

The Antiseptic

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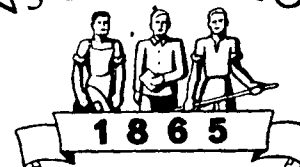
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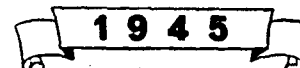


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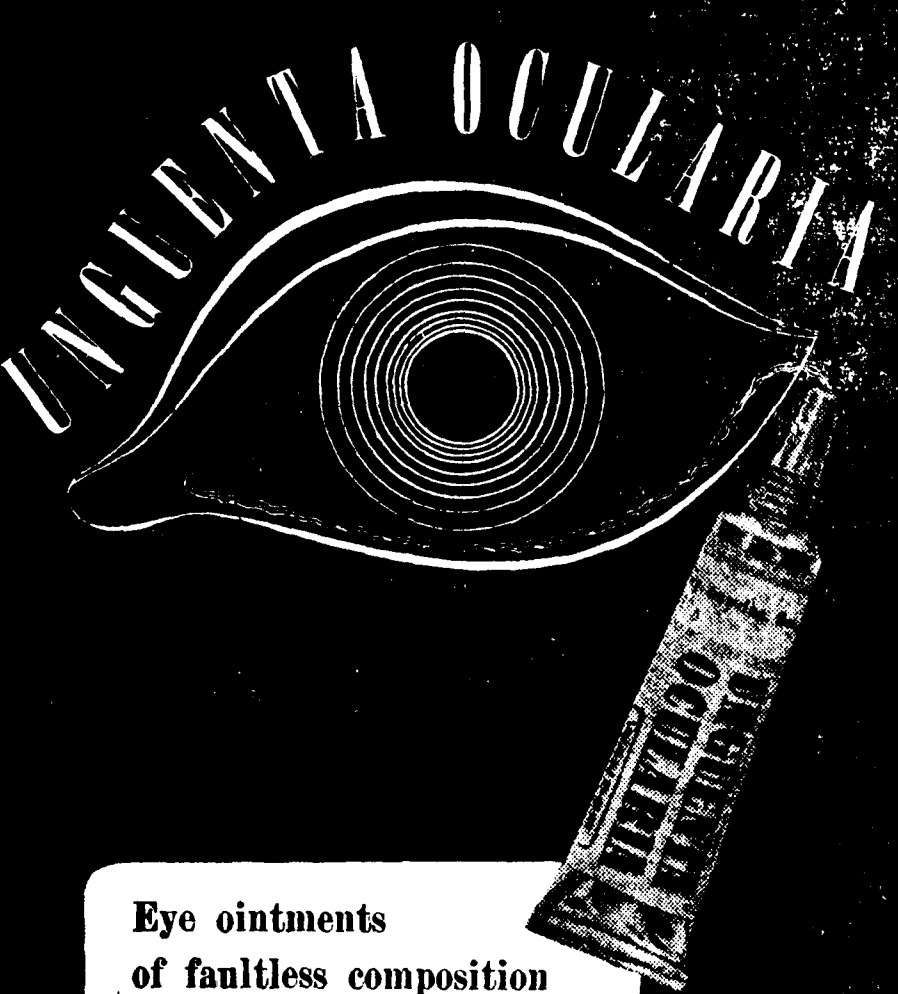
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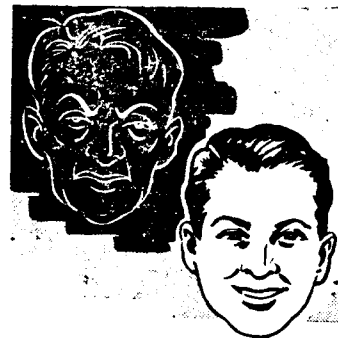
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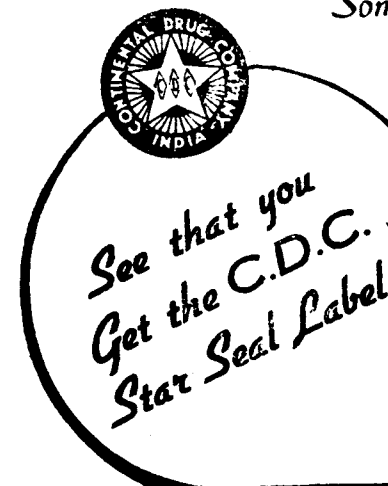
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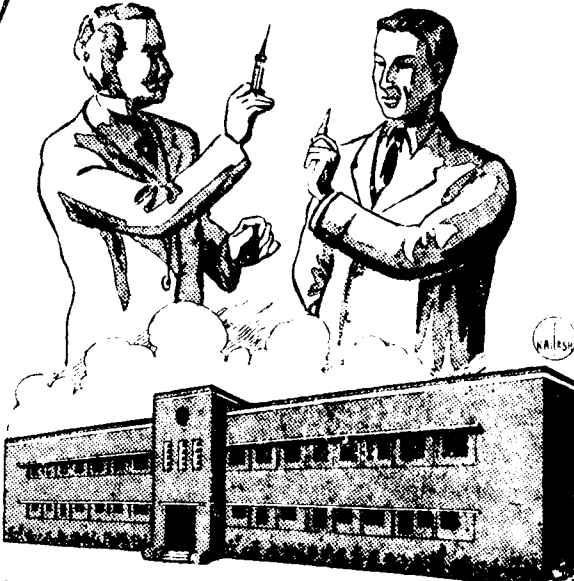
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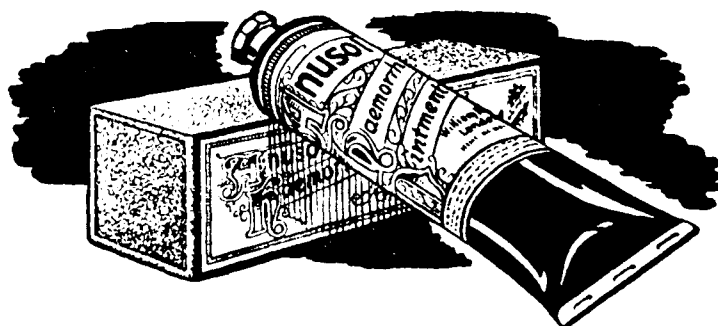
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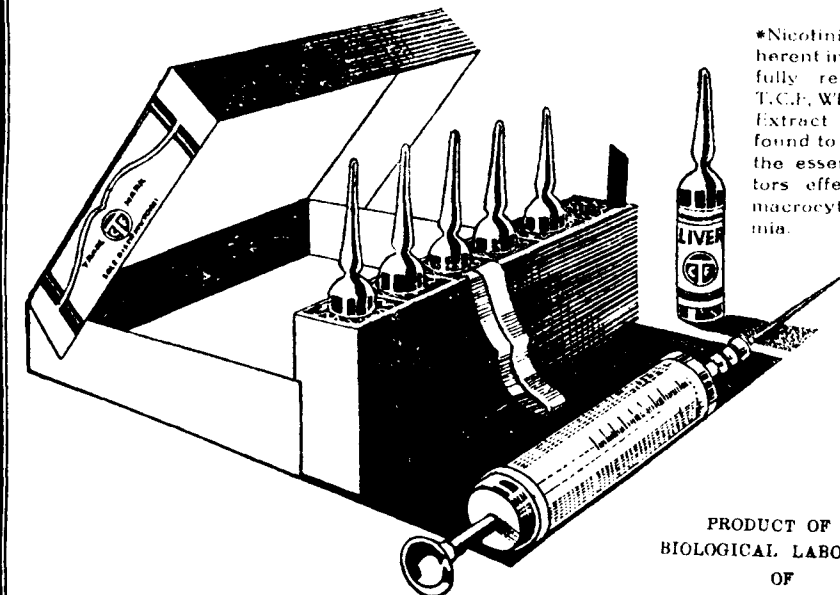
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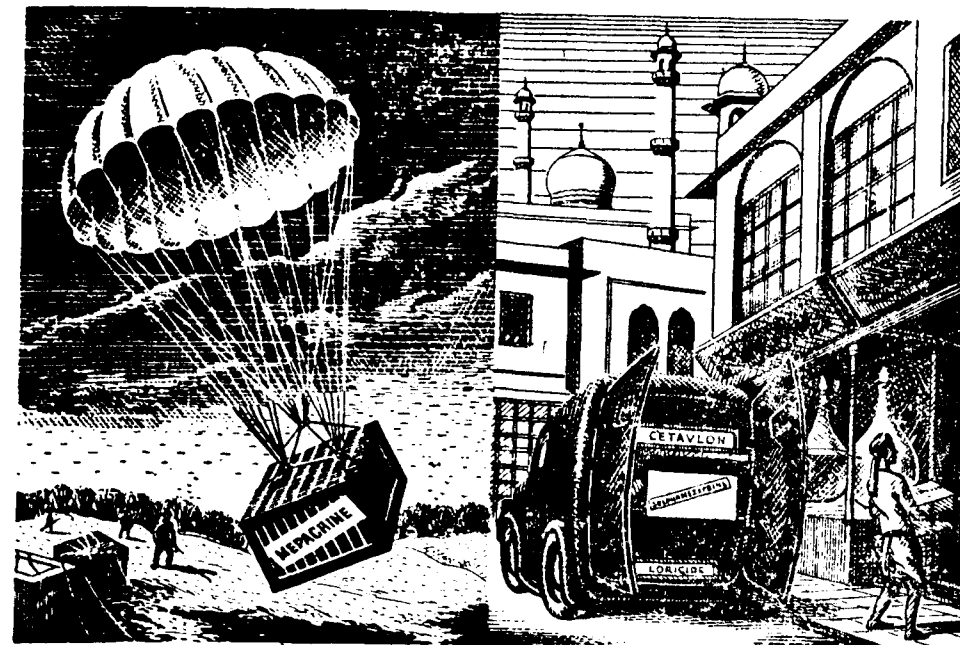
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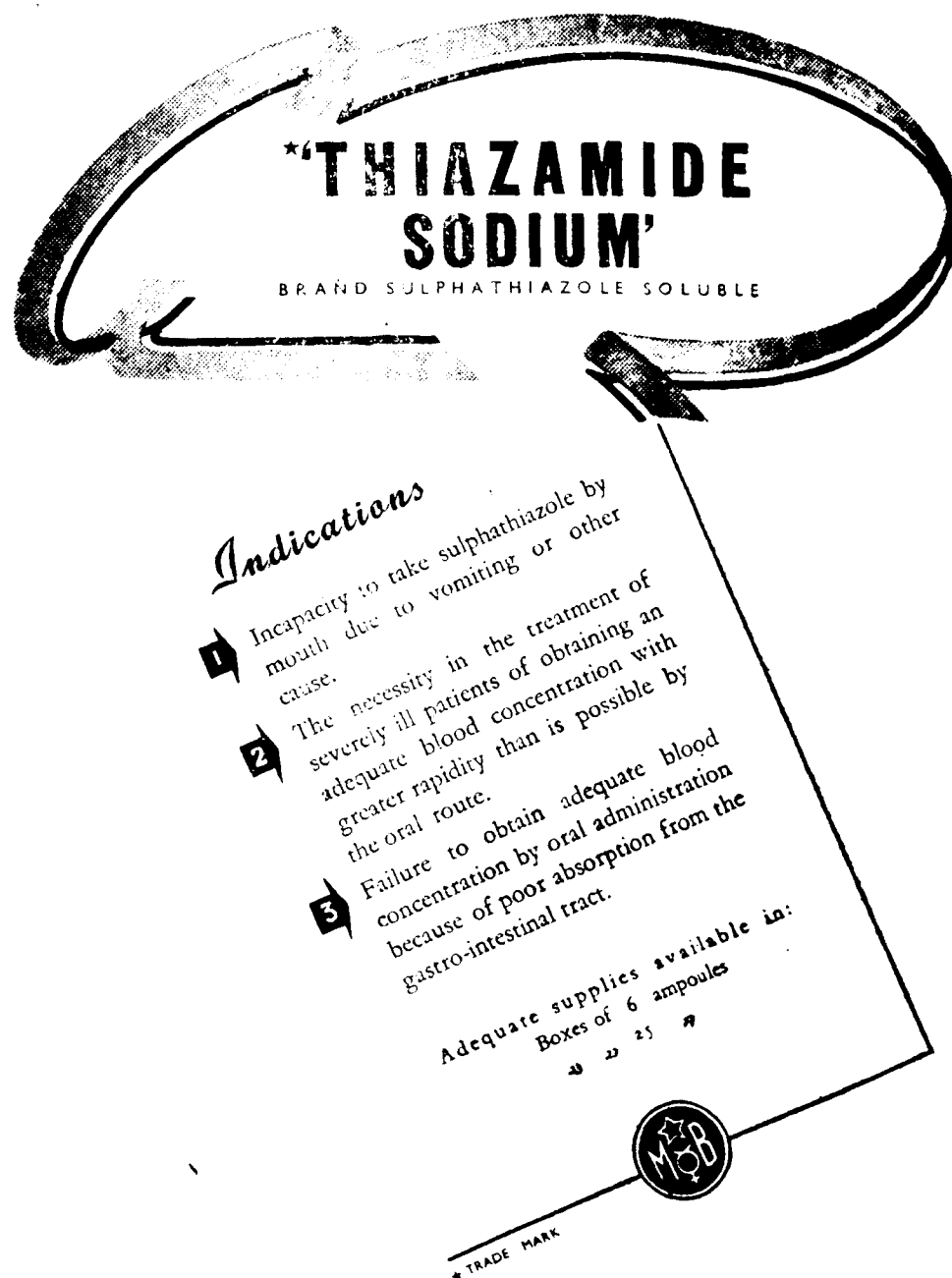
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JULY, 1946

No. 7.

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Discussion on the Cases of Adenomyosis and Endometriosis

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THE excuse that can be set forth in choosing the above subject is that it is not so rare an affection as to be seldom encountered in gynaecological practice, that it presents signs almost like chronic salpingo-oophoritis, so that it becomes nearly difficult to distinguish between the two, and that the symptoms exhibited, especially when the uterus itself is the site of this affection, are in most cases indistinguishable from those due to submucous fibroid of the uterus. So it is quite essential that while examining a patient with morbid affections of the uterus and its appendages, a gynaecologist ought always to remember this very important affection, endometriosis, and rule it out first before he can arrive at a correct diagnosis. For lack of this precaution a true case of endometriosis may be mistaken for chronic salpingo-oophoritis. Under this supposition an operation for freeing the uterus and its appendages may be undertaken. But the adhesions produced in both these conditions vary in intensity, those in endometriosis being very dense and deep. So in the event of the case being, or turning out to be, a case of endometriosis, the operator has to exercise great care, patience and judgment while breaking down the adhesions lest the structures, especially the intestines which are more often involved, should be injured. Should the damage done to the bowels escape notice during the operation, the grave complication of peritonitis is sure to follow which may cost the patient her life and the operator his reputation. In consideration of these conditions an attempt has been made to study the subject.

The term endometriosis itself implies the nature of the affection. It is the endometrium or the tissue almost resembling it in its appearance and its function which invades the foreign parts. First the surface is invaded and then the invasion spreads to the deeper parts. Thus the peritoneum is first

* Specially contributed to THE ANTISEPTIC.

involved and afterwards the parts underneath it. In this way the musculatures of the uterus is raised from the side of its serous covering or from the mucous membrane. The grafts are also seen to thrive in the tissues of the utero-sacral ligaments as well as of the round, utero-ovarian and broad ligaments. The implantations of the endometrial tissues are very often seen growing deep into the recto-vaginal septum. The other structures in pelvis that are implicated are the Fallopian tubes, the ovaries, the pelvic colon, the rectum, the bladder and the vagina. Endometriosis may be seen even outside the pelvis; it may be at the umbilicus, or an old abdominal scar following laparotomy; at the bottom of the sac of an inguinal or femoral hernia; on the wall of a coil of the ilium or the appendix or at the labium majus.

It would look idle on my part if I venture to go into the historical survey, aetiology and other things, when they are described in detail in every book on gynaecology and gynaecological pathology. Suffice it to say that there are two main theories: Implantation theory and serosal theory. They are referred to in short below.

Implantation theory.—In 1921 SAMPSON from America not only showed that in chocolate cysts of the ovaries and pelvic endometriosis is to be found proliferation of the aberrant endometrium but also offered the interpretation that during menstruation under certain conditions, such as retroflexion of the uterus or partial occlusion of the cervical canal from fibroid polypus, regurgitation of the menstrual fluid may occur through the Fallopian tubes into the peritoneal cavity, that the regurgitated fluid may contain minute portions of the shed endometrium, which are implanted on the wall of the ovaries or surface of the peritoneum, and that the grafts take root and continue to grow. This theory does not afford the right explanation for all varieties of adenomyosis in different situations; yet it has still some advocates.

Serosal theory.—This theory is advanced by ROBERT MEYER on the histological grounds and appears to account for all the types of endometriosis developed in the pelvis as well as outside. His points of view are during the development of the embryo the endometrium germinal layer of the ovary and the very epithelium lining of the peritoneum are derived from coelomic mesothelium or primitive peritoneum. Endometrium is the fully developed form, while the germinal layer of the ovary and even the lining of the pelvic peritoneum are the intermediate stages of development; but they possess the potentiality of undergoing metaplasia and of developing to further stages. This is quite evident from the fact that the flat epithelium of the pelvic peritoneum under inflammation or with ectopic gestation is observed to undergo metaplasia and acquire tall columnar shape. These facts lend support to the serosal theory, which explains adequately the cause of adenomyosis of the uterus, endometriosis of the ovary and of the pelvic peritoneum by metaplastic proliferation of the surface epithelium.

Instead of going into pathology I take up the discussion of my cases. I could in a short time at my disposal gather 40 cases, 14 from my own private hospital and 16 from Cama Hospital. For the latter cases I gratefully extend my thanks to Dr. J. JHIRAL, the Superintendent of the Hospital. Of these 40 cases, 6 were discarded for very vague history or inconclusive pathological report.

Age incidence—In 34 cases, the lowest age is 23 and highest 47.

TABLE I: Between 21 — 30	10 — 3 = 7
31 — 40	= 15 + 3 = 18
41 — 50	= 9 = 9

The maximum age incidence as given in books is between 30 and 40. In my table (1) there are 3 cases at the age of 30. If they are transferred to the second column, there would be 18 cases between the ages 30 and 40, while 7 between 21 and 29 and 9 between 41 and 50. Yet it may be remembered a fair number of cases do occur in the third decade also.

Among the above cases, two were single, not married.

Personal history.—TABLE II:

	CASES		CASES
(a) Bleeding per vaginum ...	11	(e) Painful and difficult micturition.	1
Irregular bleeding (9)		(f) Retention of urine ...	1
Continuous bleeding (2)		(g) Pain in passing motion ...	1
(b) Painful menstruation ...	7	(h) Mass felt in the lower abdomen.	2
(c) Pain in the lower abdomen.	10		
(d) Backache ...	2		

Patients came to the hospital for bleeding per vaginum in 11 cases. It is present in most cases of adenomyosis uteri; because in them the cavity is enlarged or the endometrium may be hyperplastic. Dysmenorrhœa is fairly common, present in 7 cases.

Pain in the lower abdomen was complained of in 10 cases. It is experienced in pelvic endometriosis, especially with chocolate cyst. Some of these cases had pains on either side in iliac regions due to the involvement in adhesions of the tubes and ovaries.

Backache was mentioned by 2 patients. It is due in many cases to spread of the affection along the utero-sacral ligaments.

Difficulty in passing motion was felt by one patient owing to the involvement of the rectum. Pelvic endometriosis affects the anterior wall of the rectum when endometriosis of the peritoneum at the posterior cul de sac grows deep in the recto-vaginal septum as well as extends to the rectum.

Trouble in passing urine may result from endometrial deposits in the utero-vesical pouch or increase in size of the tubo-ovarian masses in Douglas' pouch.

Patients feel a swelling in the abdomen when the pelvic mass increases in size and rises in the abdomen or when a fibromyoma is present along with adenomyoma of the uterus.

Previous operation.—TABLE III.

	CASES
Dilatation and curettage operation ...	10
In the case No. 22, D & C was done twice	
Removal of polypus ...	1
Ovaristomy for Pseudomucinous cyst ...	1
Oophorectomy with right salpingo-oophorectomy ...	1

The previous operation of dilatation and curetting betrays the fact that the affection was not recognized in the beginning. This operation shows conclusively that endometriosis, especially of the uterus, may occasion bleeding per vaginum, suggesting the need of that measure to control hæmorrhage. In one case (22), it was repeated twice at short interval.

Per vaginum—*Contd.*

Uterus felt enlarged in

15, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 31 — 12.

Uterus anterior but tethered behind in

12th case.

Uterus retroverted and fixed in

7, 8, 11, 33 — 4.

Adnexa affected in

9, 15, 18, 23, 24, 27, 28, 30, 34 — 9.

In adenomyosis uteri, the uterus is enlarged and becomes asymmetrical when infiltration and proliferation has increased and become diffused. The diffuse infiltration is in most cases found in the posterior wall of the uterus. A localized adenoma may be felt at one corner or both. This affection can be distinguished but it does not present as hardfeel as a fibromyoma. If pelvic endometriosis has advanced and endometrial cysts formed, the uterus might be retroverted and fixed. In case of chocolate cyst, a thickwalled cystic mass is palpable behind and on one side or both.

Associated affections.—TABLE VII:

Myoma	...	5	Hæmorrhagic corpus leu-	
Retroflexion	...	2	tian cyst	...
Hydrosalpina	...	2	Cystocele and rectocele	...
Ovarian cyst	...	1	Hypertrophied cervix	...
Twisted ovarian cyst	...	1	Metropathia hæmorrhagica	6
Fimbrial cyst	...	1		

Myomas of the uterus may be not infrequently seen along with patches of adenomyosis of the uterine wall when operation is performed mainly for fibromyoma and when macroscopic examination is done on removal of the organ. In the same way when a woman is suffering from a refractory and severe type of metropathia hæmorrhagica, when she is between the ages of 30 and 40 and vaginal hysterectomy has to be done, definite affection of the uterine musculature by adenomyosis in the form of deeply situated islands of endometrial glands with distinct hæmorrhages in them may be accidentally noticed. This fact may be remembered while carrying out pathological examination of the uterus removed for metropathia hæmorrhagica.

Diagnosis.—Doubtful or real diagnosis of endometriosis was made in 9 cases only; while it was done in some cases during operation when a small, thick-walled, ovarian cyst burst, letting thick chocolate fluid escape as the adhesions are being broken down. Dense, firm adhesions especially when with bowels may lend themselves to the suspicion of pelvic endometriosis. In several cases diagnosis is established only with microscopic examination.

A case of endometriosis has to be diagnosed from one of chronic salpingo-oophoritis; for in both the symptoms are almost identical. The physical signs too are nearly similar; in both the uterus may be retroflexed and fixed; the appendages are thickened, tender and adherent. But in infective inflammation there may be history of previous infection; whereas the pain felt in endometriosis during menstruation is very severe and may

even be told to be quite different from what was felt previously as dysmenorrhœa. If the uterosacral ligaments are felt thickened and tender and especially if one or more nodules are made out on one or both the ligaments by vaginal as well as rectal examination and if, at the same time, the rectum is felt thickened and tender owing to endometrial infiltration, presumptive diagnosis of endometriosis can be made with some degree of certainty. During operation the adhesions caused by gonococcal infection are in general thin and membranous.

Tuberculous affection of the uterine adnexa presents still more difficulty in diagnosis. But general condition of the patient may be very poor; there may be other focus elsewhere, such as the peritoneum or the lungs; menstruation may have stopped. But if endometrium is simultaneously invaded by tubercle, there may be menorrhagia. Leucorrhœa may be a prominent symptom.

Macroscopic and microscopic examination—Macroscopic appearance:—Cut surface shows an area of whools of striae with grey spaces about. There is no capsule formation. It has honey-comb appearance as there are small openings of glands, from which blood may be squeezed out.

Microscopic appearance—Aberrant endometrial tissue complete by itself, presenting proliferative or secretory phase according as the endometrium of the uterus is in one or the other cyclic change. Certain variations may be observed: the gland may be sparse or the stroma poor; or epithelium of the glands may be young and immature, not responding to the progesterone influence. In some even no endometrium may be traced. Yet in this, a broad zone of large endothelial leucocytes of pseudoxanthema cells is to be seen. The presence of such a zone indicates with certainty that the cyst is the result of endometrial invasion.

Treatment.—TABLE VIII:

	CASES
Subtotal hysterectomy (1), (2), (3), (26), (29)	...
Salpingo-oophorectomy (5), (9), (28)	...
Oophorectomy (4), (6), (7), (11), (12), (14), (15), (30) (8)	...
Subtotal hysterectomy with salpingo-oophorectomy (24), (27), (34)	...
Complete hysterectomy with salpingo-oophorectomy (10), (16)	2
Vaginal hysterectomy	...
Ventrosuspension with resection ovary	...
Radium therapy (Case No. 30).	...

In one case regression was observed after pregnancy.

No.	Name	Age	Married or single	Patient's history	Previous operation	Menstrual history	Obstetric history
1	S. G.	30	Married	Previous—Nil. Present—Tumour 8 years.	Nil	4-5 days 28	Abortion (1) 2 F.T.N.D.
2	G. L.	40	Married	Bleeding per vag. off and on for 3 years.	Submucous Polyp. removed in 1931, 1934 and 1937. Microsc. examined in 1937. Re—Fibro—Adenoma.	3-6 28 getting irregular when polypus removed.	Abortions 4
3	D. L.	41	Married	Pain in right iliac fossa, stabbing pain left side, five months.	10 months ago cystadenoma right tube and ovary removed. Part of the right ovary left behind. Operation for cystocele treated 10 years ago.	1-5 30 5-6 15 during 2 months.	2 F.T.N.D. No pregnancy 1 year.
4	P.	31	Married			Regular normal	Nil
5	A. S. R.	36	Married	Painful micturition. Difficulty in emptying the bladder completely, especially in period.	Operated 3 years before the suspected ectopic but turned out to be incarcerated gravid ut.	4 26-30 Scanty.	2 F.T.N.D. Last 9 years ago

Symptoms	Physical signs	Structures affected	Provisional diagnosis	Treatment and findings
1. Menorrhagia 8 years. 2. Dysmenorrhoea 8 years. 3. Sterility 8 years.	Per abd.—Irregular firm mass, 2 fingers' breadth below the umbilicus. Partially movable. P.V.—Ut. Ill-defined: adnex—nothing found.	Ut—Uniformly enlarged.	Adenomyosis.	Op.: Supravaginal hysterectomy. Findings—macroscopic—Diffused enlargement of the posterior wall, honey-comb appearance, small hemorrhagic points, apparently glandular openings filled with menstrual fluids. Microscopic—Adenomyosis uteri.
Menorrhagia off and on.	P.A.—Firm smooth mass, rising from the pelvis, more marked on the right side, smooth surface, well defined margins. P.V.—Polypus through os. size of a lemon friable. Path. report—adenomyoma.	Ut.—Irregular enlargement, Fibroids felt.	Adenomyoma.	Supravaginal hysterectomy, few adhesions with right ovary. Findings—Macroscopic—Ut—Uniform enlargement of the size of a cocoanut. Both cornua thickened. Tube—normal. Fibr: cyst right side, ovary—right cystic. On section—Enlargement at both cornua. Two small fibroids on the posterior surface. Small polypi in the cavity.
Menst.—Nothing important. Pain—iliac fossa.	P.A.—A mass felt. P.V.—Ut—Not enlarged, anterior and to the right of the mass. Mass—firm and elastic filling the pelvis, somewhat fixed.	Not determined.	Not determined.	Supravaginal hysterectomy. Uterus adherent cystic mass, which in turn was adherent to the surrounding structures. The cyst burst while freeing, chocolate fluid escaping. Partial removal of the cyst, as the wall was firmly adherent to the sigmoid and rectum. Findings—On section—macroscopic. Ut—cavity normal, wall thickened, two intramural fibroids. Ovarian endometriosis.
Nothing important.	P.V.—Ut—Anterior, normal in size. Firm irregular mass behind and to the right.	Ovarian tumour right side.		Right oophorectomy, cyst adherent, burst during operation, chocolate fluid escaping. Left ovary fibrosed. It was scarified. Microsc.—Ovarian wall thickened. Thick layer of large cells containing hemosiderin. Small areas here and there of the endometrial tissue. Diag.—Chocolate cyst.
(1) Dysmenorrhoea. (2) Backache. (3) Scanty menstr.	P.A.—A mass felt two fingers below umbilicus, soft, slight lateral mobility. P.V.—Cervix hypertrophied. Firm elastic mass rising in abdomen. Ut—Cannot be defined. Sp: vaginal—small bluish polypi behind cervix.			Right oophorectomy, left salpingo-oophorectomy and appendicectomy. Large bluish coloured ovarian cyst 4" x 3" adherent to the back of the ut. on the right side. Left ovary—small hemorrhagic cyst. Dense adhesions with the pelvic colon.

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No.	Name	Age	Married or single	Patient's history	Previous operation	Menstrual history	Obstetric history
3	Mrs. C.	39	Married	Pain in the lower abdomen 3 years.	Nil.	3-4 20 regular normal.	4 F.T. N.D. 1 abortion.
7	R. C. R.	30	Married	Pain in the lower extremity. 2 years. good condition.	...	2 20-24 Scanty	1 F.T. Forceps. Child died soon after 3½ years ago.
8	Mrs. A. K.	35	Married	Pain in the lower abdomen. Severe for 2 weeks. condition good.	...	3 28 regular.	1 abortion 4 years ago.
9	G. R. I.	28	Married 11 years.	Pain in the lower abdomen, severe attacks of pain, vomiting and fever after M.P. G. condition—good.	D and C done 3 years ago.	5 28 regular.	No conception.
10	M. V. G.	30	Married 2 years.	Retention of urine. 5 days. Gen. condition fatty.	Left ovariectomy, right salpingectomy.	4 28 regular	No conception.
	Mrs. H.	29	Married	Dysmenorrhoea, painful defaecation for 9 months. Thin.	Nil.	6 28 regular.	3 F.T. N.D.

