milk of Magnesia and a mild throat paint but no improvement was noticed. On the third day of treatment it struck me that it could not be an attack of malaria or K.A. but some other ailment.

I advised the relatives of the patient to get the blood examined. No malarial parasite was found in the blood; in diff. count, there was indication of K.A. The patient was sent to Calcutta for further treatment and there the attendant physician decided to push Neostibosan. Before injection the blood was again examined and from the result of examination the disease was diagnosed to be a case of leukæmia. Many eminent physicians were consulted in Calcutta but with no satisfactory result. The patient died in the latter part of November, 1943. The fever ranged from 98° F to 101° F (irregular, intermittent). Spleen was enlarged upto navel and was hard and not tender. No anorexia was present. The patient was somewhat constipated from the beginning of the attack. Bronchitis was increased at the time of death and patient was emaciated and anæmic. Diet was breast milk and lactose.

The treatment was Ferr et ammon citras, Liq. Arsenicalis, Glycerine, Celin tab. with liver powder, etc. Deep X-ray exposures were given. The liver was diminished (3 fingers) after X-ray therapy. On the whole 8 exposures were given.

Blood report:—Before X-ray exposure.

7-10-43. Total leukocyte count 44,450 per cmm.; total erythrocyte count 2,030,000 per cmm.; hæmoglobin 40%; diff. leukocyte count—Polymorphonuclear neutrophils 13%; lymphocytes 85%; polymorphonuclear eosinophils 1%; blast cells 1%; plenty of normoblasts and evidence of anisocytosis and poikilocytosis.

Routine blood report:—After X-ray exposure.—

10-10-'43. Total leukocytes count 42,812 per cmm., diff. leuko count—polymorphonuclear neutrophils 21%; lymphocytes (small) 74% lymphocytes (large) 4.5%; myelocytes 5%.

17-10-'43. Total leukocyte count 14,062 per cmm.; diff. leukocyte count—poly. 40.5%; lymphocytes (small) 53.0%; lymphocytes (large) 4.0%; monocytes 5%; polymorpho. eosinophils 2%.

21-10-'43. Total leukocyte count 6,875 per cmm.; haemoglobin 35%; diff. count—poly neutrophils 49%; lymphocytes (large) 3%; lymphocytes (small) 42%; monocytes 1%; poly. eosinophils 5%.

9-11-'43. Haemoglobin 12% (2.04 grms. per 100 c.c.); red corp. 1,100,000 per cmm.; white corp. 20,614 per cmm.; diff. count—myelocytes 2%; neutrophils 9%; lymphocytes 73%; monocytes 15%; eosinophils 1%. Abnormal red cells—neucleated cells in large number 22%; poikilocytosis marked; anisocytosis marked (helo 7.64%); polychromatophilia slight; malarial parasites nil.

Past history.—At the age of 2 months he had an attack of diarrhoea with mucus (colitis) with slight fever. M & B 693 tab. $\frac{1}{2}$. Entero vio form tab. $\frac{1}{2}$, C.I. Gluconate gr. ii, T.D. was prescribed. The boy was cured within three or four days. Otherwise he was healthy and well nourished.

Family history.—Mother and father (Hindu Bengali) are both strong, healthy and young. Delivery was normal. This baby was the 2nd child. 1st child is 3 years old and is very healthy. The mother had a miscarriage about 5 years back. The case is interesting because leukaemia is rare in a baby so young.

Observations on Some Cases of Acute Bacillary Dysentery Treated with Dagenan

K. C. GHOSH, L.M.F.

Medical Officer, Red Bank Tea Estate, Duickong P. O. Jalpaiguri Dist.

THOUGH much has been communicated through the columns of medical journals regarding the dramatic effect of Dagenan in bacillary dysentery, still there are some doubts in some schools which raise the question of its total absorption in the upper gut, and that to be effective in acute bacillary dysentery, it should act locally. Having none at my disposal, I am sorry, I have no experience with those Sulphonamides, e.g. Marshall's Sulphanilyl Guanidine, or Succinyl Sulphathiazole recommended for their non-absorption in the upper gut, and cannot, therefore, compare their effect with that of Dagenan in this disease. For economy of space and for the fact that there were no noteworthy differences in respect of symptoms in my series of cases, I have decided to communicate only three of them with a view to lend my humble support to the advocates of Dagenan in bacillary dysentery. From my humble experience I am firmly of opinion that Dagenan is the drug of choice, specially in treating children for this acute condition.

Case No. 1. A male child, son of a tea garden Babu, aged two years, began to pass only mucus and blood along with a rise of temperature from 3 p.m. on 2-5-'43. I was informed after the child had five such motions. I saw the case and found his condition as follows:—

Temperature 102° F; spleen not palpable; tongue—coated, not dry; stools were frequently passed, alkaline in reaction, consisted of mucus and blood with traces of faecal matter, generally green, sometimes

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single mass of mucus. There was tenesmus; no vomiting and no rigor. For want of facilities, examination of blood for M.P., or ordinary examination of stool to help the diagnosis could not be done. There was no case then in the neighbourhood diagnosed and treated as bacillary dysentery. However, taking the case to be bacillary dysentery, dysentery and intestinal phage of two different reliable firms—which being only available—were given combined with the hope of mobilising at least two different strains. The first dose was bigger and the following doses were adequate and repeated at three hours interval.

The child passed eight more stools during the night, character remaining the same microscopically.

3-5-'43. Phage was continued as before. Temperature came down by 1° F. in the evening. Other conditions and character of stools remained the same. He had 19 motions during the last 24 hrs.

4-5-'43. Finding no improvement in his condition (temperature again 102° F., motions as before and some prostration) Tab. M & B 693 was prescribed in $\frac{1}{4}$ gr. doses 4 hourly, preceded and/or followed by Sodi Bicarb cum Glucose drink. Tablets were finely powdered, gr. v of Sac. Lactis was added to each dose and administered suspended in water. Sac. Lactis was added with the idea of sweetening the powder and for the reason that it was recommended as one inhibiting the growth of dysentery bacilli. It is apparent that no higher initial dose nor generally recommended doses were administered subsequently as the infection was thought not so severe. In the evening temperature touched normal and the patient passed altogether 7 stools from 7 a.m. to 10 p.m.—mucus and blood gradually disappearing. From 10 p.m. till next day, no stool was passed and the child slept quietly.

5-5-'43. One motion at 6 a.m.—no mucus or blood was noticed. Dagenan was continued in the same dosage but every 6 hourly, from 12 noon. There was no rise of temperature afterwards and he had two more motions uptill next morning. There was faecal matter in the stool which was not formed. No mucus and blood were seen as stated by his father.

6-5-'43. General appearance was very good, the child was more lively. No motion during 24 hrs. Dagenan 8 hourly in the previous dosage was continued.

7-5-'43. Dagenan two doses were only given. One formed stool clear of any mucus or blood was passed. From next day Dagenan was discontinued and no after treatment was given. The child had no relapse.

Case No. 2. A female child, aged one year, daughter of a tea garden Babu, and next-door neighbour of Case No. 1, had frequent passage of mucus and blood and a rise of temperature from 12-5-'43. Character of her stool being the same as in Case No. 1, with the variation that her motions were less frequent and there was less blood in comparison with Case No. 1. Suspecting this case also to be a bacillary infection M & B 693 was given in the same manner and doses as in Case No. 1. Total of 12 doses were given and she was relieved of her symptoms.

Case No. 3. A boy aged about 14 years had an attack of malignant malaria on 15-9-'43. His temperature remained normal throughout the 19th and 20th after he had three intramuscular Quinine injections of gr. 10 each. He was getting over, but from 21st morning he was passing frequent stools—only mucus without having any rise of temperature. There was tenesmus. He had ten motions of similar character upto 2 p.m. when

I saw the case. The stool was alkaline in reaction and the masses of mucus seemed to bear a very slight greenish tinge. Thinking this also an attack of bacillary dysentery, tab. M & B 693 one, finely powdered and compounded with sugar of milk, was given four hourly preceded and/or followed by a dose of Glucose Sodi Bicarb drink upto 6 doses with marvellous result, frequency being much diminished and faecal matter appearing with the lessening of mucus. Dagenan was then administered 6 hourly upto 4 doses. From the third day of this dysenteric attack he passed formed stools clear of any mucus or the like. As maintenance doses the drug was continued for the next two days twice daily in the above dosage. The boy required 14 doses in all—14 tabs. to get a clinical cure and had no relapse of the symptoms.

For want of facilities no microscopical examination of stool of my cases could be done. They were diagnosed solely based on typical signs and symptoms—which are no less important from diagnostic point, —and treatment with M. & B. 693 was very successful.

Remarks.—Though purgative treatment still maintains its position and though most of my previous cases of acute bacillary dysentery were treated and cured by Mag. Sulph., Sodi. Sulph., Kaolin, Bismuth, etc., Dagenan or M. & B. 693 now appears to me the drug of choice, specially for children, where very frequent passage of watery stools caused by Magnesium and Sodium salts are very distressing to the patient and annoying to the parents. The question of availability, price, and possible risk of anaphylactic phenomenon of serum are also considered along with my experience in making the above remarks. It required only 15 doses = $2\frac{1}{2}$ tabs. in case No. 1, 12 doses = 2 tabs. in case No. 2 and 14 doses = 14 tabs. in case No. 3 of 0.05 gm. each, which were evidently smaller than generally advocated and were found effective. The smaller quantity required might be due to the moderate infection. I am not very sanguine about phage in bacillary dysentery from the humble experience I have had. Case Nos. 1 and 2 at least, in all probability, seem to be a shiga infection, where bacteriophage therapy is expected to give good results.

The time required in these cases to effect a clinical cure though not compared with other methods is evidently much shorter. Here, in these cases average of only 4 days of Dagenan therapy was necessary. If given in smaller doses, the question of its total absorption in the upper gut and that none reaches the seat of trouble was raised with the presumption that to be effective in bacillary dysentery it would act locally. It is possible that the drug, even if administered in comparatively smaller doses, reaches safely the seat of trouble, being hurried down by increased peristalsis,—allowing only a fraction to be absorbed above. The portion which is hurried down seems to be adequate to effect a cure. Even if it is admitted that Dagenan is beneficial in bacillary dysentery by acting at the seat of trouble, there must have been some systemic action too.

Lastly, I am tempted to quote a line from the editorial, I.M.G., December, 1942 which I think, will prove to be true. "It seems possible that the treatment of bacillary dysentery, which in the past has never been satisfactory, is entering a new and more promising era."

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[1944]

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(Salicyl, Hexamine, Caffeine preparation)

Ampoules of 5 c.c.m. for Intravenous or Intramuscular Injection.

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Each Ampoule of 5 c.c. contains:—
Hexamine ... 2.0 Gm.
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E.g., Cystitis, Pyelitis, Pyonephrosis etc. **CASYLOTROPIN** is specially efficacious in Chronic Cystitis set up by Prostatic Hypertrophy and accompanied by alkalosis. It is also used with success as a Prophylactic to prevent Cystitis after Prostatectomy.

One Ampoule of 5 c.c.m. is injected every other day until urine remains permanently clear with acid reaction.

2. Infectious Inflammation of Bile Passages.

E.g., Chole-Cystitis

One Ampoule of 5 c.c.m. is initially injected daily when symptoms decrease, 5 c.c.m. is used every other day.

3. Rheumatic Affections.

CASYLOTROPIN lessens the Swelling, leads to a rapid cessation of Pain diminishes the liability to Pericarditis and other complications of rheumatic cases.

One Ampoule of 5 c.c.m. is injected twice a week or once a week according to the severity of the case.

Mode of Action

When **Casylootropin** is injected, the Hexamine is possibly split up in the system into Formaldehyde and Sodium Salicylate into Salicylic Acid. These split-up products act as formidable enemies to the ducts act as formidable enemies to the causative pathogenic germs or bacteria responsible for the indicated diseases. The Caffeine moiety of **Casylootropin** keeps up the Heart and act as a mild diuretic.

Clinical Results: Since the introduction of **Casylootropin** by the Medical Research Laboratory in the year 1934 extensive trial has been given by quite a large number of qualified Medical men in Bombay, Punjab, Delhi and in Bengal with uniformly successful results in varieties of complications indicated above. In cases of Chole-Cystitis **Casylootropin** is said to have acted like a magical cure.

About 75% swelling of knee-joint and the acute agonising pain of the Rheumatic patients are said to have been removed by one or two doses administered intravenously by the Medical Officers of District Hospitals in Bengal. One course of 6 injections on an average, cured the patients permanently.

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Urethane q. s.

Aqua (B.D. Distil and Sterilised) to 2 c.c.

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(ii) In anemia arising out of repeated attacks of Malaria.

(iii) Ague and all other fevers with the exception of kala-azar.

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(d) Stimulates in short, the whole system—a completely broken down system of malarial patients being reorganised.

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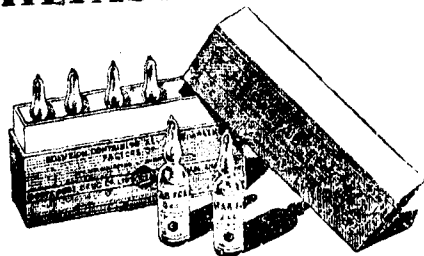
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No. 8.

Help Bengal

FOR one long year, Bengal has been battling against a calamity unparalleled in its annals. The Bengal Famine of 1943 took an alarming toll of human lives and left behind it a far more terrible legacy. Continuous starvation and malnutrition, arising from living on inedible roots and leaves, left the mass of people devitalized and an easy prey for the waves of epidemics which followed. We are told that it took a toll of 35 lakhs of her sons and daughters and that the rest numbering about 60 to 70 lakhs are left wandering like nomads disseminating diseases and to die under miserable existence.

In the wake of famine, epidemics—malaria, scabies, smallpox, dysentery—have broken out. 18 districts have been declared by the government as malaria and smallpox areas. Workers in Bengal report that more than 60% of the people are stricken with malaria in certain districts and nearly 45% are suffering from small-pox. Hunger and want have driven the poor people to mass prostitution and we are told that venereal diseases are widespread in most of the districts in Bengal.

On the top of all these comes the news of rise in prices, scarcity of food and the possibilities of a second famine. We learn from authoritative sources that only 20% of the people get rice at controlled rates while others suffer, and that in East Bengal the position is so acute that rice sells at Rs. 60/- per maund, which is 4 times the controlled price. We are told that one of the most important factors that led to the last famine was the triumph of the hoarders who took advantage of disunity of political parties and of leniency on the part of the bureaucracy, to grab all stocks and sell them at exorbitant and prohibitive prices.