Symptoms	Physical signs	Structures affected	Provisional diagnosis	Treatment and findings
1. Pain in the lower abdomen 3 years. 2. Anaemia.	P.A.—A mass is felt to the right of the midline. P.V.—Ut.—Cannot be identified.	1. Right ovary. 2. Adhesions to surrounding structures.	Not settled.	Bilateral oophorectomy. Endometrioma of the right ovary.
1. Pain in the lower extremity during menstrual periods 2 years. 2. Oligomenorrhoea. 3. Dysmenorrhoea.	P.A.—Nil. P.V.—Ut.—Retroversed fixed. Tenderness in Douglas' pouch.		Endometriosis.	Bilateral oophorectomy, adhesions, both ovaries. Bilateral chocolate cysts.
1. Pain in the lower abdomen 2 years. Severe for the last 2 weeks. 2. Vomiting. 3. Difficult micturition.	P.A.—Tenderness and fulness in the right iliac fossa. P.V.—Ut.—Retroversed, tender, bulky, fixed. Small nodule on the left side. Fornices—nil.	Ut.—Adherent behind; fibromyoma. Pedunculated post wall adherent-rectum. Ovaries—Both affected—chocolate cysts adherent.	Endometriosis. Subperitoneal fibroid.	Resection both ovaries—Fibroid removed. Both ovaries—Chocolate cysts. Tubes normal.
1. Pain in the lower abdomen, more on the right side, 3 years. 2. Severe attacks of pain, vomiting and fever after M.P. 3. Dysmenorrhoea 6 years, during and after.	P.A.—Tender on the right side on deep pressure. P.V.—Ut.—Anterior a large cystic mass, adherent on the right side. Thickening—left side.	1. Right ovary large cystic. 2. Left tube thickened and adherent.	...	Right Salpingo-oophorectomy. Left-Salpingectomy and salpingostomy. Ovary resected. Finding—Right ovary—chocolate cyst—adhesions dense. Left tube—Bulbous chocolate cyst right ovary.
...	P.A.—Distended bladder felt. P.V.—Ut.—Anterior. Somewhat pushed down by the mass. Large elastic mass filling up D.P. Small polypus in the cervix.	(Bladder high and hypertrophied.) Right ovary cystic. Subperitoneal fibroid at the fundus.	Not made.	Amputation cervix. Total hysterectomy. Right ovariectomy, ovary firmly adherent stripping off peritoneum from the rectum. While breaking adhesions it bursts, allowing chocolate fluid to escape.
1. Dysmenorrhoea Pain a few hours before and during menstruation. 2. Backache. 3. Pain during defaecation—9 months.	P.V.—Ut.—Retroversed, tender, matting together, felt posteriorly.	Uterus retroflexed and fixed.	...	Left oophorectomy. Chocolate cyst left ovary. Uterus adherent to the rectal wall. Pelvic endometriosis, chocolate cyst left ovary.

No.	Name	Age	Married or single	Patients history	Previous operation	Menstrual history	Obstetric history
12	Mrs. S.	35	Married	Dysmenorrhœa very marked 15 years. Gen. condition good.	D and C 14 years ago. Left ovarian cyst removed 10 years ago.	4 First regular and normal Last 15 years. 10-12 In clots. Pronounced pain. Starting on 3rd day and lasting up to 6th day.	2 F.T. N.D.
13	M. N.	28	Married 15 years.	Dysmenorrhœa. First for 9 years, 2 hours only. For the last 4 months 3 days. Gen. condition good.		7-8 regular 28 for last 9 years. Pain increased D & C 9 years ago.	No conception.
14	F. G.	25	Married 10 years.	Dysmenorrhœa and Dyspareunia 10 years. Gen. Condition good.	Nil.	3 Pain 3 days 28	No conception.
15	K. M.	23	Single.	Severe dysmenorrhœa pre and intra-menstrual.	Nil.	Began at 12 First 3 years. 8-10 excessive but no pain. Now 8 years—8-10 excessive with severe pain and vomiting. Indefinite history inter-menstrual pain.	(Not married).
16	S. I. S.	47	Married	Menorrhagia 8 years. Gen. condition good.	D. C. Early March 1944 (means a month ago). Bleeding started again after 3 weeks.		9 F.T. N.D. last 9 years ago.

Symptoms	Physical signs	Structures affected	Provisional diagnosis	Treatment and findings
1. Dysmenorrhœa very severe 15 years. 2. Painful defecation during M.P.	P.V.—Ut—Anterior but tethered behind.	Uterus ovary rectum rectovaginal septum.	Adenomyosis, uteri. Endometriosis vaginal septum and rectum.	Right oophorectomy. Uterus—infiltration and adhesion behind to the rectum.
1. Dysmenorrhœa. First—Slight. 2 hours only. Last 9 years marked. 2. Vomiting.	P.A.—Firm resistance 3 fingers above the symphysis rounded outline. No fluid. P.V.—Uterus—Hard, bulky, irregular. Semi-elastic mass to the left and behind. Right fornices—Nil.	Uterus—Suspected to be fibroid. Left ovary.	? Fibroid.	Left oophorectomy. Chocolate cyst-left ovary, cyst adherent at the left cornua. Small fimbrial cyst right side.
Dysmenorrhœa and Dyspareunia 10 years.	P.V.—Uterus—Anterior, bulky, adherent, irregular behind. P.S.—Purplish nodules behind the cervix.		Endometrioma.	Resection of the left ovary. Chocolate cyst left ovary—dense adhesion at the left cornua. Partially separated. Report—? Follicular retention cyst. Hamorrhage corpus leuteum.
1. Menorrhagia from the start. 2. Dysmenorrhœa. severe beginning 3 years after.	P.A.—Nil. P.V.—Difficult cervix far back Uterus—Anterior, enlarged, irregular, tender.	Uterus—Ovaries both sides.	?	Bilateral oophorectomy. Both ovaries chocolate cyst. Dense adhesion burst during operation allowing chocolate fluid to escape. Micros.—Wall lined with columnar epithelium cystic spaces filled with blood. Blood vessels in fibrous tissue.
1. Menorrhagia 8 years. 2. Constant bleeding 2 months.	P.A.—Mass 3½ months pregnancy. Uterus—Cannot be identified separately. Tumour 3½ months' pregnancy.		? Fibroid. Adenomyosis uteri.	Total hysterectomy with bilateral salpingo-oophorectomy. First subtotal hysterectomy was decided upon. During removal, something gave way getting chocolate fluid escape. On opening the uterus, a large, unhealthy, friable growth was detected which was thought to be malignant. The uterus walls were gone thick. So pan hysterectomy was done. Microscopic report—Adenomyosis uteri-glandular structure seen going up to peritoneum.

No.	Name	Age	Married or Single	Patient's History	Previous operation	Menstrual History	Obstetric History	Symptoms	Physical signs	Structures affected	Provisional diagnosis	Treatment and Findings
17	V. G. C. G.	45	Married	Irregular bleeding 2 years.	2 D and C done 2 years ago.	Past 5-6 regular 30 Present 10-15 15-20-25 Irregular and profuse.	Abortion 1. 4 years ago. 10 F.T. N.D. Lining. Last 6 years ago.	1. Dysmenorrhœa. 2. Menorrhagia, polymenorrhœa, metrorrhagia for 2 years. 3. Pain in lower abdomen, Backache.	P.A.—Nil. P.V.—Uterus uniformly enlarged.	Uterus.	Metro-pathic hamorrhage. Microscopic metropathica adenomyosis uterus.	Vaginal hysterectomy. Microscopic—Section thick, endometrium without epithelium. Glands proliferative stage. Stroma loose, oedematous no evidence of inflammation. Muscular wall, thick. Island of endometrial glands with distinct stroma. Diagnosis — Metrop: hamorrhagica adenomyosis uterus.
18	Mrs. M. M.	37	Married	Irregular bleeding 6 months. Bleeding continues from last period.	D and C 5 months ago.	3-4 regular 30 and normal.	2 F.T. N.D., abortions 4. Sepsis after the last, about 3 years ago.	1. Pain, lower abdomen, backache. 2. Menorrhagia and metrorrhagia six months. 3. Constipation. 4. B.P. 130/100.	P.A. Nil. P.V. uterus enlarged, adnexa, right chr. infl. parasetitis base.	1. Uterus. 2. Right tube and ovary. 3. Thickening along the base of the right broad lig.	Chr. infective endometritis and chronic salpingo oophoritis.	Vaginal hysterectomy. Microscopic examination, uterus, endometrium, thick, no epithelium. Stroma heavily infiltrated with plasma cells. Wall, thick. Islands of glands in the muscular wall.
19	Mrs. J. J. Z.	44	"	Back pain Irregular bleeding.	D and C 17 years ago.	4-5 First. 30 15 Some 30 years. Bleeding off and on one year. Continuous bleeding 25 days.	6 F.T. N.D., during last 15 years ago, abortion 1.	1. Pain, back 1 year. 2. Metrorrhagia 1 year. 3. Weakness, loss of appetite, headache, insomnia, giddiness.	P. V. uterus enlarged.	Uterus.	Metro-pathic hamorrhage.	Vaginal hysterectomy. Report, endometrium early proliferative. Islands of glands in the musculature.
20	Mrs. P. V. J.	36	"	Pain in the lower abdomen and back. Prolonged menstrual loss.	D and C 10 years ago.	4-5 regular 30 Present 18 Scanty 30 and painful.	2 F.T. N.D., last 3 years ago.	1. Pain in the lower abdomen. 2. Backache 4 years. 3. Dysmenorrhœa 3 years. 4. Occasional headache.	P.V. uterus enlarged hard feel at two places.	Uterus fibromyoma.	Fibromyoma of the uterus, adenomyosis.	Vaginal hysterectomy. Microscopic: Endometrium thin, deficient epithelium glands, lined by single layer of epithelial cells. Some of the cells have nuclei in the centre with pale areas at the base. Islands of endometrial glands in the muscle wall.
21	Mrs. S. N. R.	44	"	Irregular bleeding 5 years.	"	5 regular 28-30 lar. Present 3-4 bleed. 20-25 ing off and on.	9 F.T. N.D., last 10 years ago.	1. Pain in hypogastrium. 2. Backache 5 years. 3. Dysmenorrhœa 5 years. 4. Metrorrhagia 5 years. 5. Weakness. 6. Loss of appetite. 7. Headache, giddiness.	Uterus large.	Uterus contains adenomatous endometrial polyp.	Metro-pathic hamorrhage.	Vaginal hysterectomy. Microscopic report, polyp, endometrial adenomatous.
22	Mrs. K. M. B.	38	"	Irregular bleeding 3 years.	D and C twice. First 2 years ago. Second 1½ years ago.	5-7 regular 30 Amenorrhœa 9 months followed by 20-15 30-60	6 F.T. N.D., last 5 years ago, abortion 1 (3 months).	1. Pain, travelling down the legs. 2. Metrorrhagia 3 years. 3. Loss of appetite. 4. Constipation. 5. Headache. 6. Giddiness.	Uterus uniformly enlarged.	Uterus.	Metro-pathic.	Vaginal hysterectomy. Microscopic report. Islands of endometrium in the uterine musculature. Endomet. glandular hyperplasia (advanced secretory phase).

No.	Name	Age	Married or single	Patient's history	Previous operation	Menstrual History	Obstetric History
23	Mrs. D. H. S. D.	46	Married	Continuous bleeding one month.	Nil.	2-3 regular 30 Last 1 year. 15-20-25 1-2-3 months.	3 F.T. N.D., 2 abortions last 21 years ago.
24	Mrs. M. F. B. F.	37	"	Continuous bleeding 14 days.	Nil.	8-10 F.M.P. 30 7 P.M.P. 30	4 F.T. N.D., last child 10 years ago.
25	Mrs. W. R. ...	47	"	Irregular bleeding.	Nil.	First 5-6 30 P. M. H. 5-6 regular 28 Pre. M. H. 6-7 1 year irregular. 3 scanty. 30	5 F.T. N.D., last child 6 years old.
26	Mrs. R.G.N.J.	33	"	Dysmenorrhœa.	Nil.	3 scanty. 30	4 F.T. N.D., last child 8 years old.
27	Mrs. M.M.S.S.	42	"	Pain in the lower abdomen 4 months. Tumour in the abdomen 1½ years.	Nil.	P. M. H. 4 regular. 30 Pre. M. H. 6 profuse 30 and in clots 4 years.	1 F.T. N.D., 25 years ago.
28	Mrs. C.K.C.P.	35	"	Pain in the lower abdomen.	Nil.	P. M. H. 6 regular. 30 Pre. M. H. 5-6 irregu. 20-25 lar, scanty, 9 years.	1 F.T. N.D., 10 years ago.

Symptoms	Physical signs	Structures affected	Provisional diagnosis	Treatment and Findings
1. Pain in the lower abdomen. 2. Backache. 3. Menorrhagia } 1 year. 4. Metrorrhagia } 5. Continuous bleeding 1 month.	Uterus enlarged, right ovary punctured. Blood-stained fluid came out.	Uterus right ovary.	...	Vaginal hysterectomy. Microscopic report. Adenomyosis uteri. Glandular hyperplasia of endometrium. Muscle wall contains islands of endometrial glands.
1. Dysmenorrhœa. 2. Continuous bleeding 14 days.	Uterus enlarged. Appendages left affected, thickened.	Uterus left ovary!	...	Subtotal hysterectomy with left salpingo-oophorectomy. Macroscopic—post wall shows marked thickening and adenomyosis. Definite endometrial glands and stroma.
1. Backache. 2. Menorrhagia 1 year. 3. Giddiness. 4. Insomnia of late.	Uterus enlarged, elongated.	Uterus.	?	Vaginal hysterectomy. Microscopic report: Typical endometrial gland with stroma forming islands in the musculature.
1. Backache 8 years. 2. Dysmenorrhœa. (severe) 8 years. 3. Sterility. 4. Constipation. 5. Weakness. 6. Loss of appetite.	Uterus enlarged.	Uterus.	...	Subtotal hysterectomy. Pathological report—Adenomyosis uteri.
1. Pain lower abdomen 4 months. 2. Backache. 3. Menorrhagia 4 years. 4. Weakness. 5. Loss of appetite. 6. Headache. 7. Sleeplessness.	Tumour in the abdomen. P. V. A tumour felt through posterior fornix.	Uterus tubes ovaries.	Fibromyosis. Endometrial polypus. Endometrium of the uterus.	Subtotal hysterectomy with bilateral salpingo-oophorectomy. Findings: uterus irregularly enlarged, retroverted, diffuse infiltration and thickening of the posterior wall. Right corneal enlargement. Right appendages adherent to the back of the uterus and broad ligament. Right tube distended. Left appendages adherent tube forming hydrosalpingotomy.
1. Pain in hypogastrium. 2. Dysmenorrhœa. 3. Sterility. One child 10 years. 4. Dyspareunia. 5. Constipation. 6. Weakness. 7. Loss of appetite. 8. Lack of sleep.	P. A. Some fullness in the lower abdomen. P. V. Firm elastic masses felt through post fornix and either fornices.	Tubes and ovaries.	Bilateral Hydrosalpin.	Bilateral sanpingo-oophorectomy adhesions. Ovarian wall contains patches of endometrium with hæmorrhages in the endometrial glands.

Hysteria*

Its Various Clinical Aspects and Treatment with A Case-Note

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HYSTERIA is a syndrome of psychical disturbance comprising of a peculiar symptom-complex in which there are physical signs of perversion of normal functions but without signs of organic lesions, and it is characterised by nervous crisis of an emotional, convulsive or other nature and by altered reactionary disposition (both motor and sensory) without failure of intellect.

Queer series of complex efforts of the patient not only make the relatives of the diseased extremely anxious but also often put the medical attendants into dilemma with great public discredit to the profession. Though this syndrome presents a clear clinical picture yet its varied and protean types of symptoms which are manifested by individual patient often lead practitioners astray.

Case note.—S.K.D., a Bengali Hindu young man, aged about 30, native of the local village, not much educated, a business man with the practice of daily hard labour. Date of examination 20-4-'46.

Family history:—His father was alcoholic. His mother is of melancholic nature. His wife is rather selfish. No history of hereditary disease.

Personal history:—Lives on simple Bengali diet. Occasionally drinks country-made intoxicants.

Previous illness:—Nothing remarkable except small-pox some 10 years ago. No history of venereal diseases.

History of present illness.—The patient complained of malaise and feverishness for the last two days, and on 20-4-'46 at about 9-30 a.m. all on a sudden he was taken ill and complained of severe pain and tenderness all over the body, specially on the loins and back. Probable immediate cause was not obvious but, later, on investigation it was revealed that he was not on good terms with his family members, notably with his wife; on economical problems. Still later, further investigations revealed that he has had an idea that for some reasons the deity, Mahadev of Joyrampore, became angry on him. The course of the malady presented a marked rapid progress. As his relative attendants grew more anxious, the worse became the condition. Spasmodic contractions of left limbs, associated with perspiration all over the body, slightly raised temperature, slightly hurried respiration, fine tremors of the fingers, reddish eyes with flushed face, agonising groaning, stiffness of the left arm and intermittent unconsciousness presented a picture which became worst when suddenly the patient lost the power of speech. This is the first attack of this kind in his life.

Physical examination:—Height, weight, development, muscularity, quite normal. Flushed face, agonising expression. Flat but contracted and stiff decubitus. Temperature 98.8°F. Pulse 94 p.m. Resp. 22 p.m.

Nervous system:—Symptoms—Constant pain all over the body, exaggerated pain on movement, chilliness, formications, tingling, faintness and vertigo present. Catalepsy of the left hand. No aura.

* Specially contributed to THE ANTISEPTIC

Cerebral and mental functions:—Intelligence not much impaired, understood all the questions asked and answered by play of movements. Inattentive to attendants. Aphonia and mutism but on coughing and groaning sounds were clear. No other delusion and delirium except to seek help and pity from the deity, Mahadev of Joyrampore (it was understood by guessing on his movements).

Objective signs:—Tenderness all over the body except on the left leg which was partly anæsthetic (stocking type anæsthesia) hyperæsthesia of loins. Sense of position normal.

Motor functions:—"Clavus hystericus" present. Fine tremors. Contractions and stiffness (these were not self-injurious). "Astesia abasia."

Special senses:—Eyes—pupil moderately dilated. Field of vision probably diminished. Conjunctival sensitivity lessened. Hearing not impaired. Responded and understood all the questions asked and answered by suggestive movements. Smell and taste impaired.

Reflexes:—Tendon reflexes rather increased. Plantar reflex flexor. Babinski—absent. True ankle-clonus absent but presence of pseudo-ankle-clonus was suspected. Pupillary reaction normal. Sphincter-control normal. No trophic change.

Locomotor system:—Polyarticular joint-pain and tenderness very marked, no remarkable physical abnormality.

Circulatory system:—No gross physical abnormality of the heart. Face is flushed. Slight engorgement of the conjunctiva. Pulse 94 p.m.

Respiratory system:—Spasmodic cough without expectoration but no obvious abnormal signs except slightly hurried respiration (22 p.m.).

Alimentary system:—Neither marked symptoms nor gross physical abnormality. Lips dry. Tongue dry and white coated.

Urine:—Amount rather profuse and clear.

Genital system:—Normal.

Glandular system:—Normal.

Cutaneous system:—Normal.

No laboratory examination was done.

Interesting and salient points:—(1) Intelligence quite clear; (2) pain with cramps; (3) aphonia but on coughing sounds clear; (4) catalepsy of the left hand; (5) "astesia abasia;" (6) stocking type anæsthesia; (7) restricted field of vision; (8) no definite signs of organic lesion; and (9) sudden onset as well as sudden cure (which was followed later after treatment).

Treatment which was adopted.—Though it was quite abrupt as the disease was, yet it was quite fruitful. As soon as the syndrome was suspected to be a case of hysteria a course of psychological and environmental treatment was adopted in the shape of infusion of strength and courage into the patient's mind to convince him that no gross organic lesion was present but it was simply an attack of rheumatism (!) which would be cured in no time just after taking one dose of mixture. Mr. enthusiastic and anxious relatives were removed from the patient's surrounding. A light massage and dry heat application for about 20 mts. the left hand removed the anomalies of stiffness and catalepsy. As the patient insisted by his movements (as was guessed) that the dei

(Mahadev of Joyrampore) wrath made him extremely ill, it was told to him that his idea was quite right, and to seek pity of the God some of his relatives would just start to win the God's favour and in the meantime one draught of medicine which would be made with the god's *Charanamrita* and be prescribed by me (a Brahmin) by the name of the God would cure him practically instantly. However, it took almost three quarters of an hour to make the patient amenable to psychological treatment. Besides these types of treatment, medical treatment was adopted thus:—

(1) One injection of Calcibronat 5 c.c.

(2) A mixture—

℞ Sodi Bi-Carb	...	gr. 10
Sodi Salicylas	...	gr. 10
Pot. Brom.	...	gr. 5
Ammon. Brom.	...	gr. 5
Liq. Ammon. Acet. Dil.	...	℥ 2
Tr. Valerian Ammon	...	℥ 10
Tr. Asafoetida	...	℥ 10
Syrup	...	℥ 2
Aqua menth. pip ad	...	℥ 1

Mft. Mist. 4 every 4 hourly.

Moreover, I instructed the nurse accordingly.

However, the result was rather strange. After half an hour of the first dose of medication the patient began to speak and gradually within an hour the patient became practically symptomless. After the second dose he became quite cured except that a slight degree of headache persisted. However, the patient was on this line of treatment for two days and fortunately he was not re-attacked within this period. On the 3rd day morning he went to Joyrampore as a pitiable devotee of the God Mahadev. I was not further informed about the patient up till now.

To have a more descriptive picture of this symptom-complex some of the interesting but important points of the syndrome should be noted as (1) hysterical manifestations never occur during sleep or when an audience is believed to be impossible; (2) symptoms are misleading, bizarre and contradictory from the point of organic disease; (3) no physical signs can be ascertained in relation to the symptoms; (4) sudden onset and sudden cure is the usual course; (5) psychological abnormality can be found in most hysterics, and it is the psychological phenomenon which plays the leading part in the manifestation of the symptoms; (6) organic disease may co-exist and may induce onset of hysterical manifestation; (7) hysterics are quite unaware how the symptoms arise; (8) intelligence is never seriously, if at all, impaired in true hysteria; (9) the patients are generally extremely egocentric and selfish; and (10) there may be an outward show of great emotion but probably there is little real emotional content.

Etiology.—There are various theories about the origin, causation and occurrence of hysteria. Some of these are noted here: (1) patient shows symptoms for the sake of some advantage without full consciousness of this motive (AUBREY LEWIS); CHARCOT considers this to be due to some perverted fixed ideas; (3) it is a manifestation for dependence on others, and the hysterics solve their problems by production of some symptoms with unconscious desire that their difficulties will be removed. This temporary solution brings them a characteristic composure and indifference

("la belle indifference" of Janet) and mental relief: (4) BABINSKI regards the symptoms as a phenomenon of an unstable mind overpowered by suggestions; and (5) FREUD is of opinion that it is a mental traumata of sexual nature, occurring before puberty. Such traumata may be suppressed into the subconscious mind but may resume activity at a later stage and cause the symptoms of hysteria.

Age:—Commonest between 15 and 30 years. Rare in younger subjects. When established may persist throughout life particularly in women.

Sex:—Females disproportionately predominate. Rare in adult males. It is more common in adolescence and at menopause.

Heredity:—Mostly occurs in families with a history of schizophrenic, alcoholic or other neuropsychopathic disorders.

Race:—Latin, Jewish and Slav races are specially susceptible. It is practically confined to civilised races, very rare among uncivilised. In India, perhaps it is mostly found in Bengalees.

Personality:—It occurs generally in a hysterical type of personality, characterised by egotism and an attitude of posing or make-believe. Such patients show a marked emotional susceptibility and are readily influenced by and imitate those who appeal to them (SAVILL).

Exciting causes:—(1) Shocks, specially physical, e.g., love-affairs, fright, disappointment etc.; (2) Strain, worry or anxiety for a long time; (3) Long continued pain or sufferings specially when the patient thinks that the relatives are asympathetic; (4) Sexual excesses—the disease is not uncommon among prostitutes; and (5) depressed general health.

Prognosis.—The prognosis depends mainly on modification of the personality with the readjustment of the etiological factors and the mode of treatment adopted. Though the individual symptoms are fairly easy to ameliorate, the liability to recurrence is great and may persist for many years, usually diminishing after the age of 40. In monosymptomatic cases and in young prognosis is more favourable. In a given symptom, e.g., paralysis, duration cannot be foretold: after existing many years may disappear suddenly, often following a shock. No symptom is necessarily permanent. Mortality rate can be ignored except in cases of anorexia nervosa, and rarely, persisting vomiting, where there is definite mortality.

Diagnosis.—There are mainly three difficulties to come to definite diagnosis, as: (1) Exclusion of organic disease of nervous system; (2) presence of both hysteria and organic disease; and (3) distinction of hysteria from pure malingering.

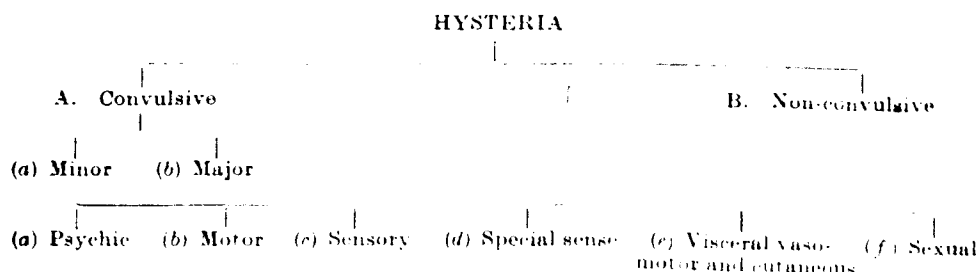
Previous hysterical symptoms, family history, personal history and complete physical examination are of immense help to come to the point. And the special points which are in favour of hysteria are: (1) Abrupt appearance and disappearance of symptoms after trifling causes; (2) a series of symptoms not explicable by an organic cause; (3) absence of symptoms to be expected in organic disease; and (4) general anaesthesia removes some of the symptoms as contracture, phantom-tumour, etc.

Some of the critical symptoms are also helpful, as: (1) Psychical symptoms; (2) anaesthesia; (3) constricted field of vision; (4) clonus hystericus; and (5) exaggerated symptoms with negative physical signs.

Finally a brief description of general symptoms is essentially noteworthy.

Symptomatology.—Symptoms are varied and of every degree and these may simulate any organic disease. Often many varieties co-exist or follow in the same individual.

Classification of Symptoms



A. I. Minor convulsive attacks.—Some sort of psychopathic emotional disturbance is followed by irregular clonic movements with gradual or sudden loss of apparent consciousness.

Movements are injudicious but not self-injurious: the patient does not bite tongue or pass water. Gradual recovery may follow passing of flatus or pale urine. The patient may hazily recollect the occurrence. Torpor or catalepsy may persist or recur.

II. Major convulsive attacks.—(Syn. hystero-epilepsy). Generally after an aura tonic stage begins (even opisthotonos) which is followed by leaping or struggling with purposive movements, accompanied with screaming, singing, talking or clonic spasms and consciousness. Epileptoid convulsions followed by various contortions thus signifies this rare condition to be neither epilepsy nor hysteria (MAJUMDAR). It is most prevalent in Latin races.

B. I. Psychic symptoms.—Increased suggestibility and "fixed ideas" are the two characteristic symptoms which are to be discovered by careful investigation. Alternate laughing and crying, "globus hystericus," and constriction in the throat with fainting, excitement and emotions (may rarely pass into mania) are common accompaniments. Defective will-power and self-control and disproportionately exaggerated effect of an external impulse may lead the patient to a delirious state which is often accompanied with or followed by hysterical amnesia, fugue and somnambulism, as a result of mental dissociation. HYPNOID states as trance, catalepsy and 'status hystericus' are the manifestations of severer forms. The twilight state, otherwise known as Ganser syndrome, is generally confined to prisoners who attempt to appear innocent: their purposeful nature is obvious. When psychic symptoms take a chronic course a morbid desire for sympathy leads to exaggerated symptoms and to some sudden transitions of character from joyousness to sadness or from apathy to alertness, even it leads to wilful additions of some real signs, as, raised temperature, factitious skin eruptions, self-inflicted wounds, etc. Finally, self-mutilation may be met with and suicidal threats and attempts may be made.

II. Motor symptoms.—In motor symptoms the movements are affected but not the individual muscles; and it will be convenient to describe the symptoms in different categories, as:

1. **Paralysis characters:**—(a) Paralysis is rarely absolute (asthesia abasia); (b) movements are opposed by contraction of antagonistic muscles; (c) no trophic change; (d) anæsthesia is common; (e) reflexes rather increased: plantar reflex flexor: no true ankle clonus but pseudo ankle clonus is fairly common; (f) electrical reactions normal; (g) duration may be transient or for years; and (h) the loss is of voluntary acts only: the muscles may act on involuntary functions as coughing, laughing, etc. **Distribution:** (a) paraplegia commonest; (b) hemiplegia; (c) monoplegia: usually with stocking or glove type anæsthesia; (d) laryngeal as aphonia and mutism, but the patient can phonate normally on coughing and laryngoscopic examination often reveals that cords are not sufficiently approximated; (e) paralysis of other cranial nerve is rare; and (f) sphincters unaffected.