We learn also from reliable sources that there is great difficulty in getting proper supply of medicines. The Government of Bengal, we understand, have released 1,24,000 lbs. of Quinine so far. But the workers tell us that even double this would be a bare minimum. The report of the Madras Medical delegation confirms this. We are also told that Quinine does not reach ultimately the patient in its pure form. It is quite possible that these medicines find their way to the hoarders from whom they emerge at exorbitant prices. It would be well if the Government place a stricter watch over the distribution of Quinine and see that every patient receives it in its proper form and in its adequate dosage.

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This is only one side of the picture. The patriots and people of Bengal are making heroic efforts to fight the epidemics. The medical men of Bengal took the initiative and brought all patriotic parties and relief organisations into a single organisation to fight epidemics, the Bengal Medical Relief Co-ordination Committee with Dr. B. C. Roy as its President and Drs. B.N. Basu and B.K. Basu as Secretary and Field Inspector respectively. The Muslim League Relief Committee, the Bengal Civil Protection Committee, People's Relief Committee, Dr. Shyamaprasad's Bengal Relief Committee, the Indian Medical Association, and the United Teachers and Students' Relief Committee are among those represented in this Central Organisation. The appeal made by these organisations have evoked very good response from all over the country. Thanks to the efforts of these organisations, more than 70 medical units are working in 22 districts of Bengal and during the last few months have treated more than 10 lakhs of people. 12 medical delegations have gone to Bengal from other provinces and are working side by side with the Bengal units.

Madras has played its part in the campaign against this disease. The Madras Students Organisation has sent a medical delegation to Bengal in co-operation with the Student Christian Movement. The squad consisted of 15 senior medical students and was equipped with Rs. 5,000 in cash and medicine worth Rs. 1,500. The Madras Muslim Students' Federation also sent a medical squad consisting of 6 students. A Committee of the citizens of Madras which was constituted in the city to intensify and co-ordinate all efforts for the relief of Bengal, has done some useful work and has so far collected over Rs. 15,000. But all this is not enough. Dr. B. C. Roy reminds us that 2 crores of people are in the deadly grip of epidemics. He estimates that 1,500 medical centres are necessary if "epidemics are to be effectively fought," but he thinks 200 centres will "save Bengal from ruination." He has appealed to raise Rs. 5,70,000 to run these 200 Centres for the next six months, each Unit costing Rs. 4,500 to run for 6 months.

Last year patriotic India rose to a man to succour famine-stricken Bengal. The call has again come. Let us act like true sons of India, wide awake to reality and conscious of our duty to the people of Bengal. Bengal, the home of Indian Research and Science, the home of Tagore, Bose and Ray, is fighting the greatest medical and social problems today. Its call is irresistible. The Medical Profession must answer it back.

We hope this appeal of ours will not fall on deaf ears. We earnestly appeal to everybody to help the Peoples' Relief Committee and render relief to devastated Bengal.

We trust doctors will be willing to volunteer for this humanitarian and patriotic service and help to fight the epidemics. It lies in the power of every doctor to save thousands of our countrymen from death. This way alone can we vindicate the honour of our noble profession.

To Our Subscribers

As a result of the Paper Control (Economy) Order of the Government of India, we have been obliged, much against our wish, to reduce the number of pages. We need hardly say that we shall revert to the original format as soon as the present restrictions are withdrawn.

Gleanings From Medical Press

MEDICINE and THERAPEUTICS

Cause and treatment of sprue.—The cause of sprue is unknown, but it is suggested that a dietetic deficiency, possibly some components of the vitamin B₂ complex but not of nicotinic acid only, brings about a metabolic breakdown leading to interference with the normal absorption, particularly of fats, from the alimentary tract. The basis of treatment consists in a high-protein, low fat, low carbohydrate diet. The total initial amount of diet depends on the severity of the disease and the tolerance of the patient, and is gradually increased as the patient improves. The absorptive failure in sprue may be associated with secondary nutritional defects. Commonly there is a macrocytic anaemia requiring the administration of liver extract—e.g., equivalent to half pound fresh liver daily while vitamin C and nicotinic acid, e.g., 200 mg. daily are important. Riboflavin may also be indicated where there is stomatitis, and hydrochloric acid in the presence of achlorhydria. Under proper dietetic treatment the symptoms usually subside rapidly and the patient steadily improves, but occasionally diarrhoea or meteorism may be troublesome for a time; in such cases castor oil followed by powdered chalk, charcoal, and kaolin may be useful. Any associated infection, e.g., B. coli urinary infection, which is not uncommon, should be treated on ordinary lines.—B.M.J.

Spontaneous pneumothorax.—Kuchler states that within a comparatively short time spontaneous pneumothorax may produce threatening symptoms such as piercing thoracic pain, angor, severe dyspnea, cough, pain in the epigastric region and cyanosis. In other cases pneumothorax is hardly noticeable; the patient is at the most somewhat dyspneic. Sometimes spontaneous pneumothorax is discovered accidentally in the course of a roentgenoscopy, and in other cases it may remain unrecognized. Diagnosis can be based on the loud percussion sound, greatly decreased or completely lacking respiratory sounds, enlargement of the involved half of the chest, smoothing over of the intercostal spaces, differences in form and movement of the two halves of the thorax and lessening of the voice fremitus over the pneumothorax. Percussion often reveals shifting of the heart and mediastinum. The percussion sound has a decidedly tympanitic overtone as long as the air in the pneumothorax is not under great pressure. As the pressure increases, the percussion sound becomes more like that over a normal lung. Respiratory and heart sounds are usually weak. Amphoric respiratory sounds are often typical of pneumothorax, but they occur also in the presence of large cavities. Metallic sounds are likewise frequent in pneumothorax. Roentgenoscopy gives the most conclusive evidence

of pneumothorax. If severe dyspnea and cyanosis exist, relief must be provided at once by insertion of a hollow needle anteriorly into the first or second intercostal space. If dyspnea recurs soon, the needle must be left in situ. A valve that permits escape but prevents external intake of air can be improvised from a rubber finger-stall. The patient should be hospitalized as soon as it is possible to transport him. If dyspnea continues to return after repeated withdrawal of air, it is best to introduce a permanent cannula with valve. Dyspnea as well as cough must be suppressed by medication with a combination of morphine and atropine. The patient should be kept in the sitting posture, and roentgenologic observation should be continued. It has been advised that the pneumothorax be continued for from six to nine months after closure of the pleuropulmonary fistula. After the pneumothorax has been absorbed, the patient should be advised to avoid sudden movement and the lifting of heavy loads. In case of spontaneous pneumothorax on a tuberculous basis, a prolonged pneumothorax treatment becomes necessary so that the subpleural tuberculous changes may have a chance to heal. Emphysema blisters may be treated by touching them with the cautery under thoracoscopic guidance. Traumatic pneumothorax in the war wounded demands that the wound be closed in order to avoid further intrusion of air.—J. A. M. A.

Cerebral thrombosis.—In the acute stage of cerebral arterial thrombosis, treatment is directed towards preventing extension of the thrombus by increasing cardiac efficiency, diminishing the clotting power of the blood, and reducing progression of arterial degeneration, which is the prime cause of the thrombus. (Any cause for the degeneration, such as renal disease, diabetes, or syphilis, should be sought). Rest, fluids and glucose are needed; purgation should be avoided, and, if fibrillation or cardiac embarrassment is present, digitalis should be given. Injections of strychnine may also help. The clotting time can be increased by injections of heparin, and sodium iodide by mouth is said to diminish the spread of a thrombus, though this is very doubtful. Once the acute stage is passed the patient should be got up, and exercises, massage, and movements of the affected limbs should be undertaken. It is most important to keep the muscles working and the patient alive.—B.M.J.