2. **Spasmodic contractures:**—Far more common than any other motor symptoms. Onset is spontaneous or following emotion, pain, fit, injury or any other psychic disturbance. Spasms are powerful and often increased by efforts to relax but it is readily relaxed under anæsthetics. Its general character is entirely flexor in arms: in legs, toes are flexed; foot dropped and inverted and knees and hip are extended. Mono—, hemi—, and paraplegias are the usual distribution. Ptosis, trismus, and phantom-tumour which is originated from the contracture of abdominal muscles are rather rare manifestations.

3. **Spasmodic movements:**—Generally there are three types, as: (a) Irregular—choraic type; (b) rhythmical or repeated—habit spasms; and (c) complex and purposive.

4. **Tremors:**—Tremors or incoordinate movements usually of hands, either fine or coarse and irregular are often increased by 'voluntary' movements and may closely simulate those of paralysis agitans or dissiminated sclerosis or may take the course of "intention-tremor" type. Hysterical blepharospasm is fairly common.

The fact should be noted that due to excessive muscular efforts and respiratory struggle during the fit sometimes alkalosis is caused and consequently true epileptiform convulsion may supervene (MAJUMDAR).

III. Sensory symptoms.—These also should be described in different categories, as—

(a) **Pain:**—May simulate many diseases, e.g., appendicitis, caries spine, gastric ulcer etc. But commonest of all is 'clavus hystericus' (a sensation as if a nail is driven into the head).

(b) **Hyperaesthesia:**—In various areas—may be to light touch or to deep pressure.

(c) **Hystero-genic spots:**—These are sites of extreme tenderness and pressure on these spots often induces a convulsion or other symptoms. Common sites are ovarian, infra-mammary regions and over the dorsal vertebral spines.

(d) **Anaesthesia:**—It is far more important than any other sensory symptoms. It is to be noted that, apart from anæsthesia, muscular sense may be apparently lost or may be preserved. Usually the patient does not complain of the anæsthetic distribution which may be complete to all

sensations or occasionally of dissociation type. The distribution never corresponds accurately to that of spinal segments, nerve roots or nerve trunks. There may be unilateral loss of one or other special sense on the same side of anæsthesia. SAVILL is of opinion that 'sensory loss affects only cutaneous sensibility' but TIDY provokes that it also 'includes deep structures and mucous membranes within area.'

However, as regards types of anæsthesia the following are to be noted, as: (a) Hemianæsthesia; (b) glove type; and (c) stocking type. And moreover there are some peculiar points about it, as: (a) Allocheiria (rare)—the stimulus applied to one side is referred to the other side; (b) the affected area does not bleed much on pricking (TIDY); (c) after treatment, the affected side may be changed and then reverted; (d) duration cannot be foretold; and (e) sometimes 'yes-no test of Janet' is found positive, i.e., the patient is instructed to close his eyes and say 'yes' every time he feels a pin-prick and 'no' every time he does not feel it. The peculiarity is that the patient will say 'no' every time he is touched over the apparently anæsthetic area, thus indicating that really he feels but does not comprehend that he feels.

IV. **Special sense.**—1. The ocular disturbances are the most important and the various characters are: (a) Contracted field of vision, field diminishes in a spiral (perimetric observation) or unequal constriction of the visual fields in the two eyes (hysterical emblyopia); (b) dyschromatopsia: imperfect discrimination of colours; reduction greatest for blue and least for red, it is contrary to organic disease; (c) crossed emblyopia; (d) sometimes conjunctival anaesthesia may supervene; (e) hysterical blindness, often sudden, may be complete or incomplete; but in the complete form the patient may occasionally avoid obstacles placed in his visual path; and (f) rarely diplopia may be present.

2. **Hearing:**—Deafness of deaf-mutism may occur but it is peculiarly characterised by the fact that patient is wakened from sleep when called by name, but when awakened he cannot hear. Sometimes excessive sensitiveness to sound is characteristic (Hyperacusis).

3. **Absence of taste** is fairly common. Water is sometimes tested to be sweet.

4. **Smell** sensation is also impaired. Hysterical anosmia, i.e., loss of sensation to ammonia vapour, which is the function of the 5th, not the 1st nerve (SAVILL) is common in plumbers after gas explosions.

V. **Visceral, vasomotor and cutaneous symptoms.**—1. **Cardio-vascular system:**—(a) Tachycardia is commonest; (b) precordial pain and pseudo-angina; (c) hysterical personality occasionally may suffer from effort-syndrome (SAVILL); (d) flushing and sweating; and (e) 'Stigmata' or hæmorrhages into the skin are mainly, if not invariably, fraudulent.

2. **Respiratory system:**—(a) Loud barking cough without pulmonary disease, paroxysmal tachypnoea; (b) hiccough and yawning; (c) rapid respiration and deep breaths; and (d) hæmoptysis also may occur usually from pharynx (TIDY).

3. **Alimentary system:**—(a) Various types of dyspepsia with failing appetite or hyperchlorhydria and œsophageal spasms are not infrequent. Flatus and borborygni are common (peristaltic unrest).

(b) **Diarrhoea**—often resistant and of lenteric type. Constipation may alternate with it.

(c) **Vomiting**—in some patients nothing is retained and persistent vomiting may be alarming but generally the case is that certain types of food is vomited out but some other palatable food is retained. Any profound loss of weight seldom occurs. This type of malady should be judged very carefully because 'the chronic abdominal pain of hysterical women are often the cause of repeated laparotomies for adhesion, fixation, etc. The patient, unaware of the true cause of her malady, often invites operation with amazing eagerness' (SAVILL).

(d) **Phantom-tumours**—Due to the spasm of diaphragm with relaxation of abdominal muscles, intestines are distended with gas (hysterical acrophagy) and simulate tumours or pregnancy.

(e) **Globus Hystericus**—It is a sensation of a 'ball of wind' rising from the epigastrium to the throat; it is very distressing to the patient as it interferes with the swallowing.

(f) **Anospiasm** is another rare phenomenon.

(g) **Anorexia nervosa**—It is the most important as well as dangerous symptom as regards prognosis. It is a condition without any recognisable organic disease, characterised by complete loss of appetite resulting in severe wasting and pronounced loss of weight. Repugnance develops for all kinds of food, even a small meal gives a feeling of distension and nausea. But patient's physical energy is retained and she asserts that her health is perfect. It occurs mostly in young females with amenorrhœa. Temperature is normal, pulse often slow and basal metabolic rate low. Gastric juice is normal but constipation is often accompanied. There may be growth of downy hair on limbs and face (SAVILL.) Specific evidence of vitamin deficiency is rare (TIDY). Blood sugar curve is not constant. Peripheral circulation is often impaired.

Possibly, it is the only complication from which a hysteric patient may die, from cardiac failure, tuberculosis or coma (probably hypoglycæmic—TIDY).

4. **Urinary system.**—Anuria is much more common than polyuria. Incontinence never occurs except rarely by overflow. A large quantity of pale urine may be passed after paroxysm of fit.

5. **Locomotor system.**—Severe pain with superficial and deep tenderness and with muscular contraction may be felt in large joints as hip or knee. There is neither any change in radiogram nor any real shortening. In small number of cases œdema and warmth on joints may be noticed. Disuse atrophy may occasionally follow.

6. **Pyrexia.**—Temperature practically always normal; a slight rise may be found in a few cases, but hyperpyrexia (if not due to other diseases) mostly, if not invariably, proved fraudulent.

7. **Cutaneous symptoms.**—Dermatitis artefacta or certain self-inflicted minor injuries may be caused either from an idea of illness or to attract sympathy. Blueness, coldness and erection of hairs, etc., may sometimes be met with, particularly in hysterical paralysed limbs. Dermographism is common (SAVILL).

VI. **Sexual aberration.**—Sometimes present either in the form of impotence or vaginismus, or nymphomania.

Besides the brief symptomatology of hysteria described above, one more anomaly is left behind and that is traumatic hysteria or traumatic neurasthenia or otherwise called workmen's hysteria. In this syndrome, after an accident the patient develops hysterical symptoms. It generally occurs in workmen, especially those engaged in dangerous occupation and who work at a height from the ground (SAVILL). Particularly the head or spine injuries excite the hysterical symptoms.

Finally, before going to treatment, it will be useful for ready reference to have a graphic picture to distinguish between hysterical fit and epileptiform fit as regards their physical signs. (This is taken from SAVILL's system of Clinical Medicine).

Signs	Hysteria	Epilepsy
1. Periodicity and onset.	Attacks follow upon an emotional crisis. Never during sleep.	Attacks occur mostly at definite hours of the day or night.
2. Incontinence	Never incontinence.	Incontinence of urine during attacks. Albuminuria found later.
3. Self-injury	The patient is not hurt in the seizure, although onlookers may be.	Tongue, cheek or lip biting, with blood in the mouth.
4. Movements	The movements are purposive and spectacular, the eyes screwed up and the hands clenched. Never conjugate deviation of head or eyes, often convergent spasm of eyes.	Involuntary movements, are common if present, or have a definite march and are accompanied by conjugate deviation of head and eyes.
5. Breathing	Never stertorous.	Stertorous, with cyanosis.
6. Duration	Attack often prolonged specially before an interested audience.	Attack usually is of short duration.
7. Plantar reflex	Usually flexor and may be absent at the toes.	Extensor during and immediately after the fit.

Treatment.—As it is a disease of psychical origin, the treatment will also be mainly psychical. As mere relief of local symptoms is inadequate, a thorough investigation is essentially necessary to find out the emotional disturbances responsible for the syndrome and also the patient's tempera-psycho-analysis. The physician should assert a strong personality, and must first gain the patient's full confidence and "a combination of firmness and kindness with discretion is an essential part of his equipment" (HANDERSON). The patient should neither be intimidated with the diagnosis the actual mode and method of the treatment. The physician should infuse strength and courage into the patient's mind to make him or

her convinced that no organic lesions have taken place and he or she should recover from illness within a very short period. But the physician, if necessary, should explain the symptoms as manifestations of some other easily curable disease. One of the worst factors is the presence of over-sympathetic attendants; so these people should be removed. Next, environmental factor should be carefully judged and readjusted accordingly. "The thought of return to arena of conflict is, in some cases, insupportable, e.g., the childless woman will not return to her drunken husband" (SAVILL). In a nut-shell, patient's environment should be more tolerable.

However, to take up the treatment more systematically we should divide this chapter into the following groups and sub-groups:—(1) Psychological method of treatment including 'faith-therapeutic'; (2) Weir-Mitchell method of treatment; (3) Hydro-therapeutic; (4) Rest-cure and occupational therapy; (5) Organotherapy; (6) Hygienic and dietetic treatment; (7) Electrical treatment and external applications; (8) Medical treatment; (9) Treatment of individual symptom and complication; (10) Preventive treatment.

1. **Psychological method of treatment.**—Consists of (a) reassurance to gain patient's confidence and to arouse her self-control capacity; (b) Persuasion to impress her that no organic lesion is present but she is suffering from an easily-curable disease; (c) Suggestion to implant ideas of a corrective nature; here a very careful hypnosis may be necessary to gain entrance into her subconscious mind; and (d) Psycho-analysis, as an adjunct to Freud's method of mental catharsis. A minute study of patient's psychological life by some special method as dream analysis, hypnosis and free-association may reveal the mental want and imbalance of the patient, specially sexual—according to FREUD, and the object of treatment will be to remove the gap of memory and to clear up all doubt of the patient's psychic history and thus to make subconscious factor accessible to consciousness. JUNG has taken a special method—"word association"—on this line of treatment. Psychoneurotic rather than psychotic people get more benefit from the psychoanalytic method of treatment. Old aged patients and patients with organic disease are bad subjects to undergo this line of treatment. Details of this method should be taken from a higher text book.

Faith-cure is also a sort of psychological treatment. ST. PAUL remarked, "faith is the substance of things hoped for; the evidence of things not seen." Amongst metaphysical teaching and obsolete philosophy faith is the most wonderful and the best vehicle to get the best result after a course of psychological treatment. Faith is something more than relief. So, we may see that a single hypodermic injection of water may render a strange effect on a hysteric when she gets the faith on the physician and on the treatment.

This method of psychological treatment is efficacious both in acute and chronic cases.

2. **Weir-Mitchell method of treatment:**—Isolation, absolute rest, happy surrounding, easily assimilable nourishing diet, massage and electricity stand as the principle of this treatment. This treatment also is applicable to both acute and chronic cases.

3. *Hydro-therapeutic*:—French physicians recommended cold douches on the trunk and limbs avoiding the head and hyper-sensitive zones. The utility of this method is perhaps much less than other methods.

4. *Rest-cure and occupational therapy*:—Some physicians have got result with perfect rest and isolation. After being cured with rest, the patient should be given an appropriate occupation which does not include merely 'doing something' but an interesting and happy form of work which after successful completion evokes a pleasure in the patient's mind and naturally helps towards self-confidence. Specially the chronic cases are benefited with this method.

5. *Organo-therapy*:—Though the rationality is not decided yet this mode of recent therapy has come into prominence and for the purpose several endocrine preparations are in use as ovarian, orchic, pituitary, thyroid etc. Hysterics with uterine dysfunction may get result from the use of appropriate hormonal preparations. In this connection two other methods as convulsive or malarial therapy and hypoglycæmic or insulin therapy may be mentioned, although their evaluation is quite obscure.

However, only the chronic cases may have a fair trial of these methods of treatment.

6. *Hygienic and dietetic treatment*:—A regular habit, open air exercise, a regular cold bath and good moral environment are practically essential to all psychic patients and besides these palatable nourishing diet with high caloric value and vitamin-content (but not bulky) is of immense good. Phosphate containing diet may be specially stressed upon.

7. *Electrical treatment and external application*:—Faradism, galvanic current, ultra-violet or infra-red rays and other electrical methods of treatment may all be fruitful though the mode of action is not fully known but psychic impression of the treatment brings about a cure, even of an obstinate case.

Poultice, compress, massage, spray etc. all have their uses. Catchword of external application is "manner of giving is worth more than that which is given" (CHUNDRA).

According to merit of the case these methods are applicable to both acute and chronic cases.

8. *Medical treatment*:—It is practically absolutely vague to search for a specific or a routine medical treatment of hysteria, but something should be given only to usurp patient's confidence. For an ordinary case of hysteria a Bromide-valerian mixture or a Salicylas mixture is quite good. Anyhow, it is probably likewise to prescribe mixtures of a deep colour or with a bad taste or pungent smell. The same purpose may be served by an injection either of Calcibronat, or Valerian or nothing but an ampoule of water. A Methylene-blue pill or a pill of any other dye preparation may be prescribed, so that after a while discoloured urine may give a cure-impression to the patient. Where epilepsy is suspected to be present Dilantin Sodium treatment may be tried. General tonics with Phosphate and Strychnine may do good. Polyglandular tonics also are helpful.

9. *Treatment of individual symptoms and complications*:—It is the most important part of the treatment. Although no dogmatic measure

can be taken yet the following line of treatment of symptoms may be useful:

(a) *Fits*:—Cold affusion, a sharp faradic current, inhalation of ether, chloroform, ammonia or amyl-nitrite may be found successful. Burning vapour of a piece of a blotting paper may remove the fit. Morphine is better avoided. A sharp pressure on 'hysterogenic spots' may bring the crisis to a termination.

(b) *Anaesthesia or paralysis*:—Faradism, massage or passive movements are quite good.

(c) *Pain*:—Embrocation with Iodine and Methyl-salicylas or mustard oil externally and some analgesic internally may be sufficient.

(d) *Headache*:—Some Caffeine preparations may do good.

(e) *Contracture of the limbs*:—Passive movements and, in obstinate cases, general anaesthesia may prove beneficial. Treatment by suggestion is also advocated.

(f) *Aphonia and mutism*:—Ethylchloride spray or Faradic current to the nape of the neck may evoke the patient to shout as 'aha'. From this incident suggestive treatment (that the patient can phonate normally) may bring recovery.

(g) *Digestive troubles* as dyspepsia may require Tr. Asafetida; constipation may be removed by Castor oil emulsion or Agar-agar preparation; in persistent vomiting Bromide and Hydrocyanic acid may do good or Tr. Iodine 1 m. in 1 oz. of water every half-an-hour may be tried, and in obstinate cases rectal feeding may be necessary. In hysterical 'lock jaw' and dysphagia DR. CHUNDRA recommends injection of Apomorphin Hydrochlor to induce vomiting which subsequently removes the ailment. Oesophageal spasm is a very distressing symptom and it is very difficult to treat. In cases of failure of suggestive treatment, chloroform anaesthesia may bring an abrupt cure. In one case of the writer, when all other available measures failed, the patient was instructed to have a skiagram of the throat; he was given barium meal which was interpreted to be the specific medicine (!) and the skiagram picture to be the criterion of cure (!). However, the patient was cured thereafter.

Hiccough and phantom tumours: Alleviate under general anaesthesia. A mustard plaster may also be applied on epigastrium. DR. CHUNDRA recommends in obstinate cases an injection of twenty drops of iced distilled water into the diaphragm.

Anorexia nervosa: Rest, isolation and psychotherapy, and restricted but gradually increasing and palatable diet certainly are measures of utility. Stomach tube feeding may be necessary. Dilute Hydrocyanic or Hydrochloric acid may be tried. Pieces of ice or ice-cream may be given. Stomachics may be tried. Menthol or Chlorbutol may do good. Parenteral Glucose and vitamins, and hormonal preparations are of immense benefit.

(h) *Retention of urine*:—Ice application on the bladder region and a mixture of Tr. Asafetida, Tr. Valerian, Ammon Chlor, and Inf. Buchu may be helpful.

(i) *Hyperpyrexia*:—Cold application on head and Quinine internally may be useful. CHUNDRA advocates a mixture of Tr. Valerian Ammon, Tr.

Quinine Ammon, Liq. Ammon Citras, Spt. Ammon Aromat and Aqua Chloroform.

Anyhow, inspite of all the scientific manoeuvre, procedure and endeavour some hysterics may not be amenable to treatment but some sort of empirical or spiritualistic activities still domain on them. An 'Ojha' comes, he utters some 'Mantras' and his obscured veil of charlatanism cures the patient.

10. *Preventive treatment*:—To speak the truth, nervous heredity is still beyond the physician's control yet, neurotic tendencies may be minimised by a wise education and skilful up-bringing, with balanced diet (with excess of phosphatic articles), open air exercise and good moral environment. Love for fine arts and music is much beneficial. In some women matrimony plays a good part.

The last, but not the least, a further paragraph should be devoted to another aspect of hysteria and that is 'hysterical personality'. With due apology and cordial gratefulness and thanks to Dr. DEB, the writer is presenting these excellent lines from his book.

A handbook of psychological medicine:—"These people (hysterical personalities) are ambitious and social. They always want to remain in the lime-light (centre of the stage feeling)—in this way they deceive themselves. They appeal to the immediate moment—the world at the moment is all that they care. Their judgement is faulty and their emotional response superficial and distractible. They do not go deep—anything new has a charm for them. Hysterics are easily influenced by persons and ideas that appeal to them—they are very much suggestible. They have good memory but it is one-sided. They are fond of day-dreaming. Their emotions are easily roused—they are exceedingly excitable and temperamental. Their behaviour is childish. Any sort of pain is terrible for them. They are untruthful and unreliable. They are often incapable of permanent affection or sexual relationship—they are good short time flirts. They frequently suffer from sexual frigidity."

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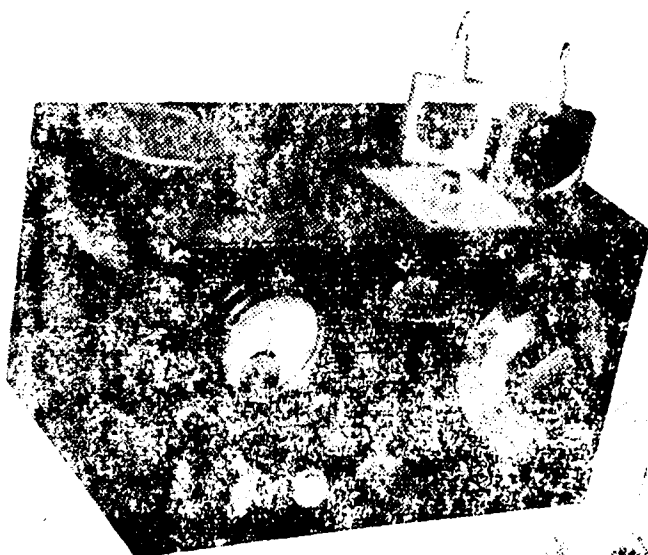


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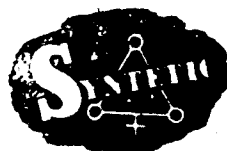
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Cataract*

C. K. BHALERAU, M.B., B.S., L.O. (MAD.),

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OPAQIFICATION of the lens, as a result of the coagulation of its proteins, is known as cataract.

Normally the lens is a transparent colourless body, which allows the rays from an external object to pass through it and form an image upon the retina. When the lens becomes opaque, no rays can pass through it and as such, a condition of temporary blindness sets in.

Anatomically, the lens is situated between the iris in front and the vitreous humour behind. On either side, it is supported by the suspensory ligament which is attached to the ciliary body. Thus the lens along with this ligament divides the eye ball into an anterior and posterior chamber.

The lens consists of nucleus, the cortex and a capsule. The capsule is adherent posteriorly to the vitreous humour, while anteriorly it is free.

Lens metabolism.—The constituents of the lens are divided into two groups (1) Inorganic and (2) Organic.

1. The inorganic contents are the salts of sodium, potassium, calcium, magnesium and phosphorus. In a cataractous lens, the potassium salts diminish considerably while the calcium salt in a cataractous lens is above 35%.

2. The organic contents are—(1) *Albuminoids*—They are insoluble in water and constitute about 17% of the total protein content.

(2) *Globulin* :—They are soluble in water and are of three varieties—

(a) α Crystalline	... 11%
(b) β Crystalline	... 6.8%
(c) γ Crystalline	... 0.2%

The total protein content is therefore about 35%.

Coagulation of protein, which ultimately leads to the development of cataract, occurs in two stages.

(1) Denaturation by hydrolysis, so that the colloidal system becomes more labile.

(2) Agglutination.

This process of coagulation is an irreversible chemical change, and hence, once the cataract develops no medicine can either check the progress or cure the condition. The only treatment, therefore, resolves itself in its surgical removal when it gets mature.

The lens has no blood supply of its own and its metabolism is therefore dependent upon a system of autoxidation. It receives its nutrition from the aqueous humour.

Cataract is classified into the following groups.

1. *Primary* :—Its cause is not known.

* Specially contributed to THE ANTISEPTIC.

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2. *Secondary*.—It is due to the malnutrition of the lens, brought about as a result of some local disease of the eye or general constitutional disease.

3. *Complicated*.—It is a primary cataract associated with some local or general disease.

4. *After cataract*.—Opacities left after removal of a cataract due to persistence of the posterior and a part of anterior capsule is known after cataract.

Primary cataract.—We still do not know why, in some individuals, opacification of the lens occur. It is further subdivided into—(a) Congenital; and (b) Acquired.

CONGENITAL PRIMARY CATARACT.—This condition occurs in infancy and early childhood and is often associated with some congenital defect in some part of the body. Thus, for instance, the Lamellar cataract is often accompanied by defective enamel in some of the permanent teeth.

Following varieties of the primary congenital cataract have been observed.

(1) *Anterior polar cataract.*—It may be further subdivided into anterior capsular cataract and anterior cortical cataract, depending upon the site of opacification. Anterior capsular cataract develops as a result of the proliferation of the cells of the capsule brought about as a result of any contact between the capsule and the cornea. The opacity may spread from capsule to the cortex leading to a total cataract.

(2) *Lamellar cataract.*—This condition occurs as a result of malnutrition at some stage or intra uterine or early infantile life. The opacities radiate outward from the centre and appear like the spokes of a wheel when seen through an ophthalmoscope. Thus the concentric rings of opacity account for the successive period of malnutrition. The cause of malnutrition is either rickets or congenital syphilis. A history of convulsion is common.

The cataract is usually stationary throughout life. Both eyes are usually affected. It is often accompanied by a defective enamel in some of the permanent teeth. The teeth have an eroded appearance. The incisors uterine life and canines are chiefly affected.

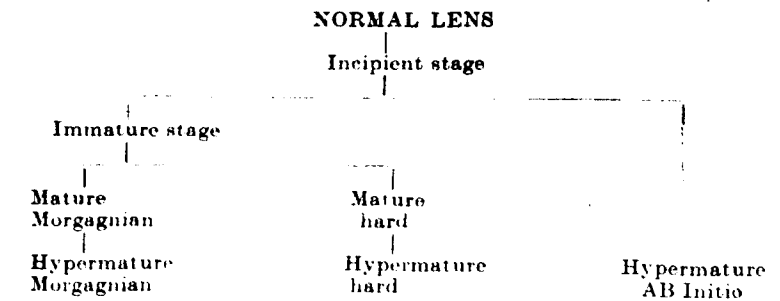
The usual diameter of the opacity and the permanent teeth affected indicate the period of malnutrition which corresponds to intra or a period shortly after birth.

(3) *Posterior polar cataract.*—This is generally due to the persistence of posterior hyloid artery. So in the strict sense, it is not a cataract but an opacity at the posterior pole of the lens.

However there is a condition like true posterior polar cataract occurring in some individuals.

There are other varieties of congenital cataract like fusi form, discoid, punctate cataracts occurring in children. They are named according to the shape and size of the opacity and needs no further explanation.

ACQUIRED PRIMARY CATARACT.—This type of cataract occurred in later years of life. It is classified into following stages—



Incipient stage.—The most important symptom at this stage is the gradual loss of distant vision. Near vision is not affected to any great extent. Patient sees better in a bright light. When seen through an ophthalmoscope, no definite opacities are noticed in the lens. The details of the fundus are seen, but not as clearly as that in a normal eye. The distant vision is reduced to about 6/9 or at the most 6/12. However, as the disease progresses, dust-like opacities appear at the periphery of the lens and then gradually spread towards the centre.

Immature cataract.—In this stage it is the centre of lens where opacities develop. The cortex is transparent to light and this is responsible for the sign of the shadow of the iris being visible on the central unclear opacity, when the eye is illuminated obliquely. As the central portion of the lens gets more opaque than the periphery naturally the patient sees better in a dull light than in a bright one, because in a dull light the pupil dilates and allows the rays to pass through the periphery which is less opaque. The lens gives an appearance that of a faint bluish sheen which is quite unmistakable.

Mature cataract.—It is a condition in which both the centre (nucleus) as well as the cortex become opaque. Mature cataract is of two varieties: (a) Morgagnian and (b) Hard.

Mature Morgagnian cataract.—Here the nucleus is very small, while the cortex undergoes liquefaction and swells up pushing the iris in front, thereby making the anterior chamber shallow. Sometimes this leads to increased intraocular tension. Pupillary reaction is sluggish though normal. Generally the fluid from the cortex gets absorbed and the lens assumes a normal size.

Hard mature cataract.—In this variety the nucleus reaches a larger size. The cortex does not become liquefied and retains considerable degree of adhesion to the capsule.

Hypermature ab-initio.—Here the nucleus is not well formed and the whole lens is transformed into a viscid homogenous material firmly adherent to the capsule. This variety of cataract occurs in the age between 35 and 45 years.

After having discussed the primary cataract, we come to the study of secondary cataract which develops as a result of any of the following conditions—

1. *Traumatic cataract.*—The trauma, which ultimately leads to cataract, is of two kinds.

(1) Blind trauma in which the cornea is not injured but the trauma is sufficient enough to bring a partial dislocation of lens, with injury to its capsule resulting in the onset of cataract.

(2) Penetrating trauma, in which the cornea is injured and the anterior capsule of the lens is damaged. This variety of cataract is very dangerous and requires great clinical skill in dealing with the case. If the ciliary body is injured there is every possibility of developing sympathetic ophthalmitis and hence in all such cases enucleation should be done early. In other cases, the treatment should be done in a most cautious manner depending upon the symptoms. Once the wound in the cornea is healed up and the eye is quiet, only then the cataractous lens be removed either by intra capsular method or needling.

2. *Glaucomatous cataract*:—In glaucoma, it is the degenerative process, which ultimately affects the metabolism of the lens, resulting in the development of cataract. The cataractous lens appears greenish. The pupil is oval and does not react to light. The tension is high and there is neither perception nor projection to light. Such an eye is quite useless for operation as no benefit is likely to occur after the operation.

3. *Cataract secondary to irido cyclitis*:—Chronic irido cyclitis often leads to the development of cataract as a result of chronic irritation of the capsule.

The best way to deal with the condition is to remove the cataract by intra capsular method after a preliminary iridectomy.

4. *Diabetic cataract*:—Diabetics in young adults assumes a malignant role and, in such cases, the lens metabolism suffers and hence becomes cataractous. It is necessary to treat the diabetes first and then remove the cataract.

Having thus dealt with the different varieties of cataract we now come to the line of treatment to be adopted in different varieties.

The treatment of cataract differs according to age and the type of cataract. It is therefore divided according to the following groups:

Treatment of cataract occurring in children and adults below the age of 30:—Needling is the operation of choice for cataracts occurring in infants and children. At this age, no nucleus is formed and hence, once the capsule is ruptured, the cortex gets absorbed by the aqueous humour and the vision is restored to normal after wearing suitable glasses. There have been cases on record where even after needling the cortex (which is at times jelly-like in consistency) does not get absorbed and hence requires repeated needling. If such a condition is diagnosed clinically before the operation, then instead of needling, the lens should be extracted by intra-capsular method.

As to the cataract in young adult, there is a common belief that no nucleus is formed below the age of 30 years. Col. SMITH is however against this view and he has put on record cases where the nucleus has formed as early as 15 years of life. Therefore, he is of the opinion that, in adults, the cataract should not be needled but extracted by intra-capsular method.

Treatment of senile cataract.—1. *Incipient stage*:—Col. SMITH has recommended the following line of treatment which, according to him, improves the vision considerably and maintains this improvement for a long time. He advises sub-conjunctival injection of Cyanide of Mercury 1 in 4,000 solution followed by the application of Yellow Oxide of Mercury 1% ointment. With this treatment, the vision has been restored from 6/12 or 6/18 to 6/9 or even 6/6.

Col. SMITH attributes this improvement to the fact that the injection produces a hyperæmia and irritation of the conjunctiva and thereby improves the nutrition of the lens.

2. *Immature cataract*:—Most of the surgeons are in the favour of waiting to get the cataract matured fully before advising its extraction. They believe, and rightly too, that there is a risk in removing an immature cataract especially if it is removed by extra capsular method. If an immature cataract is removed after capsulotomy, much of the capsule and cortex (which is adherent to the capsule) is left behind resulting in the formation of secondary cataract which can never be cured even after needling.

Col. SMITH who is a great advocate of removing cataract by intra-capsular method, is of the opinion (and he has given proof to that effect by his results) that if the immature cataract is removed by intra-capsular method no complications set in and the vision is restored to normal after wearing suitable glasses. He therefore recommends this method for immature cataracts. The advantage in such a removal is, that the patient is not required to wait indefinitely for the lens to get matured. Sometimes this period of waiting is a pretty long one and as such it greatly incapacitates the person from doing any work. Thus from the economic point of view it is a great handicap to the patient.

The technique of the operation is not difficult and once the lens comes out intact, there is no danger of after cataract or post operative irido-cyclitis.

3. *Mature cataract*:—The only treatment for a mature cataract is its surgical removal. There are two techniques by which matured cataract can be removed.

(1) Extra capsular, i.e., removal of cataract after capsulotomy either with or without iridectomy.

(2) Intra-capsular, i.e., removal of cataract along with the capsule with or without iridectomy.

I will just in short enumerate the steps of operation in the two techniques mentioned above, as more detailed information could be had from any of the text books of ophthalmology.

1. *Extracapsular extraction of the lens*:—First a corneal incision is made by a Graefe's knife, the size of the incision being about 2/5th of the corneal circumference, i.e., about 144°. The capsule is then ruptured by a capsulotomy and the nucleus is removed by a gentle pressure applied from below upwards. After this some people prefer to do a button-hole iridectomy to prevent its prolapse, and then the anterior chamber is irrigated. The cut ends of iris are replaced properly and the eye is bandaged.

2. *Intracapsular extraction of cataract*:—Corneal incision is made as before, but here in this case, the size of the incision is bigger, i.e., about half the circumference of the cornea—180°. After this incision, it is advantageous to do a broad peripheral iridectomy so as to make enough room for the lens to come out. Then by tenotomy hook, the lens is first dislocated from its attachment at the suspensory ligament and then it is gradually pushed upward till it comes out along with the capsule. The pressure must be just sufficient as not to disturb the vitreous humour, as otherwise, there is a possibility of its escape, even before the delivery of the lens leading to serious complications.

It is worth while considering the advantages and disadvantages of the two techniques before deciding upon the technique to be adopted.

Extracapsular method.—*Advantages*:—(1) This technique is easier, especially for a beginner, because there are very few chances of the vitreous escape during the operation. A beginner always dreads this calamity and hence it is much desirable for him to select this technique.

(2) There is a diminished risk of prolapse or iris.

(3) There is no risk of developing post-operative glaucoma.

Disadvantages:—(1) More instruments are required.

(2) There are always chances of developing secondary cataract requiring needling. People who have undergone the first operation, are very much reluctant to submit themselves for a second one, especially in India.

(3) Due to secondary cataract, the vision cannot be restored to normal or near about normal. Usually the vision remains up to 6/18 or 6/12 after wearing suitable glasses.

(4) Complications like iritis and iridocyclitis are very common after the operation.

(5) As more instruments are required and as anterior chamber needs irrigation, there are chances of developing intraocular sepsis.

(6) Lastly dressings have to be changed quite frequently, in order to avoid the onset of any complications and as such more additional staff is required to do the post-operative treatment.

Intra-capsular method.—*A. Advantages*:—(1) This technique is simpler. Once the surgeon knows how much pressure is required to detach the lens from its attachment (without disturbing the vitreous humour) it is quite simple to remove the lens along with its capsule.

(2) As iridectomy is done, there is no danger of prolapse of the iris.

(3) As no capsule or cortex is left behind, there are no chances of developing either secondary cataract or iritis. Therefore the vision can be restored to normal with suitable glasses (provided of course that the fundus is normal).

(4) Post-operative complications are few or nil and hence no post-operative treatment is required. Thus there is no necessity to engage any extra staff to do the post-operative treatment. Thus with a small staff it is possible to undertake mass work.

(5) As there is no secondary cataract, needling is not required.

(6) As few instruments are required and as there is no necessity to irrigate the anterior chamber, there are very few chances for developing any post-operative intraocular sepsis.

Disadvantages:—(1) Prolapse and at times even loss of vitreous, during the delivery of the lens is common.

(2) As a result of the escape of vitreous, there is a possibility of developing sepsis.

(3) Rupture of the capsule of the lens (especially a Morgagnian one) during its delivery is common.

(4) If the corneal incision happens to be of a small size, it may be found difficult to deliver a hard cataract.

After having said all this, I would like to sum up and say that the extra-capsular methods are suitable for a beginner while the intra-capsular method should be undertaken by those who have had enough experience and confidence in ophthalmic surgery.

Finally I will deal with some of the post-operative complications and their treatment before I close the subject.

1. *Conjunctivitis*:—Simple Boric wash and instillation of Silver Nitrate 1/2% solution is all that is required.

2. *Increase in the intraocular tension*:—This condition can be dealt with by frequent instillation of Eserine 1/2% solution and application of leeches to the temple.

3. *Iritis and iridocyclitis*:—Frequent instillation of Atropin 1% solution is necessary to prevent this complication.

4. *Blood in anterior chamber*:—Injection of Calc. Gluconate and vitamin 'C' with local instillation of Dionin 2% solution and Yellow Oxide of Mercury 1% ointment often clear up the condition.

5. *Shallow anterior chamber*:—Intraorbital injection of 'Cyanide of Mercury 1 in 4,000 1-2 c.c. has been found to remedy this condition.

6. *Sepsis*:—Sulpha drugs, when given in time and sufficient quantity can prevent the onset of sepsis. But if sepsis has already taken place, Penicillin injections (sub-conjunctivally) as well as 2-hourly instillation of Penicillin solution has been found to remove the sepsis and the vision has been restored to normal.

7. *Secondary cataract*:—Needling is the operation of choice to remove these adhesions.

8. *Prolapse iris*:—Cauterisation of prolapsed iris has given good results.

9. *Intraocular haemorrhage*:—Very little can be done in this condition. Enucleation of the eye ball should be done to prevent further damage and relieve the agony.

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3. Sir John Herbert Parson—'Diseases of the Eye.'

Sulpha Drug in Small-pox*

S. G. SONARNAY, M.B., B.S., (BOM.),
Ambarnath (G.I.P.)

IN this small article, I shall put before the medical profession my observations of small-pox epidemics and the use of Sulpha drugs, during the last two years, which I hope will be of great use to those who are practising in the small-towns and in villages where special treatment cannot be carried out due to lack of facilities.

Three main drugs were used in order of merit:—(1) Sulphathiazole; (2) Sulphapyridine; (3) Sulphanilamide.

The above Sulpha group of drugs which were tried in small-pox cases proved to abort or minimise the pustular stage of 'pox' if exhibited in due time. Severe confluent cases showed complete recovery without any complications, and when the lesions were completely healed and scabs

* Specially contributed to THE ANTISEPTIC.

fallen there were slight or no pittings or depressions which we used to see in the cases which were not given any Sulpha group of drugs. After two or three months skin becomes practically normal and there is hardly any evidence of small-pox. These results are well marked in those cases which are vaccinated at least once in their childhood.

I have observed children 6-8 years who were successfully vaccinated on both the arms during infancy, suffering from small-pox whenever there is an out break of an epidemic. This shows that primary vaccination confers immunity for an average of five years. I have seen some of the cases in adults also who were revaccinated two years back. But in majority of them the lesions were discrete and very few, and other signs and symptoms like pyrexia, toxæmia, or prostration were absent or very mild in nature.

Of all the drugs Sulphathiazole proved to be the best. The patients hardly showed any signs or symptoms of intolerance or toxicity. Sulphapyridine caused nausea and vomiting in some cases and giddiness and frequency of stools in others. Sulphanilamide does not seem to abort the pustular stage completely and in some cases secondary fever was noticed. Nausea and vomiting were less with Sulphanilamide as compared with Sulphapyridine.

Intolerance or toxicity can be lessened or avoided if Sodabicarb and Nicotinic acid are given along with Sulpha group of drugs. Patient should be encouraged to drink large amount of fluids and if he has good appetite may be allowed to take his normal diet. Children tolerate Sulpha group of drugs well.

It is advisable to exhibit Sulpha drugs the moment you diagnose or when you see distinct papular stage. The dosage should be according to age. But it is not necessary to give massive doses which we give in cases of pneumonias or meningitis. If the case is diagnosed early Sulphathiazole 1 tab. Q.D.S. should be given for seven or eight days. If the case comes under observation late, Sulphathiazole 2 tablets Q.D.S. or T.D.S. should be given for five or six days. If iron tonics such as Liq. Ferri Perchlor with Liq. Strychnine and Hydrochloric acid and Liq. Arsenicalis are given along with Sulpha group of drugs convalescence is hastened. I have found the following prescription very useful:—

R Liq. Ferri Perchlor	...	℥ x
Liq. Arsenicalis	...	℥ ii
Acid Hydrochlor dil	...	℥ x
Liq. Strychnine	...	℥ ix
Aqua ad	...	℥ i
Mft.	...	℥ i. t.d.s.

It is also useful to administer vitamins A and D in the form of Cod-liver oil.

Hæmorrhagic small-pox cases did not come under my observation and in those cases one may not get good results and the mortality rate might be high. Other complications should be treated symptomatically.

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Unfamiliar Smallpox*

DR. A. C. KAPOOR,

Assistant Medical Officer, Baghaura, B.N.R. (C.P.)

SMALL-POX is a very common disease. Persons who suffer from it are either unvaccinated successfully or unprotected by a previous attack. There are exceptions, rarely a man vaccinated successfully may get an attack of small-pox, but it is of a modified form, of shorter-duration and passes off easily, or a man who had small-pox before in very rare occasions gets another attack which again is of a modified form and does not cause much harm. There are exceptions and exceptions should remain exceptions and should not become a rule or very frequent.

In the present epidemic of small-pox in Chattisgarh Division of Central Provinces, which is raging severely for the last 5 months (now sufficiently under control), such diversions from normality have been seen that I feel tempted to put them before the medical world.

Almost all the authorities on medicine are unanimous in their view that successful vaccination affords immunity against small-pox for 5 to 7 years. There is nothing to doubt the efficacy of vaccination, but in the present epidemic, it was found that vaccination was not of much help in many cases. Such cases were not infrequent where persons had been vaccinated successfully a month or two or a year before and yet they suffered from small-pox and in many cases they suffered badly from it, in some cases even losing their lives.

It is commonly known that one attack of small-pox affords immunity for the rest of the life, secondary attacks are rare. If at all there is a second attack it is of a modified form and passes off easily. About half a dozen such cases came to my notice in this epidemic where persons had an attack of small-pox 2 months, 4 months or some years back, and they were again attacked by small-pox and they in no case suffered less severely than others and in some cases suffered even more than those who were unprotected by a previous attack. I am citing two such cases below:

1. Shanti, a girl of 11 years, had an attack of small-pox in the month of March '46, she again got an attack of small-pox in the first week of May 1946. Temperature went upto 104.5°F., there was marked toxæmia and prostration, the patient became semi-conscious on the third day when typical rashes appeared. On the 4th day the patient began collapsing, the pulse became very feeble and rapid. Intravenous Glucose 25% 50 c.c. was given and intra-muscular Coramine was given. This had no effect and the child began collapsing more and more. Another injection of Coramine was given, but in spite of all efforts she expired on the 4th day.

2. Swaroop, a male child, aged 4 years, had an attack of small-pox in the month of March '46, he again got an attack of small-pox on 24th May '46. On 26th May the temperature went upto 105.5°F., there was muttering delirium and semi-consciousness. Cold sponging was done and ice applied over the head. Typical rashes appeared on the 27th May. He is much better now and recovering.

Although no age is immune to small-pox and persons of any age can get it, yet I for the first time saw infants aged 10 days and 12 days getting

* Specially contributed to THE ANTISEPTIC

small-pox, without their mothers or anyone else in the family getting it. The point of interest in these cases is, where did these infants get the infection, while in the womb itself or outside? A child may be born or aborted with rashes of small-pox from a woman who is actually suffering from small-pox, but if the women did not have small-pox and their children aged 10 and 12 days show small-pox eruption then where did they get the infection from? If the children got the infection while in the womb, the infection could have reached either per vagina or through the mother's blood. If the infection would have been through the mother's blood she herself should have suffered from small-pox, in case she is immune to small-pox, then the child in the womb should also be immune to small-pox, because the same blood is running in the veins of the child in utero and there should be anti-bodies in that blood too. Then could the children get the infection per vagina? If the children got the infection after birth, was the incubation period so short that the rashes appeared on the 10th and 12th day, while the usual incubation period is 14 days when the rashes appear?

Cinchonine in the Treatment of Malaria*

S. N. DAS, I.M.F.,

M.O., Samra U.B. Dispensary, Formerly M.O., M.I. M. Ch. Dispensary,
Samra P.O., Hooghly District (Bengal).

I READ with much interest the article entitled, "My experience with Cinchonine" by Dr. G. N. SINHA (Barwadin) in the January, 1946, issue of the *Antiseptic* and in reply I have much pleasure to record some of my observations, based on 17 malarial cases treated with the drug.

Action of Cinchonine Bihydrochloride (Acid Cinchona Hydrochloride).—Cinchonine salts are less liable to be precipitated than Quinine; hence they cause less pain than Quinine when injected intramuscularly. The effect of intramuscular injection of Cinchonine Bihydrochloride is as rapid as that of intravenous injection of Quinine. So by injecting Cinchonine Bihydrochloride one can avoid the risk of intravenous Quinine injection. Cinchonine Bihydrochloride should be injected *intramuscularly* as early as possible during the attack of malaria, in order to control the fever. The dose recommended for adults is $7\frac{1}{2}$ grs. or 10 grs. (These are the observations of some manufacturers).

In the latter part of 1943 and early part of 1944, when reliable Quinine Bihydrochloride ampoules were not easily available, I treated 17 malarial cases (6 adults and 11 children) with intramuscular injection of Cinchonine Bihydrochloride. The results were quite satisfactory—100% clinical cure.

Of these 17 cases, only three children (between 7—10 years) exhibited symptoms of intolerance. To each of them $7\frac{1}{2}$ grs. of Cinchonine Bihydrochloride was injected. The symptoms they exhibited started after about an hour of injection and lasted for 2–4 hours and the symptoms were giddiness, nausea followed by vomiting, sweating with thready quick pulse and anxious expression.

All recovered with the following line of treatment:—

- (1) Perfect rest in bed and application of cold water to the head.

* Specially contributed to THE ANTISEPTIC.

(2) An Alkali-Bromide Belladonna mixture, one dose every 3 hours.

(3) Solution Adrenaline Chloride 1 in 1000— π x. under tongue, every 3 hours and, or Liq. Coramine— π xv. every 3 hours.

(4) A Sodi-Bicarb Glucose lotion to sip. ad. lib. with dab-water, barley-water, sugar-candy water and plain water as diet.

The possible cause of toxic symptoms seems to be due to big dose injected. The children could easily tolerate (which was proved by other cases) gr. v C. Bihydrochloride, and this seems to be the safe dose for a patient under 12 years and less for youngsters.

15 grs. of Cinchonine Bihydrochloride, as Dr. SINHA used for an adult, was a heavy dose for an average Indian (of weak constitution). 10 grs. is the safe and proper dose for an adult.

The toxic effects can be avoided by observing the following rules:—

(1) By using appropriate doses—10 grs. for an adult and 5 grs. or less for a patient under 12 years.

(2) Injection should be given at least 2 hours after a heavy meal.

(3) Put solution Adrenalin Chloride 1 in 1,000 π x—xx, under tongue, at least 15 minutes before injection.

All the alkaloids of Cinchona (together with Quinine) have synergistic action as anti-malarial remedy and is therefore more effective than Quinine alone. This is why smaller doses of Cinchonine Bihydrochloride are able to control the fever. For the same reason many hospitals stock Quinine-Cinchona mixture (equal parts) which is more effective and cheap than Quinine. This combination (with Iron-Arsenic) is especially of value in chronic-malarial cases. But the consensus of opinion is that provided its (Cinchona) composition is standardised, it is but little inferior to Quinine both in the production of clinical and radical cure.

Cases and Comments

Hormone Therapy

In the treatment of enlarged prostate

SUBODH CHANDRA SANYAL, B. SC., M.B.,

Nat. & Rajshahi.

SURGICAL diseases of the prostate gland are of various types and the treatment varies accordingly. The condition of the hyperplasia of the gland is considered to be due to endocrine imbalances. The hormone therapy in the treatment of the obstructing prostate gland is undoubtedly the form of treatment which is of greatest interest at the present moment. Accurate diagnosis must be carried out with the greatest care. Rectal examination reveals at least fifty percent of the cases. Laboratory test of serum acid phosphatase and blood urea are the best, and most important guides in the treatment of enlarged prostate.

Recently I had the opportunity of treating a few cases of enlarged prostate, the details of which are given below.

(1) M., aged 62 years, complaining of pain, frequency of micturition. Urine dribbled drop by drop. He had to use catheter morning and evening for the last six days. On rectal examination enlarged prostate and pain on pressure were noted. He had no history of gonorrhoea. I started

Testatone Propionate injection and rectal massage. After three injections the patient became cheerful and began to pass urine freely. The prostrate became smaller in size.

(2) An old man, aged 68 years, complained that he had difficulty in passing water for the last three months. Evening rise of temperature, pain, cachexia, and dribbling of urine drop by drop for the last four days. Rectal examination, hard, enlarged, prostrate, secondary deposits in the pelvis revealed. He became almost bedridden. I thought it to be a case of malignant nature from clinical signs and symptoms, because laboratory test in the mofussil is impossible. I began to inject Stilbesterol, four injections he was able to get up and pain and cachexia responded quickly.

From the above cases, I find that hormone therapy is by far the most hopeful line of treatment in the disease of prostrate which has yet been evolved.

M. J. very thanks to Dr. P. C. Basak for his kind assistance in my cases.

Spontaneous Recovery in a case of Mental Depression (Schizophrenic)

After Ammonium Chloride "Shock" Therapy

K. SARAIN RAO, M.B.

Assistant District Officer of Health, Health Unit, Thanjavur.

BEFORE proceeding with my case, I would like to stress one point to the profession in general and to the mental specialists in particular, that I do not profess myself legally competent to treat mental cases, but having worked in a most up-to-date mental hospital under the able guidance and personal supervision of a specialist for a period of 7 years, I felt rather much inclined to narrate this case which I came across in a mofussil dispensary, which is much interesting from the point of view of treatment and which could be easily carried out in any mofussil dispensary.

R., a Hindu Brahmin, 26 years, widower and cook by profession, was brought to me on 1-2-1946 for consultation and treatment.

Family history.—Nothing specially important; no evidence of mental disorder either on the paternal or maternal side, and no history of alcohol or fits in the family.

Previous history.—Birth and early childhood uneventful; had only primary education and became a cook in his 16th year; married 2 years ago; conjugal relationship quite harmonious; wife died 6 months ago on account of childbirth; about a fortnight after the death of his wife gradual mental depression was noticed for which all sorts of "quack treatment" were tried which proved in vain; patient's condition gradually became worse day by day and even alarming and consequently as a last resort allopathic treatment was sought.

Personality and condition on examination.—A young man of an æsthetic build, very weak and anæmic (R.B.C. count could not be made for want of materials). Hb. 45%.

Physical:—No other signs suggestive of any organic disorder with the exception of a thickly coated tongue and obstinate constipation.

Mental:—Patient very dull, depressed and apathetic; indifferent to surroundings, seclusive and did not usually respond to questions which if repeated again and again answered in monosyllables; no emotional responses to stimuli; used to sit by himself morbidly preoccupied in thought, at times repeating the name of his wife over and over again; indifferent about food and usually dirty in habits; insomnia present.

After examination, the relatives were advised to take the patient immediately to the Bangalore Mental Hospital which is near by but they refused to do so on economic and superstitious grounds; obviously I was forced to take up the case, and the treatment adopted by me was as follows:—

General:—(1) Attention to bowels by enema or mild purgatives as and when necessary. (2) Heavy doses of Liver Extract Vitamins (particularly B and C), Calcium and Iron. (3) General care and nutrition.

Special:—After making myself quite sure that he would withstand a "Shock" treatment, one fine morning, I prepared him and gave him a 10 c.c. of (10%) solution of Ammonium Chloride intravenously with all preparations to attend to the expected fit; instead of that, he gave a very violent jerk, and shouted loudly calling his wife's name, and soon after became drowsy and fell fast asleep; after about 2 hours and 40 minutes he opened his eyes and began to talk to his parents in quite a normal way as though a heavy cloud had been taken off from his mind; the recovery was uneventful since that shock, but the general routine "pick up" treatment was continued for another month. From reports that I am getting it is learnt that he is quite all right both mentally and physically.

A Case of Complicated Duodenal Ulcer

DR. HARIDAS DE.

M.D., Chatmohar (Palna) Charitable Dispensary and Auxiliary Government Hospital, Late Senior House Physician, Campbell Hospital, Calcutta.

ONE fine morning in the early part of March '46, I was called in to see a Hindu adult of about 30, seven miles off from Chatmohar.

Past history.—The patient had an attack of duodenal ulcer about 10 years back for which he had to seek admission in a hospital in Calcutta, where after medical treatment of about 3 months he was cured and discharged as he refused surgical operation. After returning home he led the life of strict diet as advised by his physicians for about 8 or 9 years. For the last one year, as he had the idea that he was radically cured and as he had no symptoms of acidity or dyspepsia, he took all diets freely. About three months back he complained of pain in the abdomen after heavy diet and complained of acidity.

He then began to take carminative mixtures to no effect. He then went under the treatment of a local physician of his village who gave him a course of 6 injections of Emetine Hydrochloride gr. $\frac{1}{2}$ intramuscularly, after which he was rather improved. 3 days before my seeing the patient one day the patient took sufficient quantity of 'Khichini' with curry of egg after which he complained of severe pain in the abdomen and high acidity with black semi-solid stool. He took carminative mixtures, lemon salt water, but no improvement of his acidity was noticed. One day before my seeing the patient he passed black tar stool, 4 times, day and night, owing to which he was very weak and went downhill and I was called in to see him.

On physical examination.—I found his tongue like blotting paper, white-conjunctiva pale white. The patient was found very weak and anæmic and pulse which was of typical hæmorrhagic type was 110 p.m. No temp. Abdomen tender on palpation on the duodenal area. Liver just palpable below the costal margin. No spleen was palpable. Heart—both sounds are very weak and feeble. Lungs—nothing abnormal could be detected. Bowels 3 or 4 times in 24 hours which are nothing but decomposed blood—absolutely black tar stool. No vomiting is present.

I advised the patient to take absolute rest in bed and I told him that hæmorrhage is taking place from the duodenal ulcer and moving will add insult to injury.

I advised him only plain barley water, dab water and nothing solid food by mouth. I adopted the following line of treatment: I injected him Larostidine Histidine (Roche and Co) 5 c.c. intramuscularly every-day for 15 days. (11) Neutralon Belladonna 3i to 3ii to be taken with one tumbler glassful of water twice a day after meals or whenever acidity is felt. Redoxon tablet one mixed with Kepalin tablet (Glaxo) in vitamin K one—morning and evening. For weakness of heart, I injected him every day vitamin B complex (T.C.F.) for 15 days.

After 15 days of this line of treatment the patient was much improved. Hæmorrhage absolutely stopped and acidity also was completely cured. The patient began to pass normal stools, yellowish in colour and he gained much in strength. I gave him soft rice diet and fresh milk twice daily and advised him to continue Neutralon Belladonna for about 6 months. After 3 days of rice diet, I, to my utter surprise, was again called in to see the same patient and found him tossing over his bed, talking incoherently. He got a temp. of about 104.6°F. Kernig's sign was present and rigidity of neck-muscles and retraction of head were markedly present. I was rather puzzled to diagnose the case and thinking it to be cerebral type of malaria, I injected him gr. x Quinine with 12% 20 c.c. Glucose solution. Next day I found his condition no better. Then I was sure of the diagnosis of the case as cerebro-spinal meningitis. I injected him Sodium salt M & B 693, an ampoule and Glucose solution 25% 5 c.c. with 40% Hexamin 5 c.c. intramuscularly. Constant douching the scalp with iced water was advised.

As the patient was semi-conscious and could not take anything by mouth I repeated injection of Sodium salt of M and B 693 in the night also. As lumbar puncture is quite out of question in a mofussil area inhabited mostly by lay ignorant people, M and B was my only resource in this case of meningitis. After 6 injections of M and B 693, the recovery of the case was uneventful and the patient is quite O.K. At present he is living in milk and rice diet.

Discussion.—The patient had a relapse of duodenal ulcer after 10 years due to errors in diet and consequently hæmorrhage in excessive quantity took place. Larostidine Roche, with vitamin C and K and injection of vitamin B complex (T.C.F.) and Neutralon Belladonna checked the hæmorrhage and other complications of the patient. But why he suddenly disclosed all signs and symptoms of meningitis after 3 days of taking rice is still a mystery to me. However M and B 693 injections worked wonder in checking the meningeal symptom of such a weak patient in a short time.

Penicillin in Gonoarthritis

K. D. LAHIRI, M.B., B.S.,
Senior Hon. Medical Officer in Skin and Venereal Department,
Patna Medical College Hospital.

V.D.O.P.D. 1477. S.R.S., Hindu male of 32 years age and is unmarried. Works as a peon in the Patna City Municipality.

Family history.—Nothing in particular except that mother is living and is suffering from filariasis and father died at the age of 58 due to electrocution. Has two brothers and two sisters. All living and are healthy.

Personal history.—*Past*:—Had gonoarthritis 11 years back for which he was treated by irrigation and vaccine. Does not smoke or drink.

Present:—He had fever ushered in with rigor on the 20-9-'45 and on 22-9-'45 developed pain over the right wrist and left shoulder joints. He was so ill that he could not move in bed even for a couple of days. On 25-9-'45 he came to the Skin and V.D. department of the Patna Medical College Hospital, when his temperature was about 100°F. and he was in agony with pain of the wrist joints and had also pain over the left shoulder and unable to move these two joints. There was no urethral discharge and the patient was very toxic and resented P.R. exam. for the examination of prostate. There was no glandular enlargement in the body.

On 25-10-'45. He was given mist. Alkaline oz. 1, 4-hourly and Sulphonamide compound 1.0 gram every 4 hours for 8 days and Scott's dressing applied over shoulder and wrist joints. 26-10-'45 to 27-10-'45. Patient was improving as regards pain and fever.

28-10-'45. Patient sent a message that suddenly his pain in right wrist has increased without increment of the pain of left shoulder and he has again developed fever. I thought the fever may be the Sulphonamide fever and pain could not be accounted for and hence I asked the patient to be brought to me.

29-10-'45. I find the right arm and palm edematous and swollen and the patient's temperature was 102°F. I opened up the bandage and found a tight string round the wrist and the whole hand has gone septic. On enquiry I learnt that the string was a sacred twine and so was not removed. As cellulitis was there and gangrene was threatened I took the case to the Surgeon Dr. S. M. NAWAB to the surgical out-patients department and got him admitted. He was admitted in the ward of the surgical specialist, Rai Bahadur S. B. DUTT. I personally requested the Resident Surgeon, Dr. G. CHATTERJI, to kindly open up for drainage and start Penicillin therapy immediately. Penicillin was started and was continued for five days while the patient was taking Sulphonamide compound also. Penicillin was given at 3 to 4 hours interval and a total dose of 8,00,000 units have been injected with such a wonderful result to the cellulitis and to the arthritis. He was discharged on 30-10-'45 from the surgical specialist's ward and is just attending the out-door.

He has no pain of the right wrist joint as well as of the left shoulder joint and this is directly due to the combined therapy of Sulphonamide and Penicillin. Patient can now move the joints freely and looks apparently cured by Penicillin therapy. The author (1945 a, b, c) has found very encouraging results in gonoarthritis with Penicillin-Sulphonamide therapy. This line of treatment has also been advocated by various other workers in the line.

Acknowledgement.—The author is thankful to the Superintendent, Dr. B. P. VARMA, M.B., Ch.B. (Edin.), F.R.C.S. (Ed.) for his kind permission and to the Surgeons Dr. S. P. DUTT and Dr. S. M. NAWAB for their help and encouragement and to Dr. G. CHATTERJI and Dr. B. N. PRASAD for help to treat and follow up this case.