Localized postural oedema.—It is more frequent than is generally realized. It often gives rise to needless alarm and is always troublesome to treat. In health, the filtration pressure of the blood in the capillaries is balanced by the osmotic pressure of the plasma proteins. The high venous pressure

in the lower extremities, particularly in the erect position, entails a rise in the capillary pressure and therefore in the tendency to filtration of fluid into the tissues. Transudation of fluid into the legs on standing can be demonstrated in both males and females, and distinct oedema will occur if the foot is allowed to hang down motionless for some time. Normally fluid does not accumulate, being expressed from the limbs by muscular movements and changes in position. It is a little difficult to see why the mechanism should break down and oedema develop in women, particularly young women. Diminished elasticity of the tissues is one factor, more particularly in association with tight shoes, high heels, and wrong posture. The water retaining properties of the female sex hormones are probably also important, and it may be found that the oedema is worse about the time of menstruation. Local treatment is more effective than general, and consists in avoidance of high heels, massage and exercises of the ankles, contrast baths, and strapping with adhesive tape. Sometimes a formal manipulation of the ankle may be desirable. Tight garters, etc., should of course be avoided and over weight should be reduced. Of general measures, restriction of salt is probably the most rational. Calcium salts are indicated in theory but disappointing in practice. Small doses of thyroid and large doses of alkalies have also been used.—*B.M.J.*

Gold in arthritis.—If gold is to be effective in arthritis it must be given in a form which is easily absorbed and which will remain in the circulation long enough to reach the site where it will exert its action. Such conditions are probably best obtained by intramuscular injections of the water-soluble gold salts. These drugs are potentially toxic and are retained in the body for a long time after the period of administration. As there is no known method of inhibiting the toxic action, which may appear late in the treatment when the patient has large amounts of gold in his system, it is essential to restrict dosage to the smallest amount known to be effective. When crystalline salts are used the equivalent of 25 mg. gold weekly, which in the case of myocrisin is contained in 50 mg. of the salt, is the minimal effective dose, and the total quantity of gold given should not exceed 1 gramme. Using this amount, the incidence and severity of the toxic reactions fell from 41 to 17 per cent, and there were no cases of nephritis, exfoliative dermatitis, or other severe reactions. Smaller doses appeared to be less effective in the treatment of arthritis, and colloidal gold sulphide gave little or no benefit. So far no dosage of gold can be recommended which is both therapeutically effective and yet below the level of toxicity. This makes it all the more important that only cases likely to obtain benefit should be selected for this treatment and it is difficult to do this, as it is still unknown where, how, or why gold salts work. Major errors can be avoided if it is

realized that the only possible action of gold is to lessen or arrest inflammation. No benefit can be expected in osteo-arthritis, which is a degenerative non-inflammatory joint disease, or in old-standing rheumatoid arthritis with irreversible bone and joint changes. Only patients in the active inflammatory stage of rheumatoid arthritis are suitable candidates for the treatment; and even in them improvement is uncertain, and there is always a risk of toxic complications.—*Editorial.* Treatment of chronic arthritis with gold. *Brit. M. J.*—329-330, March 4, 1944.—*Naval Medical Bulletin.*

Therapeutic procedures in bronchial asthma.—W. C. Spain, New York State J. Med. 41: 1631 (Sept. 1) 1942.

Since it is well established that allergic conditions are inherited, infants and children of an allergic family should be protected from the commoner inhalant and dietetic antigens. Dogs and cats should be banned from the household. Feather pillows, down, cotton and kapok bedding should be replaced. The excessive use of nuts and chocolate must be avoided, and children should not be coaxed to eat food that they instinctively dislike. Particularly, exposure to excitants identified by history or by cutaneous tests should be prevented. This principle of escape from the influence of home surroundings and diets is probably responsible for the development of many institutions and spas.

Various drugs used in the treatment of asthma are discussed. A 1:100 solution of epinephrine hydrochloride as a spray for inhalation, should be used with caution in children, since desquamation of the tracheal epithelium and loss of cilia may result. Theophylline alone or combined with ethylenediamine is especially useful in severe asthma. Morphine is especially dangerous, as it suppresses the respiratory center, abolishes the cough reflex and permits development of mucous obstruction. Patients may also become sensitive to morphine. Sulfadiazine may well be used to control infections of the respiratory tract, since such infections increase the mortality in bronchial asthma. Belladonna, calcium, histamine and potassium chloride have not yet been proved of great value. The intratracheal injection of iodized oil is "of passing value" and not without danger.

Purgation, starvation and continuous rest in bed, the use of potassium and sodium iodide and other old time methods may be tried with advantage. Above all, proper climate is of advantage in treatment; Spain advises the high altitudes of the Southwest, particularly in Arizona and New Mexico. *AIKMAN, Rochester, N. Y.*—*A. J. D. C.*

Sensory effect of wounds.—Burrows states that sudden severe wounds are at first painless and the recipient cannot always identify with precision the site of his injury. In war-time it is comforting to reflect that severe wounds are always painless until some time has elapsed after their

infliction, and that by the timely use of morphine the reactionary pain may be greatly and perhaps entirely prevented. Attention is also called to the reduced sensibility and muscular power in wounded limbs. Wounds of arteries and periarterial tissues in the arm and leg are followed by a reduced appreciation of all cutaneous stimuli and a general muscular weakness in the injured limb. The area of reduced sensibility is of the glove or stocking type and at first may extend above the wound. In the course of days or hours normal sensibility returns, the proximal zones of the limb being the first to recover. Diminished feeling of this kind has some resemblance to the numbness produced by cold and so the numbness produced by its recognition. A needs similar tests for its recognition. A curious condition follows wounds which traverse the interosseous space of the forearm or penetrate the tissues near its ventral surface. The essential feature seems to be a permanent loss of adaptability in the hand to changes of external temperature. The hand appears slightly swollen; in warm weather it is redder than its fellow and in cooler weather it becomes cyanosed. There has been found a reduced sensitivity of the affected hand with an incapacity for accurate manipulation such as occurs in normal persons when their hands are ischemic with cold. The condition appears to be associated with a paralysis of the small blood vessels of the hand and possibly may be the consequence of sympathetic nerve injury.—*J. A. M. A.*

Sulfonamide therapy of diarrhoea.—Halpern and Cunningham made a study of the effectiveness of sulfathiazole and sulfaguanidine in the treatment of acute diarrhoea. Sulfathiazole was used in 23 children in an initial dose of 1 grain per pound, followed by 1 grain per pound, divided into six equal doses. Sodium sulfathiazole was given intravenously to patients who were critically ill. The drug was discontinued 24 hours after the stools became normal. Blood levels ranged from a trace to 13 mg. per cent, with an average of 2.6 mg. per cent. No toxic effects from the drug were noted. Fever and toxicity subsided within

24 to 36 hours. In an average of five days the stools become normal in number and of good consistency.

Sulfaguanidine was given to 13 children, five receiving doses equivalent to those of sulfathiazole and the remainder receiving 3 grain per pound initially, followed by 2½ grains per pound, in six equal doses during the 24 hours. No unfavorable reactions were noted. Blood levels ranged from a trace to 2.25 mg. per cent, averaging slightly more than a trace. The clinical course was similar to that of the children treated with sulfathiazole. Stools became normal in an average of 5.4 days.

As a control, 11 children received no specific therapy. Their stools became normal in 12.5 days, but fever and toxicity persisted for many days and there was a prolonged convalescence.

Tudor made a similar study to compare the efficiency of sulfathiazole and sulfaguanidine in the treatment of infantile diarrhoea, treating 16 patients with sulfathiazole and 15 with sulfaguanidine from the day of admission to the hospital to the fourth or fifth day after their diarrhoea and other symptoms had ceased. Dosages of sulfathiazole for infants under one year of age were 1 Gm. on admission and 0.5 Gm. every four hours thereafter. The dosages of each of these drugs were doubled for infants over the age of one year. The only toxic reaction noted was prolonged fever in two patients.

Of the 31 children, 16 had clinical bacillary dysentery and 15 had parenteral diarrhoea. No pathogenic bacteria were isolated from any of the stools, which were cultured daily for three days after admission. Except for three patients, who did not improve on sulfaguanidine, the two drugs were equally effective in both the dysentery and the parenteral diarrhoea cases.

The author's impression that sulfonamides were beneficial in checking the diarrhoea in one case of typhoid fever and in three cases of paratyphoid A is at variance with the report of Hall, who noted no change in the course of five patients with typhoid fever and one with salmonella gastroenteritis after the administration of sulfaguanidine.—*Medical World.*

SURGERY

are nearly always cystic disease but a few are multiple fibroadenomas, and rarely a gelatinous or medullary carcinoma may produce this condition.

4. Multiple adherent masses: This type of breast nearly always represents an advanced carcinoma.

(a) *Acute carcinoma of the breast:*—This rapidly developing form of cancer has the appearance of an acute mastitis. The breast is swollen, reddened and tender, and there is increased local heat. The tumour is highly malignant and infiltrates the lymphatics of the breast and axilla extensively and rapidly. The prognosis is extremely unfavourable.

Tumours of the breast.—The correct interpretation of tumours of the breast involves a clinical examination of the breast as well as a study of the tissue that is removed. It is very important to examine the breast by gentle pressure with the open hand.

1. A single adherent tumour: When the tumour is adherent to the skin, or deep fascia, it is nearly always malignant. The exceptions are fat necrosis and occasional cases of mastitis.

2. A single non-adherent tumour: In young women these are chiefly benign but in older women many of them are malignant.

3. Multiple non-adherent masses: These

(b) Paget's disease of the nipple appears as a stubborn dermatitis of the nipple. The tumour is usually regarded as a primary carcinoma of the large ducts of the nipple.

(c) Mastitis of puberty: In boys and girls about the time of puberty a mass in the breast may develop which is sometimes mistaken for a neoplasm. It is a small button-like indurated mass about the nipple. The nipple is exactly in the centre of the growth. Microscopically one finds a moderate infiltration with large lymphocytes and some hyperplasia of the glandular tissue of the breast. The mild inflammatory reaction is probably a response to endocrine stimulation. It requires no treatment other than a protective dressing.

(d) Discharge from the nipple: A discharge from the nipple may be either serous or bloody and may occur with or without the presence of a palpable tumour. If a tumour is present this should be removed by biopsy and examined. It may prove to be a carcinoma or a duct papilloma.

When no palpable tumour is present the discharge is due either to a duct papilloma, a cyst communicating with a large duct or a simple ectasia of the large ducts. A conservative operation is indicated. If the discharge is serous and no tumour is palpable, surgical intervention is not necessary.

Malignant lesions are very rare before the age of 20 years, and they are infrequent in the third decade. In the first three decades the vast majority of the lesions are benign, in the fourth decade about three fourths are benign and in the fifth benign and malignant lesions are of equal frequency. After the fifth decade malignant lesions greatly preponderate.

BENIGN TUMOURS.—The benign lesions are (1) fibroadenoma, (2) cystic disease, and (3) chronic mastitis.

1. Fibroadenomas attain their maximum frequency in the third and fourth decades but a few are found in advanced life. These tumours are usually single but may be multiple. They are hard and freely movable. They should be removed because an occasional tumour becomes a sarcoma.

2. Cystic disease reaches its peak in the fifth decade and practically disappears after the menopause. There may be a single mass in the breast but usually there are multiple non-adherent tumours. Upon gross section one may see cysts or solid masses of tissue. Microscopically there are two forms, a simple ectasia of the ducts and an adenomatous growth of the lobules with or without the formation of cysts. In making the microscopic diagnosis, it is very important to examine the section under low magnification. In adenocystic disease the epithelial masses are always arranged in lobules while in carcinoma the lobular arrangement is lost. Cystic disease is not a pre-cancerous condition. Having established the diagnosis by biopsy it is safe to leave the rest of the breast undisturbed. This conserva-

tive treatment is especially recommended in young women.

3. Chronic mastitis. (a) Chronic exudative mastitis resembles chronic inflammations elsewhere except that the escape of secretion leads to the formation of pseudo-tubercles which may be mistaken for true tuberculosis.

(b) Chronic fibrous mastitis is applied to fibrous induration of the breast. There are many bonafide examples in which a part or the entire breast is thickened, hard and fibrous. Microscopically, there is merely an increase of dense collagenous tissues. However the majority of cases which the pathologist finally calls chronic fibrous mastitis represent normal breast tissue in which the glandular elements are atrophic. The surgeon picks up the lobules of the breast between his fingers and thinks he feels a tumour.

CARCINOMA.—Carcinoma of the breast are usually subdivided into scirrhous, medullary and gelatinous. The medullary and gelatinous forms are non-adherent but there is not much difference in their malignancy.

An important type is the comedo tumour. This is a solid growth which on pressure yields a column of fatty material not unlike that which may be expressed from a black-head. On microscopic examination one finds large duct-like structures filled by growing epithelium, and there is uncertainty as to the interpretation. Bloodgood found that nearly all the women with comedo tumours were well five years after radical mastectomy, and he suggested that the growth is not malignant. However, it is dangerous for the pathologist to diagnose a comedo tumour as benign on the basis of biopsy alone, since, after removal of the breast, one frequently finds areas of definite carcinoma. The safest procedure, therefore, is to recommend mastectomy. If, after thorough examination of the breast, no areas are found except the ductal proliferations, the axilla need not be dissected out.—G.P.

Baker's Cyst.—There are numerous references in the literature to cysts of the popliteal region. A Baker's cyst is a true herniation through the posterior wall of the joint capsule. To be a true hernia, the sac must be connected with the joint. It must be lined with synovia and connected with the joint by either a normal or abnormal opening.

The syndrome is fairly definite. Usually the hernia develops insidiously. Occasionally, because of trauma or inflammation, an acute effusion occurs and the sac is pushed backward along the path of least resistance. In the early stages, there may be only vague pain and a sense of fullness, which may be intermittent. The symptoms may suggest an internal derangement of the knee. As the cyst increases in size, the swelling extends down the popliteal space, under the deep fascia.

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CLEANINGS FROM MEDICAL PRESS

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Examination usually shows a soft, non-tender, fluctuant mass along the lateral aspect of the semimembranosus tendon. This cyst must be differentiated from a number of other entities, including lipoma, hyperplastic bursitis, fibrosarcoma, angioma, and arteriovenous aneurysm.

The treatment is palliative or surgical. Injection of a sclerosing material is contra-indicated.—Kuhn, H. H., and Hemphill, J. E., *Baker's Cyst; Posterior Herniation of Knee Joint*. *Radiology* 42: 237-240, March 1944.