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A Case of Naga Sore

MOHINI MOHAN RAY, M.B.,

Kotalpur, Bankura.

A WOMAN, aged 35, contracted Naga sore from Dhanbad where some military camps were stationed. The patient was a chronic sufferer from malaria, having a hard spleen reaching almost to the umbilicus. Anemia was marked with swelling of feet and legs, more so on the affected side, though the rest of the body was thin and emaciated.

She came to Kotalpur (Bankura, Bengal) about a month after contracting the sore. There were two ulcers both on her right leg at its lower third, one on the outer side and the other on the fronto-medial aspect over the tibia. There was intense pain, slight fever, anorexia and insomnia from pain.

At Dhanbad, she had been given a few injections of Urea-Stibamine and the wound used to be cleansed with Hydrogen Peroxide and a Copper Sulphate glycerine pigment applied locally; Calcium had been given by mouth.

The ulcer over the tibia was deeper with necrosis of the underlying bone. Edges of both were clean cut and there was a good deal of sloughing in both.

Examination of a smear demonstrated the presence of *B. fusiformis*. As the copper sulphate glycerine application was very painful I tried Sulphanilamide powder for dusting the sores after cleansing with warm saline. Within a few days there was some improvement, slough was less, pain diminished and granulations made their appearance. But after a few days, progress not only stopped but sloughs were seen again with reappearance of pain.

Sulphanilamide dressing was changed and Magnesia cream was prescribed instead. Again there was rapid improvement with diminution of pain, but there was a standstill after a few days, soon followed by a recrudescence with reappearance of slough and pain. Now I changed to Acriflavine lotion and gauze with very similar results.

At last I used B.I.P.P. which produced very satisfactory results within a short time and the wounds were soon covered with healthy granulation tissue. Then they were dressed with gauze soaked in Cod-liver oil, the dressing being changed every three or four days. The sores healed completely, excepting of course the necrosed bone.

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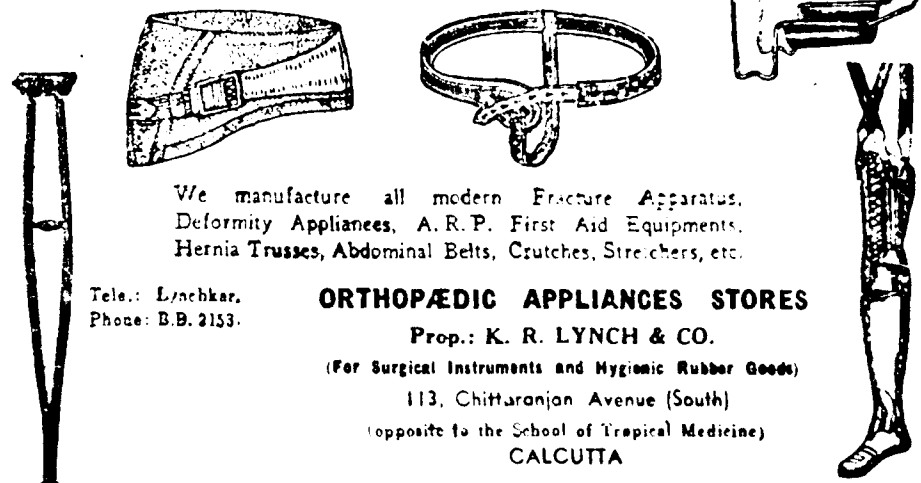
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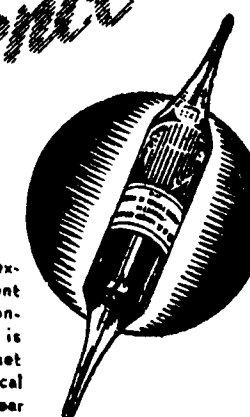
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A Right Step

WE are unable to see any justification whatsoever for the protests organised by a section of the public against the orders of the Minister for Education, Madras, directing the Principals of the Medical and Engineering Colleges to reserve 20% of the total seats available for new entrants to applicants with merit, and distribute the remaining seats on the lines laid down in the previous orders of Government, viz., communal lines. Ever since communalism was allowed to enter the portals of academic courses, merit was given the go-bye and deserving boys and girls who would, if they had been given a chance, have proved themselves ornaments to their respective professions, were driven from post to pillar, resulting in many of them, in sheer desperation, seeking uncongenial courses, to the greatest loss and disadvantage to the country. We have been compelled year after year to voice our most emphatic protest against the perpetration of this gross injustice which would not have been tolerated for a moment in any civilised country, anxious to safeguard the well-being and interests of the community. We were therefore extremely glad that the Minister for Education had realised the injustice involved in the continuance of the existing system and done something to retrieve the position. But we find a deputation waiting on H. E. the Governor and protest meetings organised and held in some places with a view to get the *status quo* restored. If it is the view of those organising these expeditions and meetings that the community or communities which they claim to represent and whose prestige and interests they profess to safeguard will not be able to compete with other sections of the population on equal terms, they are wantonly casting a slur on their communities which, we are sure, the communities themselves as a whole will resent. There are many from among these communities who are occupying great positions of trust and responsibility and enjoying the good-will and absolute confidence of the public, and it is nothing short of impudence to say that these communities cannot hold their own against others unless a premium is placed on the lack of merit. If the protagonists of the *status quo* move base their case on the fact that merit should be no consideration at all in the selection of applicants to these colleges and that anybody and everybody, merely because he

belongs to a particular community, should be given preference as against others who possess merit, we can only say that they are deliberately rendering the greatest disservice not merely to their respective communities but also to the country at large. In any case, therefore, the protest is ill-advised, unworthy and productive of the greatest harm to the country. In the days that lie ahead when great problems have to be tackled to make India a worthier and happier place to live in, and to enable her to attain the place which is rightly her due among the comity of nations, men and women with merit should be sought after and enabled to undertake the task that faces the country. It is therefore the duty of the Minister for Education not only to ignore these ill-advised protests but also to go ahead and reserve a greater percentage of seats in these professional colleges for men and women of merit, so that it can serve as an incentive to every community to put forth its best efforts if it is not to be beaten in the race. We would also suggest to the Minister for Education the advisability of reserving at least a small percentage of the seats in the medical colleges for the sons and daughters of medicos so that the traditions of the family might be maintained unimpaired.

'General Practitioner' Hospitals

COMPARED with England the facilities provided here in India to the people for the proper and effective treatment of diseases, are but a drop in the ocean. But even in such an advanced country as England the facilities now provided for have not given satisfaction even to the medicos themselves. The representative meeting of the British Medical Council passed, in 1944, a resolution urging the importance and the preservation and development of the small general hospitals and recommending to the Council that they give their earnest consideration to making these hospitals staffed by general practitioners the nuclei of one type of the future health service centres. The opinions and recommendations of the sub-committee which was appointed to go into this matter have been approved by the Hospital Committee, and have now been published in the *British Medical Journal*. The resolution of the representative body was interpreted as an expression of the frequently stated need for hospital beds in which patients may remain under the care of their family doctors when suffering from conditions, the treatment of which is within the normal scope of general practice. Though a comprehensive survey of the relation that should subsist between the general practitioner and the hospital services as a whole had not been attempted, the Council has stressed at the outset the fact that in confining its attention to the 'General Practitioner' Hospitals it did not intend to suggest that there is no place for specially qualified general practitioners in the work of the major hospitals, and if little was said about the provision of opportunities for the 'part-time consultant,' it was because it appeared to be scarcely relevant to the question of hospital facilities necessary to enable the general practitioner to function efficiently as a general practitioner.

There is no gainsaying the fact that there is much to be said in favour of a more extensive provision of hospitals in which patients may remain under the care of the general practitioner for the following among many other reasons: such hospitals enable and encourage the general practitioner to

assume fully his proper professional responsibilities; they afford him valuable opportunities for the observation of patients; they place at his disposal more complete facilities for the carrying out of diagnostic tests than are otherwise readily available to him; and they make continuity of treatment possible in cases in which removal to hospital is necessary because of domestic conditions—such as lack of skilled nursing—and not because treatment outside the scope of general practice is required. In the Association's publication "A General Medical Service for the Nation," is found the following statement:

"It commonly happens to-day that, for a social reason such as unsatisfactory home surroundings, a patient is admitted to hospital for a condition which in a more fortunately circumstanced patient would be treated at home by the patient's own doctor. It is contrary to the interest of the patient and damaging to the efficiency of general practice, if social conditions lead to a discontinuity of medical treatment."

The existence of a social condition, the lack of proper facilities at home, whereby it becomes necessary to remove a patient to the hospital and place him in charge of a doctor who has had no knowledge of the personal or family history of the patient may be unfortunate, but such a social condition unfortunately does exist, and it becomes therefore necessary to provide for the treatment of the patient in the hospital by his or her own doctor. It is in this view that Dr. A. TREVOR JONES, Prof. J. A. NIXON and Prof. R. M. F. PICKEN say in their survey of the Hospitals in South Wales and Monmouthshire:

"The main advantage of the system whereby general practitioners serve on the staff of hospitals is the extremely close and continuous supervision over patients during all stages of their illness. When the general practitioner is responsible both in and out of the hospital, the supervision is easy and it is in conformity with the patient's natural wish to have his own family doctor in charge of his case during his stay in the hospital ward. Continuity of treatment is a desirable object to aim at and, in so far as the small local hospitals achieve this object by reason of the fact that the patient's family doctor is also in charge of the hospital bed, it is an argument in favour of their existence and of their development within certain defined limits. The question will become of increasing importance if the tendency to hospitalise less seriously ill patients develops."

The opportunities provided by collaboration between local practitioners and the educative value of regular contact with visiting consultants are not the least of the benefits to be derived from the 'general practitioner' hospitals. In "A General Medical Service for the Nation" it is pointed out that an association with hospital by reason of the contacts it affords with fellow practitioners and the team work it involves, stimulates the general practitioner to a higher standard of efficiency, with consequent benefit to the community. The Volims Committee, whose report has recently been published, refers to the value of the local small hospital as a "meeting ground for men and women engaged in general practice for the exchange of ideas and for the comparison of the methods of treatment. This aspect is emphasised in the Report of the Medical Planning Commission in England. "Some Aspects of the Post-War Hospital Problems," issued by the Joint Committee of King Edward's Hospital Fund for London and the Voluntary Hospitals Committee for London, also says:

"Unless the general practitioner is provided with a local hospital where he can himself attend his patient, the quality of the whole medical service will suffer, and there will occur a divorce between the work of the general practitioner and of the hospital services. Arrangements such as those advocated by the Goodenough

Committee and other bodies for those taking part in the work of the general and special hospitals by means of clinical assistantships, etc., while highly desirable themselves, are not enough. Close association with the visiting consultant in the case of his patient at the cottage hospital will be of real benefit to doctor and patient alike."

For these and other reasons the Report says that the rural hospitals should be made "general practitioner" hospitals and that in urban areas where there are large hospitals, special blocks should be provided for them. The 'general practitioner' hospital, the report further says, should be open to every general practitioner in the locality which it serves and it should not have a restricted staff; that dental practitioners also should have access to the hospital for the performance of dental operation in cases in which a short period of post-operative care in hospital is desirable; that there should be a Committee composed of all the medical and dental practitioners attached to the hospital; that various types of cases should be admitted; that provision should be made for admitting maternity cases where there are inadequate facilities for such patients to be attended by their own doctors in special institutions; that some beds should be provided also for chronic sick or incurable patients, admitted either from their homes or from larger hospitals when there is no advantage to be gained from continued treatment there, as the experience and outlook of the general practitioner make him more acceptable than the junior resident to patients of this type; that post-operative and other convalescent cases, on discharge from larger hospitals, should be admitted for a period under the care of their own doctors, if not requiring treatment at special rehabilitation centres; that the activities of these hospitals should include also out-patient consultations with visiting consultants; that the routine performance of major surgical operations should generally be excluded from their scope though it may, at least for a time, continue to be necessary for major surgical emergencies to be treated by qualified men resident locally; that a small laboratory for simpler pathological examination should be attached to it to enable the general practitioner, if he so wishes, to be at liberty to conduct his own pathologic examination with the advice of the expert technician who should be provided for each; that similarly the services of a trained radiographer, supervised by a visiting radiologist, should be made available; that for the purpose of training of nurses the general practitioner hospital should be grouped with larger ones; that the services of consultants should be regularly available; and so on and so forth. Finally the Report says that for various reasons, and particularly because of the present building restrictions and the inadequate supply of consultants and specialists, progress must of necessity, be only gradual, but that their scheme must form the basis for future planning.

What is the position in India? The Bhore Committee says: "If it were possible to evaluate the loss, which this country annually suffers through the avoidable waste of valuable human material and the lowering of human efficiency through malnutrition and human morbidity, we feel that the result would be so startling that the whole country would be aroused and would not rest until a radical change had been brought about." To bring about that radical change they suggest: "When the necessity arises for medical attention for the individual, there should be an adequate health service to turn to, from which no question of lack of means should cut him off. The ideal to be aimed at in a National Health Service cannot

be more clearly described than in the words of the Ministry of Health in the United Kingdom in setting out its proposals for such a service. 'The new service,' it says, 'is designed to provide for every one, who wishes to use it, a full range of health care. No one will be compelled to use it. Those who prefer to make their own arrangements for medical attention must be free to do so. But to all who use the service, it must offer, as and when required, the care of a *family doctor*, the skill of a consultant, laboratory service, treatment in hospital, the advice and treatment available in specialised clinics (maternity and child-welfare centres, tuberculosis dispensaries and the like), dental and ophthalmic treatment, drugs and surgical appliances, midwifery, home nursing and all other services essential to health. Moreover all these branches of medical care must be so planned and related to one another that every one who uses the new service is assured of ready access to whichever of its branches he or she needs.'" Planning that had so far been made in India falls far short of this ideal. The recommendations made in the 'General Practitioner' Hospitals Report, every one will admit, are the best means to achieve the ideal the Ministry for Health in Great Britain has aimed at. We would therefore earnestly appeal to the governments in this country to bestir themselves and take effective steps to have their future policy shaped on these lines with such alterations as may be called for to suit the conditions that exist now in the country.

Gleanings From Medical Press

MEDICINE and THERAPEUTICS

Hypertension.—There is no specific medication curative of hypertension. Nor are there any specific curative surgical procedures. Hypertension per se is not a disease but a symptom or biologic response to continued and excessive arteriolar stimulation. The sources of such stimulation or irritation differ in each and every instance of the disorder. Therefore no 2 cases of hypertensive arterial disease should be treated identically; curative therapy invariably is dependent on eradication or amelioration of etiologic factors, and these are extremely variable. It can be predicted that no one will ever find the cause of hypertension, simply because there are many causes. Similarly, it is highly improbable that any "specific" curative therapy, generally applicable to all or most cases of the disease, will be discovered in the future.

This discussion does not imply that there are no useful drugs effective in the treatment of hypertensive disease. There are many medicaments which have great usefulness in the control of hypertension, each with specific and limited indications. These drugs may be classified under several headings:

1. Sedatives, to reduce central nervous system tension.
2. Vascular antispasmodics, including
 - (a) Soluble nitrites (amyl nitrite, sodium nitrite) and soluble organic nitrates (gly-

cerol trinitrate, erythrol tetranitrate, manitol hexanitrate).

(b) Insoluble nitrates (bismuth subnitrate).

(c) Potassium thiocyanate.

(d) Certain tissue extracts.

3. Cardiac drugs:

(a) Digitalis and allied substances.

(b) Theophylline and allied derivatives.

4. Antianemia medications.

Certain surgical procedures, particularly the Smithwick operation, designed to sever the vasomotor trunks and thus reduce the vascular hypertonia, are effective in certain instances. However, these operations admittedly are only palliative. The neuro-surgical approach fails in that it does not take cognizance of the primary etiologic factors responsible for the disease; it merely destroys one of several physiologic mechanisms concerned with the maintenance of vascular tone. A few, but only a few, instances of hypertension are secondary to unilateral renal disease, and a still smaller number are due to tumors of the adrenal gland. Surgical, like medicinal, therapy must be guided by etiologic analysis.—J.A.M.A.

Factors in ulcer formation.—Clinical studies and impressions appear to indicate that the pituitary-thyroid-gonad mechanism plays a role in human peptic ulcer. It is well known that peptic ulcer is comparatively rare in women, but when it does

occur, it is ameliorated by pregnancy and aggravated by the menopause. Also, women with ulcer show a high incidence of endocrinopathies. While ulcer in the adult is approximately 5 to 10 times as frequent in men as it is in women, in children before puberty its occurrence is equal in both sexes though rare. Of interest also is the fact that extracts prepared from urine of both pregnant and normal women have a greater prophylactic, therapeutic and "immunizing" effect on the experimental Mann-Williamson ulcer in dogs, than extracts of urine from normal men.

It is therefore probable that in the female the monthly reproductive cycle and the increased glandular function during active sex life and pregnancies (i.e., the chemical interplay between the pituitary and the ovaries throughout the active menstrual life of the female), augment the production of ulcer "protective factors" and thus serve to prevent or benefit "peptic" ulcer. The absence of these in the male could result in the lesser protection against ulcer. Abnormalities in the glandular function in both the male and the female may affect the protective factors and be responsible for ulcer formation.—Kaulbersz, J.; Patterson, T. L.; Sandwells, D. J.; and Saltzstein, H. C.; Relation of endocrine glands to gastric secretory depressant in urine. *Science* 102: 530-531, November 23, 1945.

—*Na. Med. Bull.*

Sodium balance in edema.—The capillary wall is freely permeable to sodium in either direction, and edema is not due to a change in this permeability. Therefore the phrase "tissue retention of sodium" must not be taken too literally. It really means an excess of the normal extravascular or interstitial fluid, which is essentially a low protein ultrafiltrate of the plasma containing all the diffusible electrolytes and nonelectrolytes in approximately the same concentration as the plasma, except for the small differences due to the Donnan membrane equilibrium. The chief electrolyte cation in extracellular fluids is sodium; the chief anions are chloride and bicarbonate.

Edema is brought about by a disturbance in the balance of forces favoring filtration and reabsorption respectively. When sodium, as sodium chloride, is ingested it is absorbed as an isotonic solution, increases the plasma volume and is filtered by the capillary blood pressure into the interstitial spaces. If the sum of factors in the reabsorption of tissue fluids (plasma protein osmotic pressure, tissue pressure and lymphatic drainage) is less potent than the sum of factors in filtration (capillary blood pressure, amount of protein in interstitial fluid, local tissue metabolism), an excess of saline solution will remain in the tissue spaces. If, on the other hand, sodium is restricted in the diet or its normal absorption from the lumen of the renal tubule is diminished by the action of diuretic drugs, urinary loss of sodium (and chloride and water) soon reverses the stream and more interstitial fluid

moves back into vascular channels until a new equilibrium is established. If sodium chloride could be given in large amounts with little water so as to render the body fluids high in salt concentration, diuresis would undoubtedly result even in an edematous patient. In other words, the tissues in edema do not retain sodium but simply contain an excess of interstitial fluid with their normal sodium concentration. Some tissues, like bone and certain types of collagen, contain sodium in bound form, not freely exchanging with the sodium of interstitial fluid although participating in the uptake of radioactive sodium.

The exchange of muscle cell potassium for sodium which is seen after severe trauma, burns, severe dehydration with loss of muscle cell water and its contained potassium or after excessive administration of desoxycorticosterone in animals (and probably in man) is not a true tissue retention of sodium and is not to be identified with edema in the ordinary sense, since water content of the affected muscle cell is not increased. On the other hand, the decreased excretion of sodium which is the result of desoxycorticosterone effect on the renal tubular reabsorption reflects itself in an increased plasma volume, increased blood pressure and ultimately increased filtration of plasma water and its contained electrolytes into interstitial spaces, producing edema.

On the whole, therefore, the concept of the permeability of the capillary wall is best omitted in the consideration of ordinary cardiac or renal edema, since changes in permeability to electrolytes and water do not play a significant role in this type of edema.—*J.A.M.A.*

Serum phosphorus in hyperparathyroidism.—The changes in calcium and phosphorus metabolism characteristic of hyperparathyroidism are increased serum calcium, increased calcium and phosphorus excretion and decreased serum phosphorus.

Jaffe suggested that the parathyroid hormone in some way mobilizes the insoluble calcium salts of the bone and thereby raises the serum calcium level. Since calcium and phosphate ions form a slightly soluble salt, the increased serum calcium would drive the phosphate ions from the serum into the bone and thereby lower the concentration of the phosphorus in the serum. The hypothesis is supported by the work of several recent investigators quoted by Pope and Aub, who found indications that the parathyroid hormone acts primarily to mobilize calcium from the bone.

On the other hand, Albright and Ellsworth suggested that the primary action of the parathyroid hormone is to increase renal excretion of phosphorus. This is supported by the Harrisons, who showed that parathyroid hormone decreases renal tubular reabsorption of the phosphate ion. This results in a low serum phosphorus, which allows withdrawal of calcium from the bone to raise the serum calcium level. The increased serum

calcium level then leads to increased urinary excretion of calcium.

These two hypotheses may eventually be reconciled. It is probable that both mobilization of calcium from the bone and increased renal excretion of phosphate ion are affected by the direct action of parathyroid hormone.

The omission of veal from diet lists must be presumed to be without significance, since there is no essential difference in the relative nutritive value of beef and veal. A comparison of their relative composition, such as that given in a recent publication of the Department of Agriculture, shows that they are practically identical, the only difference being a slightly greater concentration of niacin and riboflavin in veal than in beef.—*J.A.M.A.*

Cheyne-Stokes respiration, a waxing and waning type of breathing in which the respiration is at first slow and shallow, then gradually builds up to a maximum in depth and rate only to subside again and finally cease for a short time, is occasionally found in normal adults during sleep. The most common clinical conditions in which it is found are advanced renal and heart disease, asthma and increased intracranial pressure. The pressure of orthopnea in this case (as adjudged by the need of two pillows when lying down) suggests the pressure of cardiac insufficiency.

Since the therapy is decided by the chief cause of the Cheyne-Stokes respiration, a careful evaluation of the cardiac and renal status is indicated. Treatment directed toward an abnormality of cardiorenal systems probably will have a secondary beneficial effect on the respiration. Histamine has a definite tendency to produce a fall in blood pressure and thus is not indicated for treatment of Cheyne-Stokes respiration. Occasional rebreathing or the use of a 7 per cent. mixture of carbon dioxide in oxygen, may help to stabilize the respiratory rhythm.—*J.A.M.A.*

Skin reaction to mepacrine.—It has been recognized for some years that the taking of mepacrine provokes a variety of skin reaction in certain individuals who have an idiosyncrasy to the drug. These reactions may take the form of erythematous or eczematous—often weeping and sometimes infective eruption, but the most distinctive reaction is that resembling lichen planus and commonly assuming a hypertrophic form. The condition may be extraordinarily widespread, involving head and face as well as any or all other parts of the body, including mucous membranes. The head and face are rarely involved in ordinary lichen planus. The condition may be of an extreme hypertrophic and wasting type so that the patient looks like a tree trunk, but the colour and infiltration and buccal lesions suggest the diagnosis of lichen. All the hair may fall off and scarring may result. Ulceration may also occur, especially about the lower legs, and itching may be marked.

While the condition is of great severity

and the appearance of the patient is alarming, there is not any serious constitutional disturbance. The affection runs a prolonged course of several months and recovery seems to be complete, apart from the possibility of scarring. No specific remedies are recommended as yet, treatment being along the lines of that employed in lichen planus. Fractional doses of X-rays are of value in the later stages.—*B.M.J.*

The treatment of amebiasis.—J. Gerdasy, M.D., Elizabeth, N. J.

The *Entamoeba histolytica* is the most frequently encountered pathogenic intestinal protozoon infesting the human organism. Variability of signs and symptoms is a characteristic of amebic infestation. This is especially true in chronic cases which make up the larger proportion encountered in practice and which are frequently undiagnosed. The term "amebic dysentery" is a misnomer since enteric symptoms are frequently absent.

Types:—Three well defined groups are recognized:

1. *Acute fulminating amebic colitis*, epidemic and virulent in form, with a high mortality. Usually the source of infection is definitely traceable.

2. *Chronic amebic colitis*: An active protracted infectious process, usually of undetermined origin. During its prolonged clinical course, attacks of digestive disorder, bloody diarrhoea, loss of weight, anaemia and general health impairment will alternate with irregular periods of quiescence. During the latent period, the disease continues to progress and cause serious complications, such as liver abscesses.

3. A group of *atypical cases* not showing enteric symptoms. These occur with sufficient frequency to warrant brief description. The following syndromes may suggest this type of amebiasis: inconstant pain in various parts of the abdomen; symptoms of pseudo-appendicitis, severe intractable lumbar or sacral backache, pains in the joints and muscles, low grade daily fever associated with marked physical fatigue (as in tuberculosis), secondary anaemia not responding to iron or unexplained massive hemorrhage from the bowel. In these cases, diagnosis depends on finding the pathogenic amebas in the stool or by a therapeutic test.

Some explanation for the inconstant manifestations of this disease may be obtained by reviewing certain etiologic factors:

1. The location of the lesion in the colon and the extent of the involvement varies the clinical picture. For example, amebic infection of the terminal colon and rectum is usually associated with severe diarrhoea and the passage of blood and mucus.

2. The more concentrated the invasion of the host, the more virulent the disease.

3. A superimposed bacterial infection may complicate and obscure the infection, giving a syndrome suggestive of chronic ulcerative colitis.

4. The reaction of immunity of the human host to the invasion of the ameba, in

this as in other infectious processes, determines the severity of the disease.

5. The changes of morphology which the organisms undergo during their life cycle from the stage of motile trophozoite activity and tissue invasion to the stage of encystment and latency, influence the picture.

DIAGNOSIS:—The history of every patient should include a note relating to service or travel in the tropics, especially in the Near East, India and China. This will contribute much toward a diagnosis of this as well as other protozoal infestations.

It should be the rule to investigate thoroughly every case of recurring attacks of diarrhoea for protozoal infestation irrespective of the signs and symptoms.

Absolute diagnosis depends on the microscopic finding of *amoeba histolytica* in the feces or tissues. If the stool is solid, search is made for encysted forms. Motile trophozoites are found in liquid stools or may be searched for after a saline cathartic or a high tepid saline enema. The material is selected after the bulk of the stool or enema has been passed, preferably close to the laboratory to insure a warm specimen for examination. A great deal of uncertainty may be overcome by routine use of the sigmoidoscope in all suspected cases. This may reveal the characteristic ulcerations of the rectal mucous from which scrapings may be obtained or biopsy of the margin of the ulcer may be used for histologic examination. A warm specimen of loose stool or bloody mucus transferred directly under a microscope may reveal motile amebas without the necessity of a warm stage microscope. The more refined, diagnostic laboratory techniques are outside the scope of this paper.

CLINICAL MANAGEMENT:—The first problem is the earliest possible eradication of the intestinal infection to prevent extensive local bowel or systemic injury. Next problem is the elimination of cysts and trophozoites which have already invaded organs such as the liver, lungs or spleen. It is the destruction of the viable organism in the extraenteric structures which makes the problem of amebiasis so difficult and necessitates repeated courses of treatment to prevent relapses.

The drugs in common use in the treatment of amebiasis are of three types: (1) Alkaloids such as emetin hydrochlorid or emetin bismuth iodide for the acute condition; (2) the organic arsenicals such as carbarsone, Stovarsol, Treparsol, and (3) the iodine compounds such as diodoquin, viotrom and chinofon (Yatren). When many bowel ulcerations are present the iodine compounds are safer than the arsenicals; on the other hand the arsenicals are more efficacious for the encysted or tissue invaders.

Treatment of acute amebic colitis with fever, diarrhoea and prostration includes bed rest and supportive measures, such as intravenous saline-glucose solutions to overcome dehydration. In severe cases, blood plasma (in adequate doses) is indicated.

Hot compresses to the abdomen, a non-residue diet and full doses of tincture of belladonna will relieve intestinal spasm. If there is much pain the bowel is further sedated with deodorated tincture of opium, 10 minims every 4 hours by mouth or *via* the rectum as an opium suppository. Emetin hydrochlorid is given intramuscularly twice daily in $\frac{1}{2}$ grain doses for 5 or 6 days. Simultaneously capsules of viotrom 0.25 grains are administered 3 times a day for 10 days. Enemas of chinofon 4 grams to 500 cc of water are also given, repeated daily for 7 to 10 days, or until the acute symptoms subside. Precede each retention enema with a cleansing enema of 2 per cent sodium bicarbonate in a pint of warm water. A warning about the use of emetin is necessary, since effective doses lie within toxic range. Emetin Bismuth Iodid 0.2 gms. in a hard gelatine capsule, by mouth, once daily for 12 days is recommended. However, it is likewise very toxic and bed rest is essential during its administration.

CHRONIC AMEBIASIS:—Hospitalization and bed rest are adjuncts to the treatment. A low residue, high caloric, high protein diet is prescribed as well as a brief course of emetin hydrochlorid, 1 grain daily intramuscularly for 6 days. If only cysts are found in the stool or tissues a course of carbarsone 0.25 grams t.i.d. is given for 10 days. One week later examine stools for cysts on three successive days. Repeat the course using diodoquin 0.6 grams by mouth for 7 days t.i.d. The treatment is repeated if relapses occur or amebas are found. Since the margin between the toxic and therapeutic doses of these drugs is small, the administration must be discontinued at once should toxic signs and symptoms occur. A rest of 2 or 3 weeks between successive courses is advised. Vitamin therapy is indicated here as in all severe cases of diarrhoea. Proper isolation should also be practised.

Persistence in adequate and properly timed administration of these drugs is the keystone to successful treatment.

J. Am. Med. Soc. New Jersey.

Syphilis of the spinal cord—The following clinical syndromes are due to syphilitic involvement of the cord:

1. Large vascular accidents involving the spinal arteries are an unusual condition. The best known example is thrombosis of the anterior spinal artery. The signs are a segmental loss of pain, temperature and manifestations of injury to the anterior horn cells, with varying amounts of damage to the ascending spinothalamic and descending pyramidal tracts. The acute paraplegias of syphilis are due to such vascular occlusions, and they may be followed by a partial return of function.

2. A diffuse meningomyelitis, often associated with small vascular occlusions, is also seen early in the disease. This form is a more common variety. Pain, due to root irritation, is a frequent feature. The upper

thoracic cord is most often affected, and girdle pains are characteristic.

3. Erb's spastic paraplegia is a late form of syphilitic myelitis. The pyramidal tracts are most severely damaged, with only mild injury to other parts of the cord. Besides the spastic paraplegia, there are frequently bladder disturbances.

4. Progressive muscular atrophy is also a late complication. Here the anterior horn cells are the principal site of injury. The lower cervical cord is the most frequent location for this variety of spinal cord syphilis. Muscular atrophy and fasciculations are the usual clinical findings. Amyotrophic lateral sclerosis of syphilitic origin is a closely associated condition in which there is injury to the pyramidal tracts as well as to the anterior horn cells.

5. Gummata may occur after the first year of infection. The signs are those of cord neoplasia. Any portion of the cord or cauda equina may be the site of the granuloma. The results of therapy are usually good. However, there is one special form of massive, chronic gummatus process which is limited to the cervical region, known as hypertrophic cervical pachymeningitis. Symptoms and signs may be those of a mass, but they may simulate those of syringomyelia or amyotrophic lateral sclerosis. It is very resistant to treatment.

6. Tabes dorsalis is a process in which the sensory roots alone are damaged, resulting in ataxia, loss of reflexes, bladder disturbances, characteristic gait, and various forms of pain. It is usually a late complication of syphilis. One or several cranial nerves may be injured concurrently. While the site of the lesion is clear, its mode of production is not. It is not unusual to find the blood and spinal fluid serologic tests for syphilis negative. Author's Abstract. Arthur B. King, M.D., Am. J. Syph., Gonorr. & Ven. Dis., 21:336-377, 1937, New Jersey.

Toxicity of mercurial diuretics.—Neptal is the hydrony mercury-propanilamide of o-carboxyphenoxy acetic acid. Acute toxic reactions and sudden death following the injection of mercurial diuretics are well known, though they are very infrequent in relation to the large quantities of these drugs which are now used in cardiac therapeutics. Death has more commonly followed administration intravenously than intramuscularly or per rectum. Fatal accidents are probably relatively more common in patients with nephritis than in those with heart disease. Death may nevertheless occur with the first injection and from this it would appear that the reactions are not allergic or due to sensitization. They are the result of intolerance or abnormal susceptibility, to the pharmacological effects of the drug.

The exact mechanism of death is probably the toxic action of the mercurial diuretic on the cardiac muscle, with terminal ventricular fibrillation and syncope.—B.M.

Sublingual administration of penicillin.—Few practitioners are aware of the availability of the sublingual space for quick

absorption into the blood stream of penicillin-sodium in solution, by way of the ranine veins under the tongue. For some months I have been using this area to promote a rapid blood level of penicillin when oral medication was indicated. Systemic absorption from the sublingual space nearly equals that from a subcutaneous injection. Indeed, some authorities believe that it is even more rapid.

Clinically judged, results by this route have been prompt, uncomplicated and curative; only one patient so far, after a ten-day period of administration, has complained of tender mouth lasting about a day. There has been no bowel disturbance such as frequently occurs when tablets of penicillin-calcium are administered by mouth. And, in correspondingly effective amounts, penicillin-sodium costs the patient only a third as much as penicillin-calcium.

Ten thousand units of penicillin-sodium in the sublingual space hourly are more efficient than 25,000 units of penicillin-calcium given in tablet form by mouth every two hours. This is so because of more rapid and direct absorption from the sublingual space into the blood stream.

It was impossible during this time to secure reliable laboratory estimates of blood levels. Two commercial laboratories were asked as well as the Pathologic Department of a large hospital in my vicinity. Shortage of assistants, pressure of routine work and a rather complicated method of determination, without much previous experience, have been the reasons advanced by them for not being able to perform these tests.

ADMINISTRATION.—The blonde, or light yellow penicillin-sodium is more soluble and less bitter than the brunette or darker varieties and is preferred for sublingual use although either may be given.

Basic formula is 100,000 units of penicillin-sodium, 5 cubic centimeters isotonic solution of sodium chloride and one drop of spirit of peppermint which does not contain enough alcohol to precipitate the penicillin and does give a cooling, pleasant sensation to the somewhat bitter drug.

A usual prescription for 40 doses of 10,000 units each is:—

R Penicillin Sodium ... 400,000 units
(light yellow)
Liq. Sod. Chloride, Isoton. 20 cc.
Menth. Pip. ... 4 drops—m.

Place 4 drops under the tongue as directed every 2 to 4 hours. (Keep refrigerated.)

This gives the patient 10,000 units each ten drops equal to half a cubic centimeter. This dosage may be given hourly or every two hours in acute conditions. The interval may be extended to three hours when symptoms are less pressing.

To the patient the following oral directions are given:

1. Roll the tip of the tongue toward the roof of the mouth exposing the sublingual space behind the lower teeth.

2. Put the prescribed number of drops in the sublingual space for quick

3. Return the tongue slowly to its normal position and refrain from swallowing for one minute.

As sublingual medication has nothing to do with stomach secretions dosage may be carried out anytime without regard to food intake.

SUMMARY.—Penicillin may be given effectively and inexpensively by the sublingual route. Basic formula is 100,000 units of penicillin-sodium with 5 c.c. of isotonic sodium chloride and a drop of spirits of peppermint. Ten drops are placed under tongue every hour or two.—*Jo. Med. Sc. New Jersey.*

Protein synthesis.—The cells of the body synthesize the protein peculiar to their structure and function provided there is a suitable substrate from which to build. Caseinogen is produced nowhere in nature but in the cells of the mammary gland. The main site for the synthesis of the proteins of the blood plasma is the liver, and Whipple and his colleagues believe that most of the protein synthesis within the body goes on in this organ. The liver may produce those fundamental protein units for body nutrition and use, which are then elaborated by the cells of the body to suit their particular requirements.

The rate of synthesis of protein by the cells is dependent on their special role and on their natural response to growth and to the other requirements of the body—for example, those concerned with reproduction—and on supply in optimal amount of those amino-acids which the body cannot synthesize. The work of Whipple and his colleagues shows that there is a dynamic equilibrium between plasma protein and cell protein, the protein flow from cell to plasma and from plasma to cell depending on prevailing conditions. Reserve stores of protein out of which the body can produce haemoglobin in anaemia due to removal of blood or plasma protein in hypo-proteinemia, can be demonstrated in dog. Fibrinogen is the most labile of the plasma proteins; liver injury may reduce it and acute infection or tissue injury may raise it. Diet may also modify its level. Protein production in the anaemic and plasma-depleted dog occurs from almost any protein or mixture of essential amino-acids. Whipple and his colleagues consider that it may be found possible to influence the ratio of plasma proteins to haemoglobin. They found in dogs that the amino-acids necessary for growth supply the material out of which abundant plasma protein and haemoglobin can be produced and that the presence of methionine in the mixture tended to favour haemoglobin production, and cystine plasma protein formation.

Haemoglobin cannot apparently contribute to the body protein pool except when the red cell is broken up. Haemoglobin is then saved, supplemented, and recast into new protein, depending on the body's needs. Whipple et al have shown that much of this rescued haemoglobin or globin may contribute

to the building of plasma proteins. In their view then, the circulating plasma protein is the medium of exchange between the protein pool and the haemoglobin, new plasma, or cell protein.

The acidic and basic properties of amino-acids are dependent on the relative proportions of carboxy groups to amino-groups. Thus the basicity of proteins is conditioned by the proportion of the diamino-acids—lysine, histidine, and arginine. Lysine must be provided in the proteins of the diet, and there is evidence to suggest that the same is also probably true of histidine and arginine. The basic proteins are the protamines and histones. The nucleoproteins are formed of a phosphorylated organic acid, nucleic acid with a basic protein which is usually a histone or protamine. As a whole the nucleoproteins have acidic characters although their protein component is essentially basic. Proteins of an acidic character are the phosphoproteins, which have a high content of dicarboxylic groupings.

Protamines form the bulk of the proteins of the nucleoproteins of ripe sperm cells and ova. Histones are not so strongly basic as the protamines. They occur in unripe germ cells, in lymphoid tissues, and as globin, the histone of haemoglobin. The most important phosphoproteins are the caseinogen of milk and the vitelin of egg yolk. Their biological function is to provide food material for the developing embryo or the growing young. The active character of caseinogen is due to its content glutamic acid. The synthesis of protamines and phosphoproteins is thus mainly located in those organs which are concerned with reproduction.

Nervous regulation of acid-base balance.

Changes in hydrogen ion concentration affect the excitability of the nervous system. An increase in acidity reduces excitability, while a decrease enhances excitability. These effects can be demonstrated on isolated nerve; in an experimental animal the reflex time for the knee jerk can be shown to be increased by an injection of ammonium chloride and decreased by sodium bicarbonate. The convulsive activity of certain drugs is modified also, in the sense stated above, by acid or alkali. Hyper-ventilation tetany, where a shift towards the alkaline side of neutrality is caused by the 'blowing off' of carbon dioxide, is a striking example of tetany associated with various types of alkalosis.

It is suggested that these effects of altered acid-base balance may be caused by changes in the ionic balance, particularly in the proportion of ionized to unionized calcium. An increase in the concentration of calcium ions causes a reduction in excitability and vice versa. In alkalosis the ratio of ionized to unionized calcium may diminish without any reduction in the total calcium concentration.

In the body there are three lines of defence against disturbance in the acid-base equilibrium; the buffer reactions of the blood, the regulation of breathing, and the

ability of the kidney to adjust the reaction of the urine. The nervous system is, of course, concerned with the second of these, the respiratory centre being acted upon directly by the blood and receiving impulses from the chemoreceptors of the carotid and aortic bodies. For instance, a disturbance of the acid-base balance towards the acid side is compensated by increased respiratory activity, which reduces the carbon dioxide tension of the blood.—*B.M.J.*

Hodgkin's Disease.—The treatment of choice for Hodgkin's disease is irradiation. Relatively small doses are given to the involved glands. This usually causes a regression in size of the nodes and an improvement in general well-being. Irradiation is repeated when the enlarged nodes recur or

when previously uninvolved nodes become involved. There comes a time when irradiation no longer causes regression of nodes and improvement in the general condition: general deterioration and fatality ensue. The usual course of Hodgkin's disease, well managed by irradiation, is three to five years, in some instances shorter, and in the occasional case palliation may obtain for ten years or even longer. According to some, when a patient is first seen presenting only one group of nodes involved, better results are secured if that group is resected and X-rays are given to the area postoperatively, also to the other nodes in the adjacent regions. If more than one large group of nodes is involved when the patient is first seen, the question of excision does not arise.—*J.A.M.A.*

SURGERY

Surgical complications of ascariasis.—In this report Milwidsky is concerned with the surgical complications to which ascariasis may give rise. Discussing ileus due to ascariasis, he reviews 10 cases of his own and shows that there are five different types: (1) mechanical obstruction by worm convolutes, (2) a paralytic type of ileus, (3) a spastic ileus, (4) invagination by ascariasis, and (5) volvulus by ascariasis. Milwidsky has observed perforations of the bowel, postoperative disturbances due to ascariasis, appendicitis and diverticulitis as well as ascariasis of the stomach of duodenum, bile ducts and liver. The 17 cases mentioned in this paper were encountered in the course of six years. The most effective method to forestall surgical complications of ascariasis is its prevention by an antiparasitic prophylaxis and early anthelmintic treatment if an infestation has taken place. As soon as the acute abdominal symptoms appear, anthelmintic treatment may be dangerous. At that stage surgical intervention becomes imperative.—*J.A.M.A.*

Occlusive dressing for tropical ulcer.—Desert sores and similar conditions respond best to an occlusive method of treatment. An adhesive plaster of parts dressing has proved successful in the treatment of this condition.

The dressing consists of a powder rubbed into the mesh of an open weave bandage, as it would be for a prepared plaster of Paris bandage. The powder is composed of plaster of Paris 75 per cent. and tragacanth powder 25 per cent. to which is added acriflavine powder to give a strength 1:1,000. Other antiseptics such as gentian violet or sulfanilamide may be used. The powder must be thoroughly mixed and finely ground.

Indolent ulcers, which are frequently met, respond well to an application of silver nitrate and sealing with the patch.

The dressing has been used with success in the early vesicular stage of desert sores, early treatment of boils, in treatment of impetigo, and particularly in abrasions and minor wounds of the head and face. The

dressing has been applied over small lacerations, and in all cases rapid healing has been obtained.

This method of treatment is extremely economical. It is comfortable, does not restrict movement, and there is no irritation of the skin.—Hammond, A. G.: Occlusive dressing; the almaza patch. *East African M. J.* 22: 241-242, July 1945.—*Nu. Med. Bull.*

Intravenous alcohol anaesthesia as used in Russia.—Verkhovskaya (*Am. Rev. Soviet Med.* 1945), pointing out that alcohol, like chloroform and ether, belongs to those poisons which act by paralysis of the central nervous system, emphasizes that alcohol has the advantage over chloroform and ether that it is almost completely metabolized to carbon dioxide and water. In narcosis produced by administration of alcohol sleep begins quickly and analgesia soon follows. The toxic dose of alcohol is 7.7 cc. per kilogram of body weight. The dose producing analgesia and sleep varies from 1.5 to 3.0 cc. Thus the difference between the toxic and the therapeutic dose is 4.7 to 5.2 cc. The margin of safety is considerably less for ether and chloroform. The difference between the toxic and therapeutic blood concentrations is 1.5 mg. for chloroform and 4 mg. for ether per hundred centimeters.

Two cc. of a one percent. solution of morphine is given to the fasting patient half an hour before the operation. Intravenous administration of one part of 95 percent. alcohol with two parts of 5 per cent. glucose solution is started on the operating table. The average dose for complete narcosis is 2 to 2.5 cc. per kilogram of body weight. Thus a person weighing 60 Kg. requires 120 cc. of alcohol and 240 cc. of 5 per cent. glucose solution. The mixture is prepared on the day of operation. The glucose solution is sterilized and the required amount of alcohol is added just before use. The standard apparatus for blood transfusion is utilized for the infusion, which is given slowly for fifteen to twenty minutes. When sleep sets in, the rubber tube is clamped to stop the infusion. The clamp is released if signs of awakening

appear before the operation is finished, and the drip is resumed. When deep anaesthesia has set in, the vein is flushed with 30 to 40 cc. of isotonic solution of sodium chloride to prevent possible thrombophlebitis. Sleep begins following the introduction of 40 to 60 cc. of alcohol. The stage of excitement is passed without difficulty and sleep sets in quickly. The conjunctivas, face and neck become red; the pupils contract and stop reacting to light. Respirations become even and stertor and the pulse is of good volume. Sleep lasts from two to five hours. On awakening the patient is irrational and requires special observation. Thirty operations have been performed at an evacuation hospital under intravenous alcohol anaesthesia. Complications noted were vomiting in 6 per cent. of the cases, temporary retention of urine, transitory headache and the occasional appearance of albumin and flat epithelial cells in the urine, which disappeared in a few days; 20 to 30 red blood cells per high power field also were found. Verkhovskaya concludes that alcohol narcosis is an inexpensive method, devoid of the defects of some other methods of anaesthesia.—*G.P.*

Bone-marrow as an alternate route for transfusion in pediatrics.—Freisman (*Am. J. Dis. Child.*, 62, 1944), writes that the intramedullary method of infusion is useful when the veins of the extremities have all been damaged by repeated use or cannot be found, as in peripheral circulatory collapse which prevents sufficient taping of the veins. It is indicated when intravenous administration is impossible due to widespread mutilation,

burns, oedema, poorly developed or obliterated veins or shock. In the newborn and infancy, the accessible veins are very poorly developed. The superficial longitudinal sinus, the jugular veins, or the scalp veins, may offer too great a risk of extreme dexterity on the part of the operator; hence, the intramedullary route offers a good substitute.

The authors prefer the following sites for bone-marrow transfusion: the junction of the upper and middle one-third of the tibia as a site of choice, and as a second preference the junction of the middle and the distal one-third of the femur. It is not advisable to use the sternum in children under two years of age.

Blood given through the marrow method enters the circulation just as rapidly as the intravenous route. The blood may be given either by syringe or by means of a reservoir and slow drip. The authors have used two types of B.D. needles. The smaller one is a sharp, 12 bore needle and the length of the shaft is $\frac{1}{4}$ inch (19.7 millimetres); the larger is a sharp 18 bore needle with a shaft of one inch (25.5 millimetres). Because of the present difficulty of obtaining this type of needle, they have found that a special B.D. spinal tap needle, cut down to the proper length, has been satisfactory.

These physicians feel that intramedullary infusion is contraindicated in generalized infections accompanied by bacteremia, septicemia and osteomyelitis. If the needle is left too long *in situ*, infection may result.

Routine preparations for blood transfusion using the bone-marrow route, e.g., type, etc., are the same as for intravenous route.—*G.P.*

OBSTETRICS and GYNÆCOLOGY

A high-protein diet in pregnancy.—That a high-protein diet is of value in pregnancy is suggested by a small investigation carried out by Ruth M. Leverton and Thelma J. McMillan (*Journal of the American Medical Association*, January 19, 1946, 130, 134). The patients were all of adequate economic status, of good health and under 33 years of age. Originally 75 women were included in the survey, but only 33 were followed to the post-partum period, and it is these thirty-three who form the basis of the study. Eleven of the women received 5 ounces of meat daily in addition to their ordinary diet—this brought their daily protein intake up from 58 gm. to 83 gm.; eleven received a daily supplement of the vitamin B complex; the remaining eleven received no supplement of any type. These three groups were all comparable in respect to the number of pregnancies, stage of pregnancy, and hemoglobin level. The supplements were given from the fifth month of pregnancy until three months after the confinement. One month prior to delivery the mothers receiving the meat supplement had an average increase of 10 per cent. in hemoglobin and 16 per cent. in the red cell count (the initial hemoglobin and red cell count being taken as 100 per cent.); the values for

the women receiving the vitamin B supplement were 99 per cent. for hemoglobin and 100 per cent. for red cell count, while the corresponding figures for the control group were 95 and 100 per cent. respectively. One month after delivery the meat group had an average hemoglobin value 20 per cent. higher than before starting the meat supplement, the corresponding figure for the other two groups being 7 and 4 per cent. None of the pregnancies or deliveries were complicated. Oedema occurred in three of the vitamin B group and three of the control group, but it was not seen in the meat group. Eight of the women in the meat group nursed their infants for at least two months, compared with five of the vitamin B group and four of the control group. The authors conclude: "The results indicate that the recommendation to normal pregnant women to increase their protein intake by eating a generous serving of meat daily in addition to their self-chosen diets would be effective in increasing hemoglobin and red cell concentration, avoiding oedema and promoting lactation."—*Practitioner.*

Vitamin E in menopause.—Of 25 patients, seven reported complete relief of symptoms on dosages of 10 to 20 mg. daily

over periods of from one to three weeks. Sixteen patients reported very marked relief on 10 to 20 mg. daily over periods of from two to six weeks. A great reduction in the number of hot flashes per day was evident promptly upon taking the prescribed medication, and the patient's general condition greatly improved.

The relief of symptoms in patients after administration of vitamin E could not be distinguished from that obtained with the natural or synthetic estrogens. In some cases vitamin E seems more effective in relieving the symptoms of vasomotor instability than estrogens. The chief advantage of the new drug over estrogen is in the fact that vitamin E is free of any stimulative effect or change on the genital system or in the parenchyma of the breasts. Christy, *C.J. Am. J. Obst. & Gynec.* 50:85 (July) 1945. *Bull N. E. Med. Centre.*

Ante-partum and post-partum hæmorrhage.—**ANTE-PARTUM HÆMORRHAGE.**—Hæmorrhage after the 28th week of pregnancy is due usually to (1) accidental hæmorrhage or (2) placenta prævia.

(1) *Accidental hæmorrhage.*—The bleeding is rarely due to direct trauma. Albuminuria is usually present. Fortunately the really severe type, namely, concealed or combined hæmorrhage, is infrequent. In such cases the patients are gravely ill and exhibit changes similar to those seen in "crush syndrome."

Treatment.—If the bleeding is slight, then rest in bed and sedatives will suffice. Most cases under such treatment can be brought up to term. When bleeding is more severe the membranes should be ruptured. The outlook is always much happier when external bleeding is present, for this shows that the uterine muscle possesses some function and is not completely atonic, as in cases of concealed hæmorrhage. This last is a really grave emergency. When the output of urine diminishes or anuria develops, the prognosis is always serious. First, shock needs to be treated by modern methods—warmth, sedatives, plasma transfusion. Next, the aim should be to combat the atony of the uterus. For this, pitocin, 2 units half-hourly for six doses, may be helpful. When the uterus shows signs of contracting, the membranes should be ruptured. Caesarean section, particularly when combined with hysterectomy, has the highest mortality of any treatment.

(2) *Placenta prævia.*—The old nomenclature of central, marginal, lateral has been discarded. Type 1 now refers to the placenta which is just impinging on the lower segment; type 2 corresponds to the old classification of marginal; types 3 and 4 signify a placenta which is partly or wholly central across the os.

No longer is the treatment of placenta prævia considered one of emergency. Professor Macafee of Belfast University has recently shown that with conservative treatment maternal and foetal mortality can be considerably reduced. All cases of pla-

centa prævia should be treated in a hospital. A vaginal examination is condemned unless done in the operating theatre with everything prepared for the immediate performance of a Caesarean section. A careful speculum examination will eliminate cervical conditions—erosion, polypus, carcinoma—as being the cause of the hæmorrhage. Many cases, although hæmorrhage be slight and often recurring, may be coaxed to term. Adopting such conservative treatment the high foetal mortality due to prematurity is reduced. The treatment at or near term in types 1 and 2 is rupture of the membranes; types 3 and 4 should be delivered by Caesarean section.

Every woman with ante-partum hæmorrhage, whether due to accidental or placenta prævia bleeding, should have her blood grouped and her Hb. estimated.

POST-PARTUM HÆMORRHAGE.—It is necessary to differentiate between bleeding from lacerations of the soft parts (perineum, vagina, cervix) and the placental site. If the uterus is firmly contracted the hæmorrhage is due to lacerations. For the diagnosis of the tear high up in the vagina or of the cervix it is necessary to pass a speculum. These are dealt with by suture. If conditions are such that this is impossible, then the vagina should be plugged. In bleeding from a laceration of the cervix a useful emergency treatment consists in grasping the edges of the tear with a pair of swab-holding forceps. This simple procedure will stop the bleeding pending removal of the patient to hospital.

Bleeding from the placental site: (1) *Placenta within the uterus.*—After massaging the uterus, the placenta should be expressed by squeezing the fundus with one or, in the case of fat women, both hands. Many attempts to do this result in shock. Sometimes cord injection is successful. The cord is re-cut and 500 c.c.m. (1 pint) of warm normal saline injected into the umbilical vein. An easy way of doing this is with a Higginson's enema syringe to which a small canula has been attached immediately after this the fundus should be squeezed. If bleeding continues or is at any time severe then manual removal will be necessary. But before inserting the hand into the uterus a final attempt under the anaesthetic should be made to express the placenta. In grave emergency, or when an operation is not possible, an intravenous injection of ergometrine (0.125 mgm.) may prove life-saving.

(2) *After delivery of placenta.*—The uterus is massaged and blood clot expressed. Oxytocic drugs are now given. When bleeding is persistent these should be given intravenously. A hot intra-uterine douche (dettol 5 per cent.) will stimulate the uterus to contract. If bleeding persists the possibility of lacerations being also present must be borne in mind. Bimanual compression is indicated when hæmorrhage is severe. Careful examination of the placenta will indicate whether membranes, a cotyledon or a placenta succenturiata have been retained. If membranes have been retained and are

not visible in the vagina more harm than good will result by trying to extract them. As with hæmorrhage elsewhere in the body, general restorative measures, especially

blood transfusion, are essential. It is this last that has contributed so much to the reduction of post-partum mortality.

—Practitioner.

OPHTHALMOLOGY

Retinal changes associated with diabetes and with hypertension.—A comparison and a contrast.—A. J. Ballantyne. *Arch. Ophthalm.* 33: 97 (Feb.), 1945.

The retinopathies of diabetes and of the hypertensive diseases are separate entities, both clinically and histologically.

In both conditions the earliest recognizable lesions are pathologic changes in the retinal vessels.

In diabetes these lesions affect primarily the venous, and in hypertension the arterial, side of the retinal circulation.

In diabetes these changes point to venous stasis, and, in addition to the familiar hæmorrhages and exudates, consist of congestion of the veins, microaneurysms on the capillaries and gross changes in the principal veins.

Microaneurysms may occur alone and seem to be the earliest pathologic change in the fundus of the diabetic patient.

Histologically, the earliest recognizable change takes the form of minute fatty granules in the vascular endothelium, together with swelling of the endothelial cells. In hypertension fatty granules are observed more frequently in the media and the adventitia.

The most striking changes in the retinal veins of the diabetic patient appear as expansions, beading and the formation of loops, coils and networks, and the predominant histologic changes at this stage are phlebosclerosis and intraretinal and preretinal networks of large, thin-walled vessels.

The hæmorrhages in diabetic retinopathy occur primarily in the central area of the fundus, are characteristically rounded and occur chiefly in the deep layers, for the most part the internuclear layer.

In hypertension arterial changes predominate; the hæmorrhages are primarily circumpapillary and striate, owing to their situation in the nerve fiber layer. Exudates are observed in the deep layers but also

include patches of gangliiform degeneration in the nerve fiber layer.

It may be that in both forms of retinopathy toxic factors are responsible for the initial changes in the vessels, that these factors are, at least in part, specific to hypertension or to diabetes and that their selective action on the walls of the capillaries and the layer vessels accounts for the differentiations of the two forms of retinopathy.—E.E.N.T.

Foveomacular retinitis.—The condition resembles central serous retinopathy described by Duke Elder. Gifford applied the name angiospastic retinopathy to this ocular process. Cordes (*Am. J. Ophthalm.* 27: 803 (Aug.) 1944) observed a type of foveomacular retinitis in the United States Navy. This author classifies central lesions as follows: lesions resulting from contusions, nontraumatic macular chorioretinitis, hole in the macula (resulting from trauma or solar burns), and a rather unusual type of foveomacular retinitis which has recently been observed in the personnel which served in the Hawaii Islands or the South Pacific combat zone. Approximately 176 cases were studied in detail in various naval hospitals, according to Cordes. The lesion which starts with edema of the macula and loss of the foveal reflex may be difficult to recognize in the early stages. In more advanced stages "honeycombing at the fovea or a hole or cyst in the fovea appears surrounded by a gray area about $\frac{1}{2}$ to 1 disk diameter in size, composed of very fine pigment. Vision varies: in most eyes normal vision is recovered but in some the final vision is 5/200. Cordes calls attention to the fact that in 30 per cent. of cases the second eye became involved after the first had healed. In some cases a central scotoma persisted; in others it disappeared although the hole in the fovea remained.—E.E.N.T.

EYE, EAR, NOSE & THROAT

Nasal tuberculosis.—According to Myerson (*Tuberculosis of the Ear, Nose and Throat*, Chas. C. Thomas, publisher), tuberculous infection of the nose and nasal passages is the rarest form of localized tuberculosis. "Among one thousand patients suffering with laryngeal tuberculosis, only five were found to have nasal involvement. A majority of cases occur in women. The nose of the tuberculous individual does not present any special characteristics. It has been claimed that 30 per cent. of tuberculous patients have streaks of blood in their nasal secretions, but this is not in accord with the experience of most rhinologists. The skin of the external nose may be involved

by one of several forms of tuberculosis, these are most often seen by the dermatologist. Lupus vulgaris is the most common of the skin lesions.

"Tuberculous affections of the external nose, other than those confined to the skin, occur as tumefactions, abscesses or fistulae. Tumors or tumefactions of the external structures are extremely rare; their pressure is usually unsuspected, and their nature is recognized only after removal. They are covered by intact skin and give the impression of a tumor. Of interest is the fact that the patient presents no symptoms of active tuberculosis; in fact, such a tumor may be

the first indication that he is afflicted. He may, however, report having previously suffered from pulmonary or some other form of extrapulmonary disease. These lesions, as all others of the external and internal nose, are secondary to distant foci and, with rare exception, are the result of hematogenous spread of the disease. The disease originates more frequently in an extrapulmonary focus located in the gastrointestinal, genito-urinary, glandular or skeletal system. When the original focus exists in the lung, there are usually other manifestations of the disease in one or more viscera, and in the pharynx and larynx.

"The intranasal lesions are divided bet-

ween the ulcerations and the granulomata, the latter occurring more frequently. Ulcerations are usually evidence of extensive and active hematogenous diseases. The second and more common intranasal manifestation of tuberculosis is the ulcerogranuloma. This lesion has been erroneously mentioned by many authors as a hyperplastic form of nasal lupus. There are two ways in which the disease reaches the intranasal structures; by direct contact or local continuity and by way of the blood or lymph stream. The pathology of nasal tuberculous disease is similar to that encountered in other places. The symptoms are principally those caused by obstruction."—E.E.N.T.