Hand infections treated with penicillin.—Flory and Williams chose acute infections of the hand to ascertain the possibilities of using penicillin in acute pyogenic infections. The complicated structure of the hand permitted observations of the effects of penicillin on skin, areolar tissue, blood vessels, nerves, muscles, tendons, synovial membranes of tendon sheaths and joints and bones. A comparative study was made of 212 infections. About half were treated by current methods and the other half by local application of penicillin superimposed on the usual surgical procedures. The great majority of control patients remained septic for well over a week and nearly three fourths were infected till their wounds healed. In the penicillin treated series pus was eliminated within a week. Pain was scanty or absent. Relief of pain and throbbing and improvement in the general condition have been striking, and penicillin has effected a great saving in the number of dressings. The mean healing time has been considerably reduced by penicillin, but a few cases have shown undue prolongation of healing. Other factors may have caused this. In bone inflammation penicillin treatment led to rapid rarefaction concurrently with improvement in the clinical condition. Reformation of bone tissue took place readily. The rapid return of mobility to infected parts was one of the most striking features of the penicillin series, but complete normality was not regained after tendon sheaths had contained pus. The dry impure calcium salt may have harmful effects on wound surfaces not protected by granulation tissue. Some patients complained of burning when the dry salt was used. Among chronic cases penicillin proved of value in preparing infected surfaces for skin grafting and for protecting stumps for amputation and for procuring complete healing in chronic dermatitis. The total amount of penicillin used to treat more than 100 cases was not more than 500,000 units. Complete figures for 35 of the penicillin-treated patients showed a saving of about 1,000 man-days of working time as compared with the controls. These results have been obtained by careful attention to technique and consistent twenty-four hourly dressings for a minimum period of five days.—J.A.M.A.

Mammary cancer and menopause.—Dawson says that mammary cancer which shows the typical textbook picture is probably in the majority of cases already beyond cure. In all doubtful cases delay in treatment is justified only if there is proof that the condition is not malignant, and the only proof is histologic. Delay in diagnosis would be largely prevented if the principle was accepted that few considerations, apart from a young age, rule out the possibility of mammary cancer. As the age period rises, the probability of cancer increases. This is illustrated by a recent survey by Harvey and the author of all mammary lesions reported on, in one year, at their laboratory. In the period under review, 274 cases were studied. It was found that in patients over the age of 65 years all mammary lesions were carcinomas; over 60 years 95 per cent. were carcinomas; over 55 years 92 per cent. were carcinomas; over 50 years 85 per cent. were carcinomas; but under 40-85 per cent. were benign; under 30 all were benign. During the "reversal period," that is between 40 and 50 years, there occurs the point at which benign and malignant tumor percentages are equal. During this period doubtful mammary lesions present difficulties. The pathologist may not be able to give a positive verdict if only a small piece of tissue is submitted. If this does not show cancer he can still give no assurance regarding the condition of the rest of the breast. It is difficult for the pathologist not to be biased in favor of simple amputation in these doubtful mammary cases of middle age. No sarcoma was found among the malignant lesions studied by the author; more than half of the non-tumor lesions were single or multiple cysts. The author emphasizes that the age of the patient carries much weight in the diagnosis of mammary cancer. Sarcoma may be found at almost any age but tends to occur at the earlier age periods; carcinoma, a much more frequent form of malignant growth, is predominantly a disease of middle and later life. The normal growth and functioning of mammary tissue is primarily dependent on the ovarian hormones. The subsidence of reproductive activity with the gradual loss of ovarian stimuli during the climacteric implies the lessening of this control. The author describes the cellular changes which eventuate in carcinoma. The transition to malignancy may be due to (a) the loss of ovarian control over normal growth and stability of tissue in the mamma, (b) the action of other stimulating growth factors which are given opportunity as ovarian control subsides, or (c) some expressly carcinogenic principle, formed possibly by pervergent metabolism, because hormonal control is absent or aberrant; the instability of the mammary tissues at the time would then act as a predisposing factor. The large proportion of mammary and genital cancers, in relation to the total malignant tumour incidence in women, lends some probability to all three conceptions.—J. A. M. A.

OBSTETRICS and GYNÆCOLOGY

Abnormal uterine bleeding after middle age.—Mussey and Wilson review the records of 200 women who registered at the Mayo Clinic between July and September 1937 because of abnormal uterine bleeding. The cases were selected from 1937 so that the results of treatment could be reviewed after an interval of five years. Many women past the age of 35 or 40 view abnormal bleeding as a manifestation of "change of life" and fail to consult a physician. This results in many avoidable deaths. Although abnormal vaginal bleeding may occur without implying serious organic disease, no patient past the age of 35 years who notes abnormal uterine bleeding, especially metrorrhagia, should be dismissed until it is reasonably certain that a malignant condition does not exist. Women with such symptoms should not be given endocrine therapy until malignant disease has been ruled out. Many of the causes of abnormal vaginal bleeding can be discovered by thorough pelvic examination. This should include inspection of the urethral meatus and vaginal introitus and bimanual palpation of the cervix, uterus and adnexa. Examination of the vagina by speculum, with direct visualization of the mucous membrane and cervix, never should be omitted. Biopsy should be done of any lesion which arouses suspicion. Except in pregnancy, diagnostic curettage should be used in every instance in which there is doubt about the character of the uterine contents. The conditions responsible for the bleeding have been divided into benign, functional, malignant and postmenopausal conditions. After discussing these different conditions and their treatment the author says that, although a review of 200 cases should give a good sampling, relatively atypical proportions were found of (1) minor benign conditions causing abnormal uterine bleeding, (2) more severe benign conditions requiring hysterectomy or irradiation, and (3) carcinoma of the uterus. In an average office practice one should encounter a much larger proportion of minor conditions, proportionally fewer cases of fibromyoma and a still smaller proportion of malignant lesions. The authors found 80 cases (40 per cent.) of uterine fibromyoma and 27 cases (13 per cent.) of carcinoma of the uterus. Uterine bleeding in the presence of fibromyoma is usually successfully treated by hysterectomy if the patient has not reached the age of 40 or 42 years, to preserve ovarian function. Hysterectomy is done at any age if the tumor is larger than a uterus two and half to three months pregnant. Patients who are more than 42 years of age and whose uterus is smaller are given a dose of radium sufficient to bring on the menopause. Aside from exceptional cases, curettage should be performed prior to radium treatment. Radium, when used to control menorrhagia associated with fibromyoma, stopped the excessive bleeding in all but 2 of 17 cases. The results of total hysterectomy for carcinoma of the body of the uterus, with a rate of cure of 63 per cent. appears to justify continu-

ance of this treatment. The rate of cure of 37 per cent. for carcinoma of the cervix following irradiation with radium and x rays conforms to the usual results. The curability of early carcinomatous lesions is much greater than the curability of the more advanced. In a large majority of cases the warning sign of abnormal uterine bleeding was ignored too long.—J. J. M. J.

Ergotamine tartrate for sub-involution.—The chronic ergot poisoning in gangrene of the extremities. This is caused by a group of alkaloids of which ergotamine is a representative. Cases are recorded in which gangrene of the legs has occurred during the puerperium in patients who have been given ergotamine, but usually these women have also suffered from sepsis, and it is therefore difficult to judge the part played by the drug. Apart from child birth, ergotamine is often freely used by sufferers from migraine, apparently without ill effects. In view of the known properties of the drug, it is, however, prudent to limit its continued use to doses of 1 mg. twice daily by mouth for periods of not more than one week at a time. There is no convincing evidence that the continued administration of ergotamine or any other ergot preparation, influences uterine involution. In any case it can scarcely be supposed that such therapy would have effect two years after child-birth.—B. M. J.