Correspondence

'Plight of the Rural Medical Practitioners'
To The Editors, THE ANTISEPTIC, Madras.
Dear Sirs,

The melancholy note of the Rural Medical Practitioner of Sitaramapuram and the reply of Dr. Kochunni Menon betray ignorance of the aim and scope of the Rural Medical Scheme.

It was the late Raja Sahib of Panagal who initiated the Rural Medical Scheme. As an incentive to the practitioners to settle in rural areas a subsidy of Rs. 500/- per annum from Provincial Funds and Rs. 400/- worth of medicines from the District Board Funds are being given with the express permission to charge for treatment and medicines supplied to the non-necessitous poor. The Government have given the option to grant an additional subsidy of Rs. 250/- to the practitioners by the District Boards. All these and other conditions are provided in a deed of agreement, the parties of which are the District Board President and the practitioner. Express provision is made under law of contract to refer any matter or dispute arising out of the agreement to law courts. Whenever there was a breach of agreement on the part of the President, the Government or Law Courts have come to the aid of the practitioner. This one feature of the subsidised scheme is worth more than hundred jobs under the Crown. Even the tallest of the servants under the Government do not enjoy such privileges.

Unfortunately many of the rural medical practitioners have forgotten that they are private medical practitioners settled in rural areas and the Government and District Board only pay a moiety out of their funds as an encouragement for treating the indigent. The practitioners have allowed themselves to be treated as servants and the President of the District Board and the hierarchy of clerks in his office are not found wanting in exploiting the weakness of the Rural Medical Practitioners and they are overlording the practitioners. Even for a casual reader of the agreement it will occur

that there is no provision in the terms of agreement for the President of the District Board to assume the agency of master and the whole agreement is based on equal footing between two gentlemen. In spite of such unequivocal provisions in the agreement if the Rural Medical Practitioners consider that they are subordinates like a police constable they deserve to be condemned even without enquiry.

True when the scheme was originally initiated, it was thought the villagers will be able to pay for the doctor. Now there is a strong case for enhancing the subsidy on account of the cost of living. Yet there is another reason. Villages are famishing and little is left for the villagers to pay for the doctor. So the Government might be pleased to ask the doctors to charge from patients who get more than Rs. 50/- per month instead of Rs. 30/- as is the case now and enhance the subsidy.

I would request the Rural Medical Practitioners to refuse to be boosed and consider the suffering humanity alone as masters if at all a master is necessary.

Ammur, Walajah } Yours sincerely,
Taluk. (North) } DR. C. K. SWAMY,
Arcot Dt. } Medical Practitioner.

'Some Clinical Notes on the Use of Penicillin'

To The Editors, THE ANTISEPTIC, Madras.

Dear Sirs,

With reference to Clinical notes on the use of Penicillin by Dr. N. V. Patel in the ANTISEPTIC of June 1946, the mode of using it in the outlying places are to be appreciated, but his way of injecting Adrenalin along with the solution of Penicillin and thus decreasing the frequent doses of Penicillin injections is unscientific.

The effect of Adrenalin is short, say 30 minutes at the most. It cannot retard the absorption of Penicillin.

Besides Dr. Patel does not show any scientific control of estimations of Penicillin concentrations in the blood, when the drug is used along with Adrenalin.

'Infective Hepatitis after Anti-cholera Inoculation'

With reference to case note by Dr. K.G. Chadda in the ANTISEPTIC of June '46, I believe that the infective hepatitis that followed Cholera Vaccine inoculation, was due to the virus of infective jaundice contaminating the syringe. Ordinary boiling in water of syringes does not kill the virus.

Research has shown that the jaundice following N A B injections is due to the presence of the virus (which remaining latent) in the blood sucked while injecting the fluid. Even blood taken for diagnostic purposes contaminates the syringes, and this virus is not killed when the same syringe is used for other patients.

Only autoclave of the syringes thoroughly kills the virus.

Santh Pipli
Nadiad.
Dist. Kaira.
25-6-1946.

Yours sincerely,
RAMANIK H. DESAI,
L.C.P.S., L.T.M.,
Supdt. S. M. Maternity
Hospital, Nadiad.

News and Notes

British Commonwealth and Empire Tuberculosis Conference-1947.

The National Association for the Prevention of Tuberculosis proposes to hold in London, in midsummer 1947, a Conference dealing with tuberculosis in all its aspects. This Conference will probably last three days, and invitations will be sent to representatives from the British Commonwealth and Empire.

There will be visitors also from the United States and from some European countries, and this direct exchange of ideas will promote a closer contact between various countries and the Local Health Authorities responsible for tuberculosis in Britain.

The Duchess of Portland, Chairman of the Association, will preside at the Conference.

34th All India Medical Licentiate's Conference, Poona, 1946.

The 34th All-India Medical Licentiate's Conference will be held in Poona on 19th, 20th and 21st October 1946.

There will be an interesting Scientific Section, where papers on Medical and Scientific subjects will be read. There will also be a symposium on "The Problems of Nutrition in India" and members are requested to take part in the symposium as well as send their papers before the 1st October 1946 to the Scientific Committee.

A Medical and Scientific Exhibition is also being arranged in connection with the Conference.

For further information please write to the Secretaries, Reception Committee Office, 502 Narayan Peth, Poona City.

Anti-Tuberculosis Challenge Shield

The Tuberculosis Association of India, New Delhi, awards the Hassan Masud Suhrawardy Memorial Anti-Tuberculosis Challenge Shield annually to the organization or institution which has the best record of effective anti-tuberculosis work done within that year.

Competition for this Shield is open to Corporations, Municipalities, affiliated Tuberculosis Associations, District or Local and other recognised bodies doing anti-tuberculosis work on approved modern lines.

Selection of Candidates for Nursing Training.

The Director General of Indian Medical services has issued a press note, dated 3-7-46, to the effect that four candidates for Post-Graduate Industrial Nursing training in the U. K. will be sent by the Government of India early next year.

The training is for a period of one year. Candidates will, on return, be required to serve the Government for a period of not less than three years, in any industrial area to which they may be posted.

Candidates with some experience as staff nurses in a recognized hospital will be eligible for selection. Intending candidates should ask for particulars from the Director-General, Indian Medical Service, New Delhi, immediately. Those in Government service should apply through proper channel.

The final selection will be made in September 1946 and the selected candidates will be required to sail early in January 1947.

Errata

In the article "Causative Factors and Treatment in Coronary Thrombosis" published in the June issue of the ANTISEPTIC, under the head "treatment" in page 390, the dose advised should be '4 to 2 gr. of Morphine' and not '4 to 2 gr. of Morphine' as published.

In the article on "Infantile Hepatic Cirrhosis" by Dr. A.V.S. Sarma published in page 105, of the February issue of the ANTISEPTIC, the X-Ray wrist to the left relates to Case No. 29 (1938 series). Left wrist 7 carpal centre seen.

In the article "Pediculosis" by S. N. Das published in the June '46 issue of the ANTISEPTIC in page 396, the second and third words of the 19th line should be "in ovoid" instead of "to avoid."

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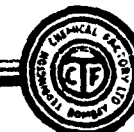
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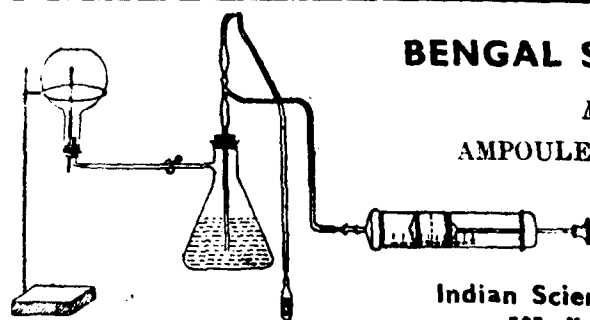
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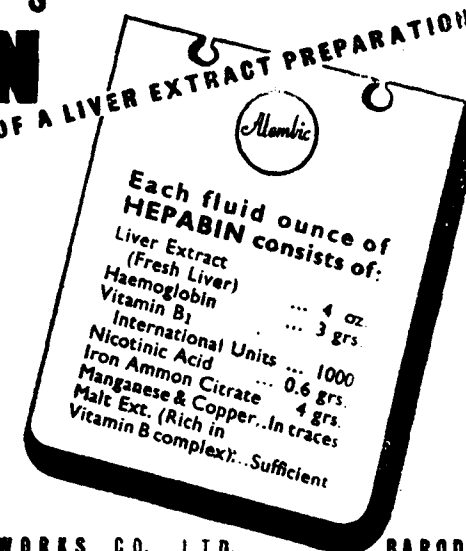


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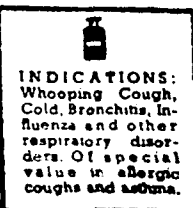
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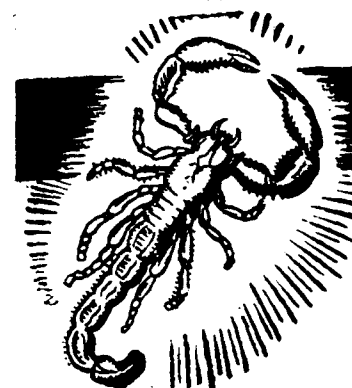
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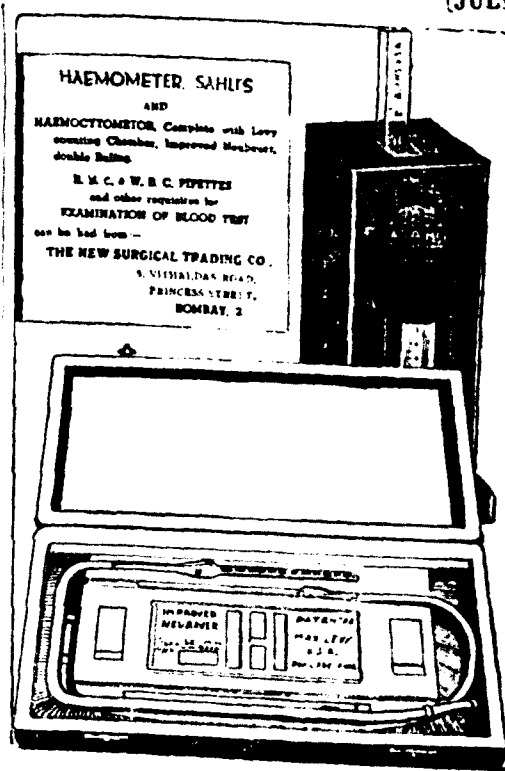


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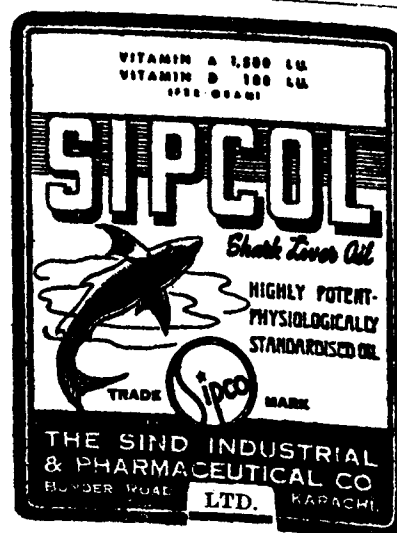
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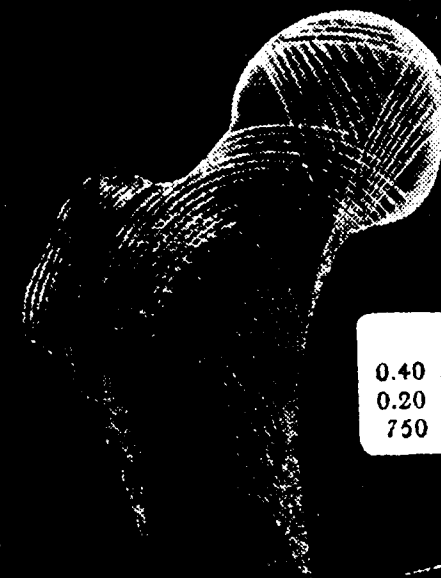
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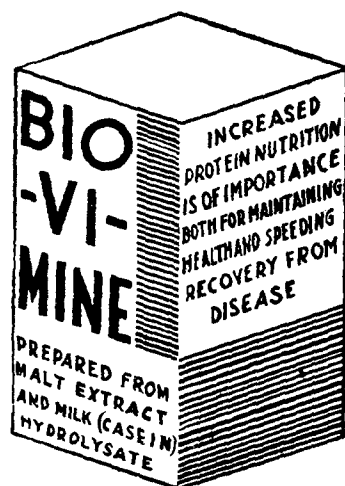
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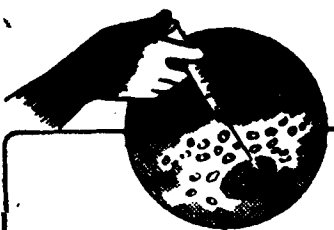
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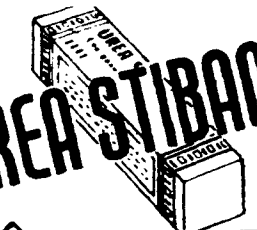


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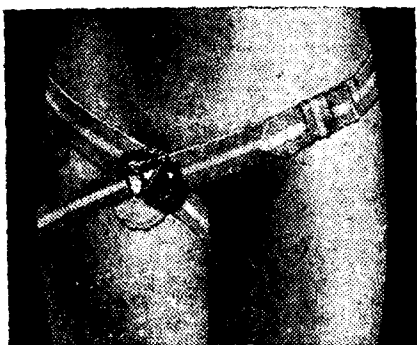
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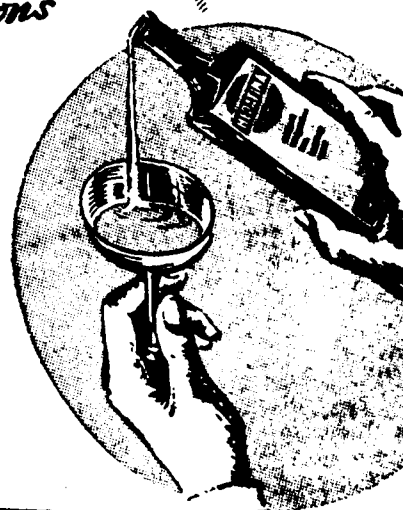
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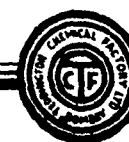
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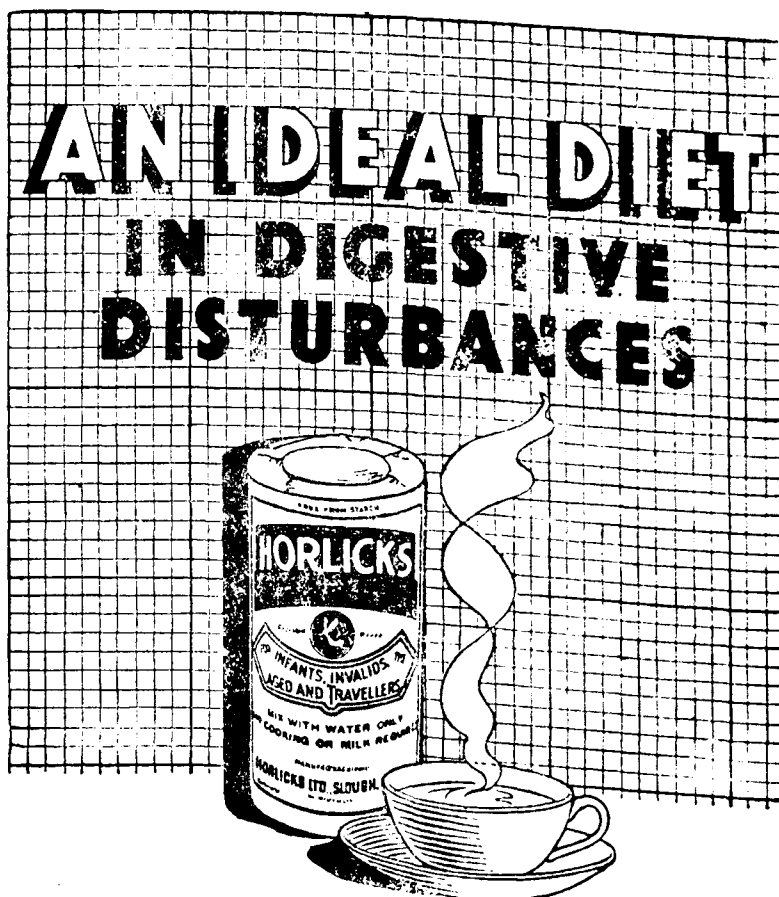
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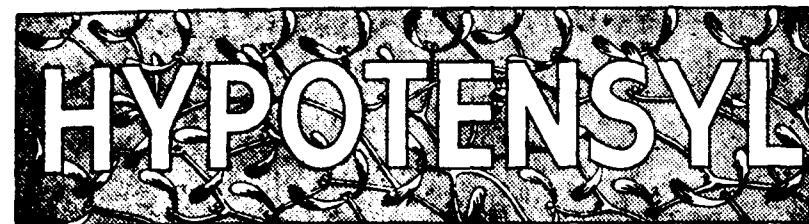
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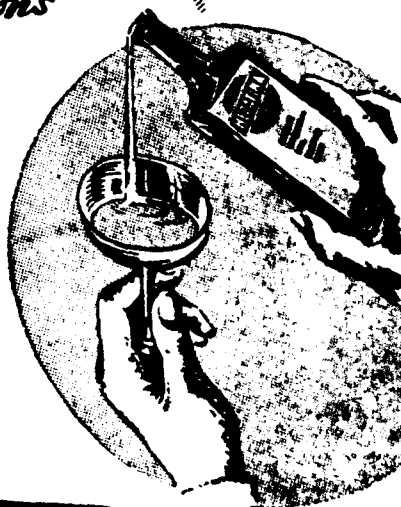
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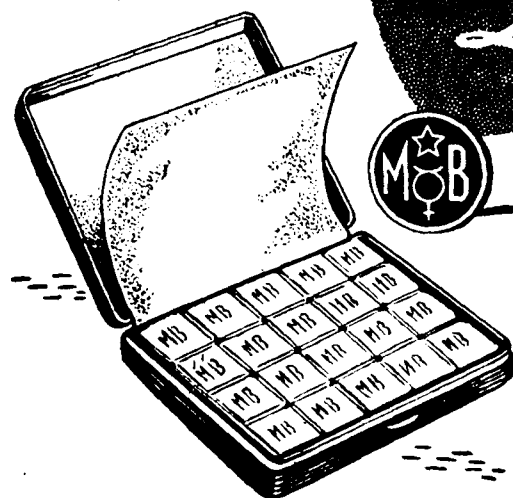
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Original Articles

Gastric Carcinoma—A Plea for its Earlier Diagnosis*

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Madras.

WE the members of the Medical Profession in general, and the Surgeons in particular, may find cause for satisfaction in the general advance of surgery during the last two decades, but upon this subject of cancer of the stomach, we can look only with gloom. It is one of the commonest of all gastro-intestinal carcinomata and though its incidence varies in different countries, it nevertheless occupies this unenviable predominance. Though the commonest, the diagnosis is delayed to such an extent that the operability in cases of patients presenting themselves with symptoms, is lower than with any other growth. Because of this delay in diagnosis, the operative mortality is still one of the highest of any major abdominal operations and the cure-rate, among those that survive the ordeal, is one of the lowest in cancer surgery. Yet, some of us who are entrusted with the relief of this depressing and almost hopeless state of affairs, find an appalling lack of interest and enthusiasm on the part of the public and the profession, in the early diagnosis of this condition—a condition in which early diagnosis alone will salvage many of these victims of an otherwise dire disease.

Within the period that is allotted to me this evening, I do not propose to go into the aetiology, symptomatology, diagnosis and treatment of this disease. In the beginning, I shall briefly enumerate the well-known symptoms and signs of gastric carcinoma. Then, I shall display the prognosis of gastric carcinoma, as it was till very recently. Before I finish, I shall endeavour to place before you a plan of action for the really early diagnosis of cancer of the stomach.

Symptoms and signs.—The so-called classical symptoms and signs are only too well-known. A carcinoma subject will have epigastric pain. This is slight at first, but gradually increases in intensity. The pain is characterised by its constant presence all through the waking hours and it does not generally have any definite relation to food, though occasionally the

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pain is aggravated by the ingestion of food. Later on, there is loss of appetite and loss of weight and, sometime later, vomiting of foul-smelling coffee-ground material, severe anæmia, and even a palpable epigastric mass.

To these general symptoms and signs certain special ones may be added, depending mainly on the situation of the growth in the stomach. Cancers situated at the inlet and the outlet of the stomach declare themselves moderately early by producing obstructive symptoms and a great deal of disturbance, whereas growths in the body of the stomach may be clinically silent and symptomless to the end or may be marked only by vague symptoms until a very late stage in the disease has been reached.

The four main clinical types are:—

- I. The pyloric obstruction type—due to growths near the pylorus;
- II. The cardiac orifice type—due to growths near the cardia;
- III. The insidious type of cancer—due to growths in the body of the stomach; and
- IV. The "ulcer-cancer" type—due to a chronic gastric ulcer becoming malignant.

I. **The pyloric obstruction type.**—Is characterised by—

- (1) A short and rapidly progressive history of dyspepsia, indigestion and continuous epigastric pain;
- (2) Epigastric pain becomes more severe after food;
- (3) At first there is vomiting of small quantity, but later, larger quantities of decomposing food may be ejected out. Due to the presence of altered blood, the vomiting may be coffee-ground in appearance;
- (4) Due to the presence of the obstruction the stomach hypertrophies and dilates; examination of the abdomen may then reveal visible gastric peristalsis, a gastric succussion splash and sometimes a hard epigastric lump to the right of mid-line.

II. **The cardiac orifice type.**—In this comparatively rare condition, the patient complains of epigastric pain particularly in the region of the Xiphisternum in front and interscapular pain in the back, immediately after food is taken, and there may be a sensation of food remaining lodged in the lower portion of the gullet. While at first solids can be swallowed with difficulty, as the disease advances and the stricture tightens, only liquids go through and, later, even this becomes an impossibility and vomiting may be present. Loss of weight sets in fairly early and is very marked. Owing to the similarity of all the signs and symptoms it is often impossible clinically to distinguish this type of carcinoma from a malignant growth of the lower end of the œsophagus.

III. **The insidious type.**—The growth being situated in the body, the symptoms are very vague and the patient may complain of lassitude, dislike for food, epigastric discomfort, or heart burn, etc. Later on, anorexia, emaciation, anæmia etc., set in or the insidious disease may manifest itself suddenly and dramatically with acute hæmatemesia or melæna, or even an acute gastric perforation.

IV. **The ulcer-cancer type.**—This, as I have said already, is a carcinoma that develops from a chronic gastric ulcer. In a patient known to be suffering from a chronic gastric ulcer of some years duration, the following symptoms would indicate or at least suggest the possibility that the ulcer has become malignant.—

chronic gastric ulcers. If there is a loss of this periodicity, then suspect a malignant change.

2. *The pain of gastric ulcer* is not continuously present throughout the day and it has a definite relation to food. If there is a loss of this regularity, if the pain becomes more or less continuous and has no definite relation to food as before, then suspect a malignant change.

3. *Anorexia*:—Which may be present in the later stages of a chronic gastric ulcer, usually increases when a carcinoma becomes grafted on to an innocent ulcer.

4. *Vomiting*:—Which had previously given relief from pain in a benign gastric ulcer, now no longer produces relief in an ulcer cancer.

5. *Loss of weight*:—Which was slight before, now increases as the malignant change advances.

Aids to diagnosis.—Having thus briefly dealt with the "classical symptoms and signs" of carcinoma of the stomach, let me now pass on to the other accessory aids to clinical diagnosis upon which we used to base the diagnosis of this malignant disease of the stomach.

The first among these will be the Fractional Test Meal. This is characterised by:—(1) *Achlorhydria*—i.e., absence of free acid in the gastric

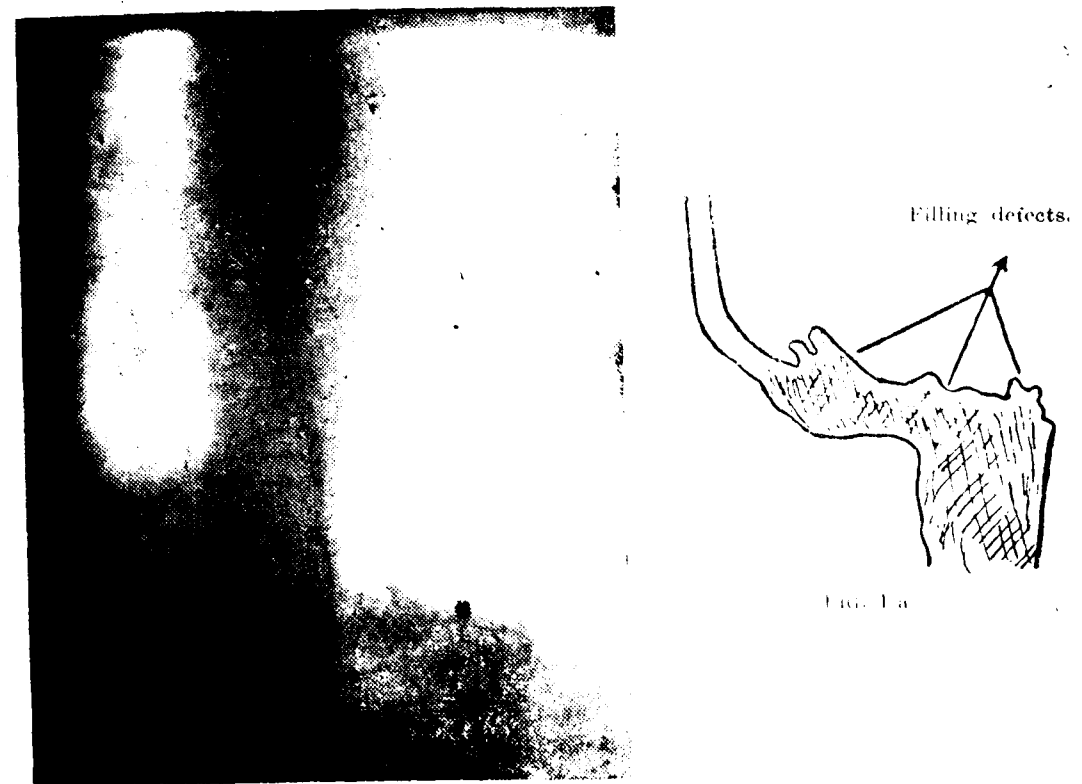


FIG. 1.
Carcinoma Cardia.

(Courtesy of Dr. P. P. Rao.)

contents; (2) *Increase of total acids* due to stasis and bacterial decomposition of food; (3) *Presence of blood* in almost every specimen syphoned out from

stomach, due to continuous oozing of blood from a malignant ulcer; and (4) Excess of mucus and starch in the gastric contents.

The second test that is very commonly employed is the Barium meal investigation and a "filling defect" is said to be diagnostic of a malignant growth, probably carcinoma. Here are a few pictures showing "filling defects."

(a) From the region of the cardiac orifice extending on to the œsophagus; (Fig. I) *vide page 511*.

(b) From the pylorus, with a moderately dilated stomach, behind the growth; (Fig. II.)



FIG. 11. Carcinoma—Pyloric region.

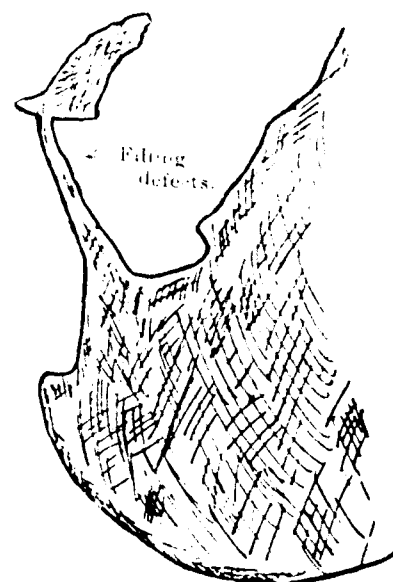


FIG. 11-a.

(c) From the body (greater curvature) of the stomach. (Fig. III) *vide page 513*.

The third test is a blood picture. In advanced cases, severe anæmia, generally of the secondary type, but sometimes of the primary type, may be present.

Treatment.—What has the surgeon to offer by way of relief, to these cases which have been diagnosed by the clinical and who has been helped by his colleagues the Biochemist, the Radiologist and the Clinical Pathologist? The surgeon can cure the patient of his disease, if it is early, and is too far advanced for the surgeon to a cure, he can give good palliation and relieve the patient of a lot of his sufferings.

In carcinoma of the pyloric region, the curative treatment will be, a radical gastrectomy which consists in removing the diseased area of

the stomach and a good bit of the stomach on either side of the growth, together with the lesser and greater omenta. The palliative procedure in an advanced pyloric growth will be to carry out a gastro-jejunostomy, so as to provide nourishment for the patient and relieve him of his vomiting etc. This prolongs life by 6—18 months.