The use of the Willett Clamp for scalp traction in delivery.—In January, 1941, one of the authors of this study published a preliminary report about the use of the scalp clamp in obstetrics, in which 23 cases were presented. Since then the scalp clamp has been used 71 times in the obstetric services of the University and Baltimore City Hospitals. This report by the authors concerns both series.

There have been a number of instruments devised for the purpose of scalp traction. In the majority of cases an ordinary hemorrhoid clamp proved quite satisfactory. The authors have used the scalp clamp in the following types of cases:

1. *Placenta prævia*.—It is used only in the marginal and partial types to tamponade the bleeding points as well as hasten labour by virtue of the irritation of the lower uterine segment. This procedure offers a satisfactory substitute for Caesarean section.
2. *Premature separation of the placenta*.—It is used particularly in the low implanted variety and in some selected cases of the normally implanted placenta. The mechanism is essentially the same as with placenta prævia. The Spanish windlass probably should be reserved for a few selected cases in which hemorrhage is an outstanding feature, or in combination with the clamp.
3. *Uterine inertia*.—It has been found that the traction force of this method brings the head in close contact with the lower uterine segment and stimulates the uterus

to contract normally. This method removes, in many cases, the necessity for the administration of oxytocic drugs, some of which have the tendency to further tire the uterus, whereas the gentle direct mechanical stimulation does not force the uterus beyond its normal contractile capacity.

4. *Induction of labour*.—The mechanism is the same as described for uterine inertia.

5. *Malpresentation of the vertex*.—Such cases may be corrected by vaginal or combined manipulation and the attachment of the scalp clamp to a portion of the fetal scalp at or near the occiput. The traction will insure the permanency of the newly converted position and thus provide relief from possible dystocia caused by the positional disproportion.

6. It may be applied to the floating head when the membranes are ruptured to prevent the prolapse of the fetal parts or cord.

7. Finally, this method can be used in cases of known fetal death to hasten delivery when this is indicated. Craniotomy and forceful extraction of the child is certainly less desirable than scalp clamp traction with the relatively small amount of manipulation that is required.

Ante-partum and intrapartum hemorrhage was the indication in 45 cases. In the 25 cases of uterine inertia, the character of the pains, as well as the frequency, was greatly improved in 22 cases. In the other 3 cases there was no change in the character.

The average duration of the latent period between the application of the clamp and the onset of labor in 38 cases was 1 hour and 24 minutes. The average duration of labor in these cases was 10 hours and 31 minutes. The complications of the fetal scalp were as follows: cellulitis, 9, all survived; abscess of the scalp, 2, all survived; hematoma, 2, absorbed; laceration, 8, all healed. (Kaltreider and McNally, Amer. Jour. of Obst. and Gyn., Nov. '43).—U.S.O.W.I.*

A working classification of the causes of abortion.—The traditional method of classifying the causes of spontaneous abortion divides them into two major groups, fetal and maternal, each of which is again divided into numerous subgroups, more or less ill-defined. As a practical basis for diagnosis and treatment, such an analysis tends to be confusing rather than helpful.

Every accomplished abortion includes three events: death of the embryo, separation of the ovum from its attachments and expulsive uterine contractions. Any one of these may be the primary event in a given case, followed sooner or later by the other two. Thus, abortion can be induced by killing the ovum with x-rays, by dislodging it with the curette, or by provoking effective uterine contractions with bougies. In the same way spontaneous abortions are initiated by accidents of three general types. A consideration of the causes from this point of view clarifies certain aspects of the problem of clinical management.

The major groupings of the classification proposed by the author are as follows:

- I. Death of the ovum a. Hereditary faults (1) Lethal and sublethal factors in germ plasma, (2) Low vitality due to infertility of parents. b. Environmental faults (1) Malnutrition from defective implantation, (2) Acute and chronic maternal diseases.
- II. Separation of the ovum a. Abnormalities of decidua (1) Insufficient endocrine stimulus, (2) Insufficient endometrial response. b. Abnormalities of trophoblast.
- III. Expulsive uterine contractions a. Factors exciting contraction (1) Mechanical hindrances to growth of uterus, (2) External violence. b. Excessive uterine irritability. (Meaker: Journal of the American Medical Association, November 1943).—U. S. O. W. I.*

Book Reviews

Surgery of Modern Warfare—Edited by HAMILTON BAILEY, F.R.C.S. with C. ALLAN BIRCH, M.D., M.R.C.P., D.C.H., D.P.H., M.M.S.A. as Sub-Editor for Medicine and compiled by seventy six contributors. Third edition, complete in six parts, 1944. Profusely illustrated, many being in colour.

Surgery of Modern Warfare has been written by a team which can certainly claim to represent British Surgery. In its pages are embodied the experiences of observers who have worked in the present conflict and it can safely be said to reflect the teachings from London, Scotland, Wales, Ireland and the services. The present edition which is the third, has been produced in six parts and its early appearance is an indication of its popularity and its importance.

PART I deals with classification of war-wounds, the manner of their infliction, their classification, bacteriology and treatment. Compression phenomenon, shock and its

treatment, transfusion and problems of resuscitation are also well discussed in this part.

PART II deals with burns and their treatment, anaesthesia, tetanus, gas gangrene, surgical materials and dressings.

PART III contains articles on surgery of the blood vessels, hemorrhage and its treatment, plaster technique and splints.

PART IV deals with wounds of bones and joints, wounds of the hand and foot and also those of tendons, nerves, spine and head and neck.

PART V deals with injuries of the brain and skull, ear, air-passages, eye and orbit and wounds of the throat and abdomen.

All the parts of the third edition maintain a very high standard. All the subjects are written by well known authorities in a very clear and practical manner. The illustrations, whether line, coloured drawings or photographs, are profuse and very

useful. The printing and general get-up are excellent and do great credit to the printers and publishers. The book is bound to be of great value as a guide to surgeons and a record of what has proved valuable in the present conflict.

Physiological Principles in Treatment by SIR WALTER LANGDON BROWN, M.A., M.D., D.Sc., F.R.C.P., L.D.S., and REGINALD HILTON M.A., M.D., F.R.C.P., 323 pages. 8th edition. 1943. (Baillière, Tindall and Cox).

This book was originally written to acquaint the busy practitioner with the leading changes in physiology and to deduce therefrom the points on which his clinical conceptions should be modified. Emphasis in this book is therefore necessarily laid on principles than on details. In this aim the authors have eminently succeeded. The present edition has been thoroughly revised. The sections on pituitary hormones, the treatment of gastric ulcer, nephritis, diabetes, hypertension and the carotid sinus reflexes have been considerably altered. An entirely new chapter has been added on the action of drugs. Principal references are given at the end of the book. The book is written in an easy and pleasant style. We are sure that it will serve a useful purpose.

Brompton Hospital Reports.—1943. Vol. xii. Published by the Research Department of the Hospital, 163 pages, Price 8 sh. net. Printed by Gale and Polden Ltd., England.

This is a collection of papers recently published from the Brompton Hospital. Most of these however have already appeared in the Medical Press. The collection however forms a valuable volume. The articles are well written, well illustrated with X-ray plates and diagrams and are very instructive. The article on childhood injection and its relation to adolescent and adult pulmonary tuberculosis is an interim report on the team research carried out in the hospital during the past fourteen years under the direction of the Medical Committee. Other interesting articles are those on "Traumatic Hemothorax," "Results of Thoracoplasty," "The Study of the Bronchi" "The health of the doctor." The medical reports of the hospital from 1939 to 1942 have been included.

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Ophthalmic Surgery and Sight-Testing.—By M. A. KAMATH, M.B. & C.M., Madras Medical Service (Retd.) 2nd Edition—1944. 297 pages with an appendix of 11 pages illustrated.

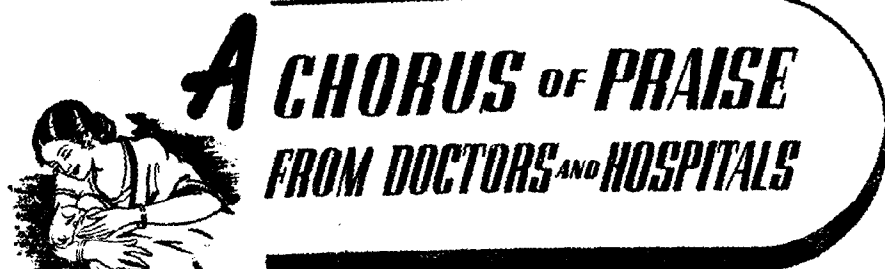
Dr. M. A. Kamath, who was for many years Ophthalmic Surgeon, Govt. Eye Hospital and Lecturer in Medical School, Tanjore, has published in a book form for the use of students and practitioners his lecture notes.

In this 2nd edition the author has followed practically the same classification as in the first, and has touched upon the methods of diagnosis and treatment of the commoner and more important affections of the eye. Pathology that is absolutely necessary for the proper understanding of the rationale of the signs and symptoms of disease and the principles of treatment are only included. The author has also wisely avoided the very intricate and as yet unsettled problems connected with the eye, such as the embryological anatomy, the nature of light, the physiology of the visual paths and perception, the pathogenesis of glaucoma, etc.

A special chapter has been added on Preventable Blindness—a matter which is receiving great attention in modern days and which figures prominently in post-war problems. The chapter on Refraction also is very well written. The Appendix contains common drugs that are used in ophthalmic practice and other very useful information. We are sure this book will be found very useful as a text-book of ophthalmology by the students and the general practitioners.

Electrocardiograms—An Elementary Atlas for Students and practitioners.—By H. WALLACE JONES, M.D., M.Sc., F.R.C.P., and E. Noble Chamberlain, M.D., M.Sc., F.R.C.P., 2nd edition, 56 pages with 53 illustrations, 1943.—(John Wright & Sons, Ltd., Bristol)

The above is the 2nd edition and is a very useful aid to students. The authors originally intended this little book to help medical students in appreciating this difficult subject. They have given a representative set of electrocardiograms which will be very useful to the students. The descriptive text has been reduced to a minimum. But beneath each record are fuller details of the electrocardiograms. The whole makes a very useful atlas. We recommend it to our readers.



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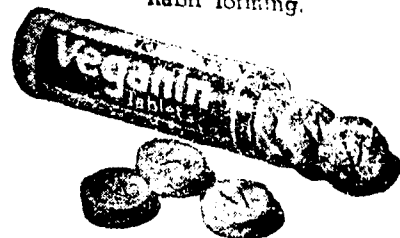


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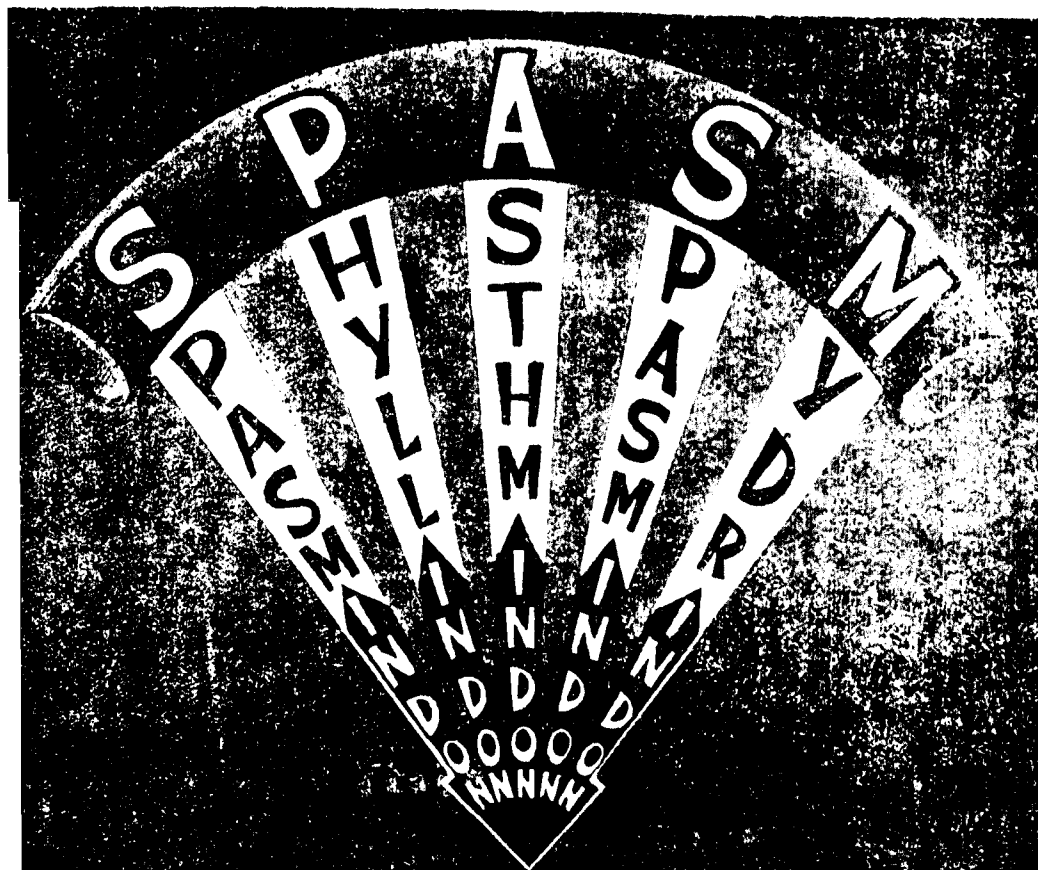
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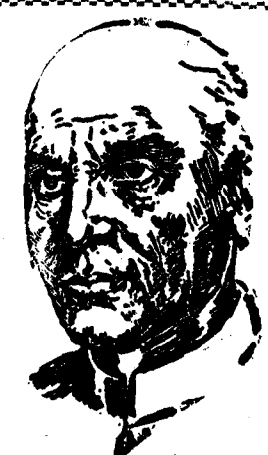
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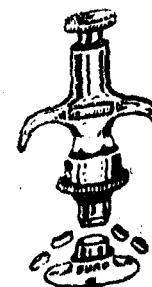
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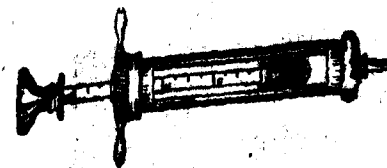
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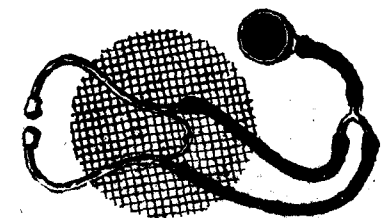
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