In a growth of the body of the stomach and in an ulcer carcinoma, the curative treatment is again a radical subtotal gastrectomy. In quite a few instances of fairly advanced carcinoma of this type, instead of a subtotal, a total gastrectomy has been successfully carried out, the continuity of the

alimentary tract being restored by an œsophago-jejunostomy (*i.e.*, anastomosis between the œsophagus and the jejunum). The palliative procedure for this type of growth (*i.e.*, where a subtotal or total gastrectomy is not possible) will be a jejunostomy—*i.e.*, feeding the patient through a tube put into his jejunum. This does not prolong life for more than 3—4 months.

In a growth of the cardiac region, the curative procedure will be a total gastrectomy and the palliative procedure will be a gastrostomy.

A question commonly asked is the place of Radium and Deep X-rays in treating this condition. Though a few Radio-therapists have claimed occasio-



FIG. 111. Carcinoma—Body of the stomach.
—Courtesy of Dr. P. R. Rau.

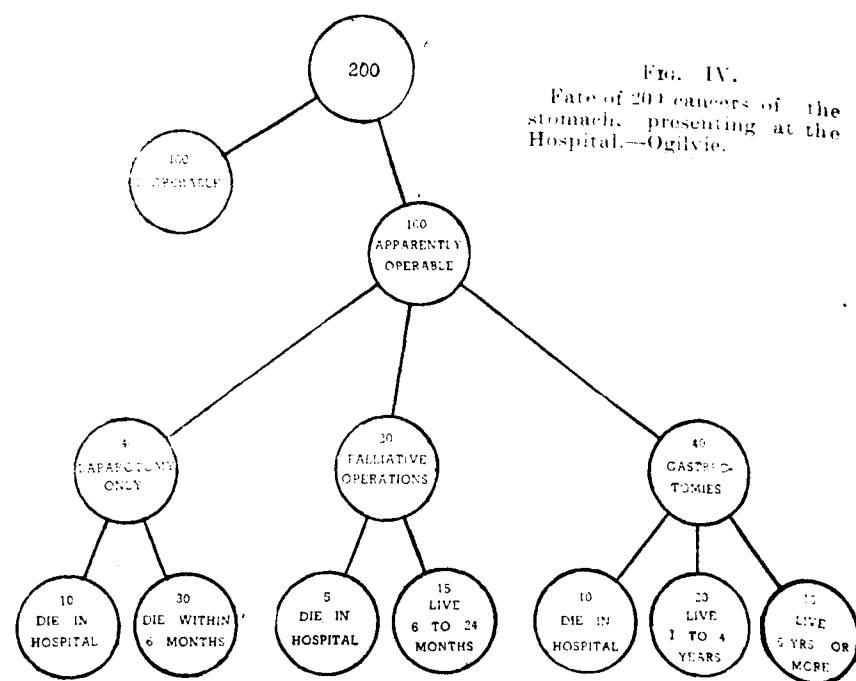
nal good results, the consensus of opinion is that it is a useless form of therapy.

Prognosis.—In addition to the treatment of carcinoma of the stomach, I have so far dealt briefly with the symptoms and signs that we, you and I, have been taught to look for in a carcinoma of the stomach and without which, the large majority of us would have hesitated to diagnose carcinoma. It is these symptoms and signs that are still described in the majority of text-books of surgery that the medical students study. What is the result of this teaching? What is the prognosis of this carcinoma, if these are the criteria of diagnosis? I have here the results of three surveys, two extensive surveys from very big teaching hospitals outside India and one from a small series of cases from one unit at the General Hospital, Madras.

CHART I.—is a survey of 200 cases of gastric carcinoma (Fig. IV by OGILVIE of Guy's Hospital, London. From his survey, you find that out of 200 cases of gastric cancer, that seek admission into the hospital, only 100 were clinically operable. Out of this 100, in only 40, were curative gastrectomies carried out; and only 10 out of this, i.e., 10 out of the original 200 cases, live for 5 years or more.

CHART II.—is a survey of 264 cases (Fig. V—Vide Page 515) by COOPER from New York Hospital; 223 were too advanced for any hope of surgical cure and were doomed to die. In only 41 were curative gastric resections carried out. Out of these 41 cases, only 25 were alive at the end of 3-5 years!

At the General Hospital, Madras, in one Unit, out of 20 cases admitted and diagnosed as carcinoma, a radical operation was possible in only two,



and even in these, the local disease was so advanced that I am sure that not one would have been alive at the end of five years!

You will agree with me that this is a tragedy—this gastric carcinoma: that this, what I have depicted here, is an appalling state of affairs: to say the least, a most horrible and sorry plight, in the midst of such happiness and gaiety that is ours to-day in Allopathic Medicine.

Who is to be blamed for this shocking state of affairs?

The blame is partly to be borne by the patient and partly, I must confess, by the members of our profession. The *patient* is to be blamed because, in spite of symptoms referable to the stomach being present at some time or other in the majority of patients with carcinoma of the stomach, the patient does not consult his physician for this early enough. This is because there are few organs in the body which the patient feels so well-qualified to treat as his own stomach. In this he is encouraged by the advertisements of stomach remedies which abound around him and his

temporary relief which may come from these even in malignant disease; yes, unfortunately even in malignant disease.

The *medical adviser* is to be blamed, in as much as he is thrown off his guard, when the patient first comes to him: (i) because of the vague and protean nature of many of the early symptoms of gastric carcinoma and (ii) because, unfortunately, he has been taught that carcinoma of the stomach means, constant epigastric pain, loss of weight, anaemia, achlorhydria and a filling defect. The picture of the emaciated, ashen-faced, sunken-eyed old man, who had been exhibited to him as a typical case of gastric cancer is still so vivid in his memory that he, the doctor, does not dare to diagnose carcinoma easily, unless these appearances are present!

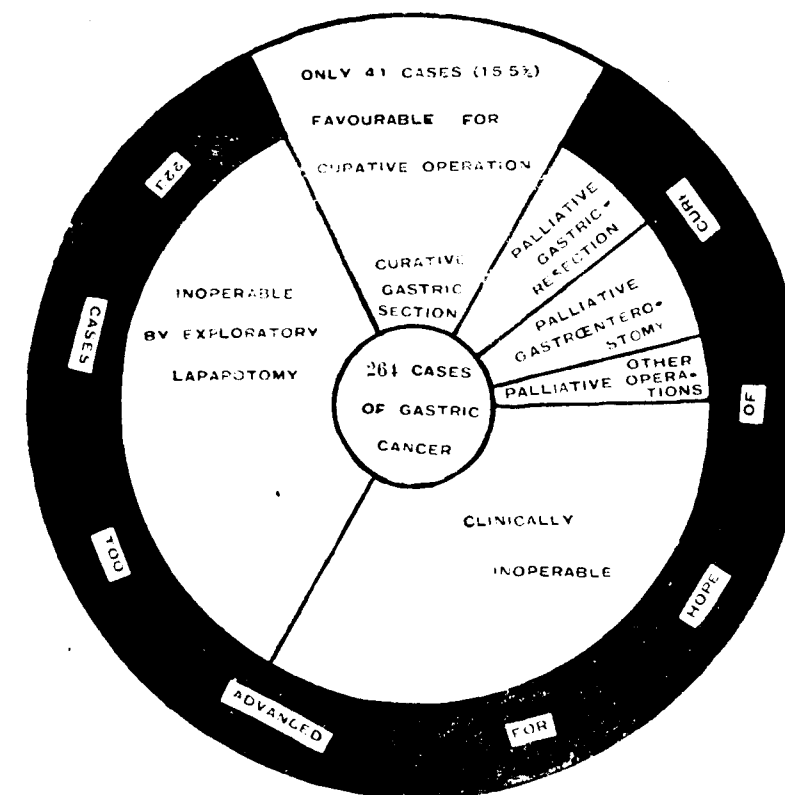


FIG. V.

Observations on 264 cases of gastric cancer.—Cooper.

What is the remedy for this woeful state? The outlook, generally speaking, would be vastly better, if certain circumstances could be brought to pass. The problem would be largely solved if every person, at least over the age of 35-40 years, could be induced to submit to routine physical examination and X-ray examination of the stomach, once every 3 to 6 months; the problem would be easier of solution if gastroscopy (i.e., examination of the inside of the stomach with an optical instrument) could be employed as a routine to demonstrate and observe the progress of certain pre-cancerous conditions like atrophic gastritis, chronic gastric ulcer and polyposis of the stomach. These objectives, however, are Utopian and it is merely wishful thinking to believe that they can ever be achieved.

Therefore, we will have to turn to less Utopian and more practical measures. These measures can be grouped under the headings of (A) For the patient and (B) For the Clinician.

(A) The Patient.—It is a fact that most members of the lay public who are past middle age, consider a mild degree of gastric discomfort to be a part of the normal process of growing older and the words "indigestion" and "dyspepsia" to most minds signify an uncomfortable, but entirely benign process. Yet, in these two words—in digestion and dyspepsia—is the whole story of early gastric cancer symptomatology. The public should be impressed with the dangerous significance of symptoms of indigestion and dyspepsia and they should be asked to report to the doctor at once, particularly, if any one, beyond 35–40 years of age.

(1) has an abrupt onset of dyspepsia, when his previous state of health was good;

(2) is losing the sense of well-being following a meal, that was once his and its place is being taken by consciousness of having eaten;

(3) is losing the appetite, or more important, has developed a change in the food desire (e.g., the patient who used to like a particular dish all his life suddenly develops an aversion to it);

(4) is developing a smaller capacity for food or a sensation of continued warmth and fullness in the epigastric region;

(5) suddenly develops fatigue and depression which continue to progress fast; and

(6) loses interest in his work and in his hobby and is physically unable to carry on without great effort.

All these are particularly ominous if there is a family history of cancer.

(B) The clinician.—To the clinician who has no specific laboratory data, chemical, serological or microscopical—on which reliance can be placed for an early diagnosis, I would strongly commend the following plan of action.

To regard as cancer any indigestion with or without symptoms, which appears after middle life, acutely or insidiously, in a previously well person.

To regard as cancer, acute digestive disturbances within this age period (i.e., after middle life) which are superimposed upon previously existing chronic digestive disturbances and which do not respond promptly (i.e., within two weeks) and permanently to routine measures which have heretofore been successful.

To regard as cancer or highly suspicious of it, such vague symptoms as fatigue, malaise, mental indifference, loss of weight, etc., even though associated gastric disturbances are entirely lacking.

Having thus suspected the dangerous possibilities suggested by the patient's history, the attending doctor should make a tentative diagnosis of carcinoma and proceed at once to prove or disprove it with all the methods at his command.

He should next carry out a thorough physical examination, which will probably do no more than fail to reveal any other cause for the patient's symptoms.

A Fractional Test Meal is then done, keeping in mind its diagnostic limitations. He must remember that though there may be a low or absent free hydrochloric acid in 70 to 80% of early carcinoma, in nearly 20% to 30% the free hydrochloric acid is normal or there may be even hyperchlorhydria.

I would request him to attach great importance to the presence of occult blood in every specimen of the Fractional Test Meal as according to

our Professor of Biochemistry, this is very strong evidence in favour of malignancy.

The clinician still pursuing his objective, must seek the aid of the Roentgenologist, who in his turn must be very pains-taking and persistent. Instead of waiting for the filling defect to appear, the radiologist must report as malignant, any one of the following conditions—

(1) Minor, but persistent defects in gastric peristalsis; (2) Lack of pliability of the stomach wall over any area; (3) Obliteration of the rugæ and changes in the normal mucosal pattern.

In addition, the roentgenologist should view with suspicion *all lesions of the greater curvature and the distal 1/3 of the lesser curvature.*

I must stress here that the patient should not be lulled into a feeling of security by a single negative roentgenographic report. The examination must be properly made, *must be repeated at frequent intervals*, and interpreted each time by the same expert radiologist. This method is so accurate that lesions as small as 1 to 1.5 c.m. have been detected by competent radiologists.

To the suspicious clinician, who has sought the help of the bio-chemist and the roentgenologist in his attempts to spot an early cancer, another valuable aid has come during recent years, *viz.*, the gastroscopy. This is a harmless procedure and can be done under local anaesthesia. With this instrument, the greater portion of the interior of the stomach can be viewed with ease and any early lesion can be detected.

It is only by adopting the routine that I have outlined, that we will be able to detect early neoplasms of the stomach and thus improve our operability rate, lower our operative mortality and increase the 5 year and 10 year cures. That I am not idly dreaming is shown by the fact that already in many American and European countries the operability has been increased very much and so has the ultimate survival rate. PACK and LIVINGSTONE have recently shown that by these methods and in cases which were detected and treated when the carcinomatous process was still restricted to the stomach, more than 50% of all patients were alive and well 5 years later. What a contrast to the state of affairs till recently and in our country, even to day!

I have finished. I hope, I have succeeded in creating in you an appreciation of the dangers of mild digestive symptoms especially after 40 years of age. No really early diagnosis of carcinoma of the stomach is possible until the exact diagnosis of indigestion and dyspepsia is taken up seriously both by the patient and his doctor. In the War against this dread and sinister disease it may be that our efforts are simply skirmishes on the margin of the battle; but even so, they must be fought with every means at our command and they call for our utmost vigilance and our most persistent effort. It has been remarked that gastric clinicians are of two classes—(1) *Those who are eternally suspicious* of gastric cancer and never find it; and (2) *Those who are never suspicious* of it and eternally miss it.

If during these few minutes of your time that I have taken I have already set in motion, a shift of the majority of your inclinations from the latter category to the category of suspicious clinicians, I shall feel extremely gratified that those few minutes have been well and usefully spent.

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The Rhesus Factor*

G. W. G. BIRD, M.B., B.S.

Major I.A.M.C.

LANDSTEINER and WEINER (1940, 1941)^{1,2} described a new human erythrocyte antigen, which they called the rhesus factor (Rh. factor). The discovery was made when they attempted to prepare anti-M serum by injecting rabbits or guinea-pigs with monkey's instead of human erythrocytes. They made use of rhesus monkeys (*MACACUS rhesus*) and succeeded in producing not anti-M serum but one which agglutinated the red corpuscles of 85% of the white population of America. It was clear that the red cells of rhesus monkeys contained an agglutinin which gave rise to agglutinin formation in the rabbits and guinea-pigs, and that 85% of white American people possessed this agglutinin in their red corpuscles. Those persons whose cells were agglutinated by this serum were called Rh positive (Rh), while those whose cells were not agglutinated were called Rh negative (rh). The serum was called anti-Rh.

TABLE I

Group	Percentage positive	Reactions with anti-Rh serum
Rh	85	•
rh	15	—

(• indicates agglutination. — indicates no agglutination.)

The distribution of Rh factor is similar in Britain (BOORMAN ET AL.) (1942)³; but racial variations have been shown to occur. The percentage of positives is higher (99%) in American Negroes (LANDSTEINER ET AL.) (1942)⁴, in Chinese (LEVINE and WONG) (1943)⁵, and in Indians (GREVAL ET AL.) (1943)⁶.

Rh seemed to be inherited as a mendelian dominant, the gene Rh determining its presence, and the gene rh its absence. There were thus three possible genotypes RhRh, Rhrh, and rh rh. As Rh is dominant the first two are serologically demonstrable as positives.

The discovery of the rhesus factor was of little importance till sera identical with anti-Rh were recovered from transfusion recipients who had had reactions following transfusion of blood which was compatible as far as the ABO groups were concerned. When it was realised that all these recipients were rh and had been transfused before, it was quickly appreciated that if Rh blood is given to an rh recipient, anti-Rh agglutinins may develop in the serum of the recipient, with the danger of incompatibility at a subsequent transfusion with Rh blood. This process of 'iso-immunisation' is slow, and the danger of serious reactions is greatest about three months after the first transfusion.

* Specially contributed to THE ANTISEPTIC.

A few cases, however, had these reactions at the first transfusion of apparently compatible blood. As all of these were rh women receiving transfusions in connection with child-birth, it was realised that the antigenic stimulus in these cases came from the foetal erythrocytes which had inherited the Rh agglutinin from the father. When some of these mothers were found to have children suffering from hæmolytic disease of the newborn (erythroblastosis foetalis) it was realised that the anti-Rh antibodies produced in the maternal circulation had re-entered the foetus and agglutinated the foetal red cells. Thus the causation of hæmolytic disease of the new-born was established [LEVINE ET AL (1941)⁷; BOORMAN ET AL (1942, 1944)^{8,9}; TAYLOR (1943)¹⁰; RACE ET AL (1943)¹¹; HOARE (1943)¹²; and STRATTON ET AL (1945)¹³]. Abortions and still-births were found to be common in families in which hæmolytic disease had occurred, and rhesus incompatibility between mother and child is believed to be responsible for these manifestations as well. Later YANNET and LIEBERMAN (1945)¹⁴ showed that a fair proportion of children with low-grade mental deficiency owed their condition to such incompatibility. This appears to result from cerebral damage, a more severe form of which is the 'kernicterus' often associated with hæmolytic disease.

Following these discoveries rh persons were always transfused with rh blood; and rh blood was used in the treatment of hæmolytic disease with encouraging results (GIMSON 1943)¹⁵. Cerebral damage, however, if present, is permanent.

Transfusion reactions were observed to result after transfusion of Rh recipients with Rh or rh blood; and Rh mothers were found to have Rh or rh children with hæmolytic disease. In fact 10% of cases of hæmolytic disease occurred in Rh mothers. Further investigation revealed that the Rh factor is a complexity of antigens, and that rh did not merely indicate absence of Rh but the presence of an antigen now called rh. The fundamental principles relating to the rhesus factor, however, still remain the same. If any Rh antigen is introduced into the circulation of one who does not possess it, the production of the corresponding antibody is stimulated, so that there is danger of a reaction if the antigen is transfused again. Similarly, if a mother lacking a given Rh antigen has a foetus which possesses it, then she may produce antibodies against the foetal antigen, with danger to herself should she be transfused with blood containing the antigen, and with every possibility of her giving birth to a child with hæmolytic disease. It should be remembered that a few cases of hæmolytic disease are due, not to anti-Rh or anti-rh but to anti-A or anti-B or to the 'extra' agglutinins alpha 1 and alpha 2, or even to anti-M or anti-N or anti-P, or perhaps to some hitherto unidentified agglutinin. One would expect more cases to result from ABO incompatibility between mother and child as this is more common than Rh incompatibility. Protective factors exist in respect of ABO which are sometimes absent in respect of Rh. Consideration of these protective mechanisms is outside the scope of this paper.

An attempt will now be made to describe briefly the facts leading to our present knowledge of the rhesus agglutinogens and agglutinins. WEINER (1942)¹⁶ was the first to show that the rhesus factor was not as simple as it was at first supposed. He found a serum in a transfusion recipient which agglutinated 70% of bloods. Those bloods which were rh did not react with this serum. WEINER concluded that there were two types of Rh agglutinin, and those persons whose cells reacted with his

new serum he called Rh 1, while those who were positive with anti-Rh serum but were negative with the new serum he called Rh 2. The new serum was named anti-Rh 1. The position now became as shown in Table II.

TABLE II

Group	Percentage positive	Reactions with	
		anti-Rh	anti-Rh 1
Rh 1	70	.	.
Rh 2	15	.	.
rh	15	.	.

This discovery increased the number of Rh genotypes to six, viz., Rh 1 Rh 1, Rh 1 Rh 2, Rh 1 rh, Rh 2 Rh 2, Rh 2 rh, and rh rh.

In 1943, RACE and TAYLOR¹⁷ obtained a serum from an Rh mother of an erythroblastotic rh child. This serum (called St) reacted with the blood of all rh and some Rh persons. Those Rh persons who reacted were identified as being heterozygous (Rhrh) in virtue of their being Rh parents or children of rh people. It reacted with 80% of bloods and failed with 20%, who were homozygous (RhRh). Family studies had shown that 38% of the population were Rh homozygotes. RACE and TAYLOR (1943)¹⁸ suggested that the 20% of bloods which were St negative were of the genotype Rh 1 Rh 1, and this has been confirmed by working out the genotype frequencies. A similar serum called anti-Hr was described by LEVINE (1942)¹⁹ which was at first thought to be distinct from St, but these sera are now believed to be identical. Another serum was discovered by RACE ET AL. (1943)²⁰ which was identified by a study of genotype frequencies as being anti-Rh 2. 30% of bloods reacted with this serum. The reactions of the six genotypes with the four sera are as shown in Table III.

TABLE III

Genotype	Frequency	Sera			
		Anti-Rh (85%)	Anti-Rh 1 (70%)	Anti-Rh 2 (30%)	St (80%)
Rh 1 Rh 1	20 %	.	.	-	-
Rh 1 Rh 2	14.4 ..	.	.	.	.
Rh 1 rh	35.1 ..	.	.	-	.
Rh 2 Rh 2	2.6 ..	.	-	.	.
Rh 2 rh	12.5 ..	.	-	.	.
rhrh	15 ..	-	-	-	.

(Note that Rh 2 Rh 2 and Rh 2 rh are indistinguishable).

In addition four more rare allelomorphs were discovered. These have been called Rh', Rh'', Rho and Rhy. The frequency of these is 1% or less [WEINER (1943)²¹; RACE ET AL (1944)²²; RACE and TAYLOR (1944)²³.] The reactions of Rhy have not been completely demonstrated. With these seven allelomorphs there are 28 genotypes.

Anti-Rh, anti-Rh 1 and anti-Rh 2 each contain one agglutinin called rho, rho 1 and rho 2 respectively. Two more sera, anti-Rh' and anti-Rh'',

were discovered but were shown to contain combinations of these agglutinins. 'Anti-Rh' contained rho and rho 1, and anti-Rh'' contained rho and rho 2. Anti-Rh' is therefore anti-Rh plus anti-Rh 1, and anti-Rh'' is anti-Rh plus anti-Rh 2. The reactions of these sera will therefore be easily understood.

For transfusion purposes bloods negative with anti-Rh should be put up with anti-Rh 1 (or anti-Rh', i.e. with sera containing rho 1), and anti-Rh 2 (or anti-Rh'', i.e. with sera containing rho 2), or the rare genotypes Rh'Rh', Rh'Rh'', Rh'rh, Rh''Rh'' and Rh''rh will be mistaken for negatives (see Table IV).

TABLE IV.

Antigens	Frequency (approx.)	Antisera					
		Rh	Rh 1	Rh 2	St	Rh'	Rh''
Rh 1 Rh 1	19	.	.	-	-	.	.
Rh 1 Rh 2	Rare	.	.	.	.	.	.
Rh 1 Rh 2	13	.	.	.	.	.	.
Rh 1 Rh 2	Rare	.	.	.	.	.	.
Rh 1 Rh 2	Rare	.	.	.	.	.	.
Rh 2 Rh 2	2	.	.	.	.	.	.
Rh 2 rh	12	.	.	.	.	.	.
Rh 2 Rh 2	Rare	.	.	.	.	.	.
Rh 2 Rh 2	Rare	.	.	.	.	.	.
Rh 2 Rh 2	Very rare	.	.	.	.	.	.
Rh 2 Rh 2	33	.	.	.	.	.	.
Rh 1 rh	Rare	.	.	-	.	.	.
Rh 1 Rh 2	Very rare	.	.	-	.	.	.
Rh 1 Rh 2	Very rare	.	.	-	.	.	.
Rh 2 Rh 2	Very rare	.	.	-	.	.	.
Rh 2 Rh 2	Rare	.	.	-	.	.	.
rh rh	13	-	-	-	.	-	-
Rh' rh	Rare	-	-	-	.	.	-
Rh'' rh	Rare	-	-	-	.	.	.
Rh' Rh''	Very rare	-	.	.	.	.	.
Rh' Rh''	Very rare	-	.	.	.	.	.
Rh' Rh''	Very rare	-	.	.	.	.	.
Rh 1 Rhy	Very rare	.	.	.	.	.	.

(Note that six genotypes containing Rhy have been omitted because they are extremely rare and their reactions have not been determined.)

Anti-Rh' serum is very common in human beings so the use of Rh' blood as negative is dangerous. The use of Rh'' blood is less risky as sera containing rho 2 are very rare. Anti-Rh' serum should therefore be used as a routine for grouping blood donors so as to eliminate all risk. When the presence of the genotypes Rh'Rh', Rh'Rh'', Rh'rh, Rh''Rh'' and Rh''rh are transfused it will be safest to give rh blood. It should be remembered that Rh'Rh' blood may make St antibody for which rh blood is incompatible. Therefore it was advised that a direct match test of donor's cells and recipient's serum in tubes at 37°C for two hours should always be done (Rhesus agglutination is not demonstrable by the slide method).

WEINER (1944)²⁴ and RACE (1944)²⁵ independently recognised an anti-body which united with homologous red cells in vitro but did not agglutinate them. If this anti-body could agglutinate it would be anti-Rh. To this, the name 'blocking' anti-body has been given by WEINER, and the name 'incomplete' anti-body by RACE. Its presence may be detected by adding the serum suspected of containing it to Rh cells normally agglutinable by anti-Rh serum. The test is, of course, performed in tubes at body temperature for two hours. Then a known active anti-Rh serum is added and if agglutination does not take place 'blocking' anti-body was present in the serum tested.

About this time RACE (1944)²⁶ described a scheme propounded by R. A. FISHER for the clarification of the complexities of the Rh genes, allelomorphs and anti-bodies. FISHER suggested that the Rh antigens are inherited from three adjacent genes occupying a small strip of chromosome, and not from a single gene as in the case of the ABO and MN groups. Each of the three genes were thought to have two alternate forms called C or c, D or d and E or e. Each chromosome carries three genes *e.g.* CDe or cDE and so on. As an individual has two of the 24 human chromosomes he will have two of the Rh chromosomes and so will have any combination of the two alternate forms, *i.e.*, if a person gets Cde from his father and CDE from his mother then his genotype is Cde CDE and so on. The relationship of the new nomenclature to the old was soon worked out and is as under (Table V).

TABLE V

Old terminology	Fisher's terminology
Rh 1	CDe
Rh 2	cDE
Rh'	Cde
Rh''	cDE
Rho	cDe
Rhy	CdE
rh	cde
anti-Rh	anti-D
anti-Rh 1	anti-C
anti-Rh 2	anti-E
St	anti-c

(FISHER used Greek letters for the sera; the use of the terms anti-C etc., were suggested by CAPPELL 1944²⁷).

This scheme predicted the discovery of a further gene CDE (Rh_z) and two more sera (anti-d and anti-e).

It should be noted that anti-Rh' is a mixture of anti-C and anti-D and anti-Rh'' is a mixture of anti-D and anti-E. Rh negative (rh) bloods give rise to anti-D in 50% of cases and to mixtures of anti-C and anti-D in the remainder. Blood for emergency issue, therefore, should not contain C or D.

Rh (CDE) was discovered by RACE ET AL (1945)²⁸; and in the same year MOURANT²⁹ found anti-e. These discoveries more than gave support to Fisher's hypothesis.

COOMBS, MOURANT and RACE (1945)³⁰ discovered a new way of detecting 'incomplete' anti-body. Normal cells were exposed to a serum suspected of containing a specific anti-body, then washed thoroughly and exposed to rabbit anti-human globulin serum. This serum will only agglutinate cells which have anti-body globulin absorbed onto their surface. The value of this test is that it can be performed quickly on a tile. The same workers (1946)³¹ have shown that the test is of use in demonstrating in-vivo iso-sensitisation of red cells in babies with hæmolytic disease, and may therefore be used as an early diagnostic measure in such cases.

RACE ET AL (1946)³² found a new Rh anti-body which enabled a third alternative form of C to be recognised. This third allelomorph at FISHER's C-c locus has been named Cw and the new anti-body anti-Cw. Half the sera hitherto called anti-C are in actual fact mixtures of anti-C and anti-Cw and only half are pure anti-C sera. The up-to-date position of Rh antigen-anti-body reactions is shown in Table VI.

TABLE VI

Antigens		Antisera						
Original	Fisher's	Anti-C	Anti-D	Anti-E	Anti-e	Anti-d	Anti-c	Anti-Cw
Rh 1	CDe	*	*	-	-	(-)	*	-
Rh 2	cDE	-	*	*	*	(-)	-	-
Rh'	Cde	*	-	-	-	(*)	*	-
Rh''	cDE	-	-	*	*	(*)	-	-
Rho	cDe	-	*	-	*	(-)	*	-
Rhy	CdE	(*)	(-)	(*)	(-)	(*)	(-)	(-)
Rhz	CDE	*	*	*	-	(-)	-	(-)
rh	cde	-	-	-	*	(*)	*	-
	CwDe	-	*	-	-	(-)	*	*
	Cwde	-	-	-	-	(*)	*	*
	CwDE	(-)	(*)	(*)	(-)	(-)	(-)	(*)
	Cwde	(-)	(-)	(*)	(-)	(*)	(-)	(*)

(The brackets indicate reactions not yet demonstrated. The reactions of genotypes have not been given because the table would have been too cumbersome. They can easily be worked out from first principles.)

RACE, MOURANT and SHIELA CALLENDER (1946)³³ have observed that the nine Rh allelomorphs give rise to 45 genotypes, and if the Rh groups are considered with the A1A2BO, MN, P and the new "Lutheran" systems, but ignoring the very rare new "LEVAY" group, there are 18,225 possible genotype combinations. It may well be that some day the individuality of the blood will be established.

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Treatment of Corneal Ulcer*

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RECENTLY I read with interest the article "Penicillin in Eye Diseases" written by Dr. K. KRISHNAMOORTHY and published in the *'Antiseptic'* Vol. 43, No. 5, May 1946. He has however dealt with only one of the diseases of the eye, viz., trachoma, in which Penicillin has been found to be of some value. There are however many other diseases of the eye in which Penicillin has given wonderful results. To name a few they are:—

1. Acute conjunctivitis.
2. Purulent conjunctivitis.
3. Gonococcal conjunctivitis.
4. Ophthalmia neo-natorum.
5. Corneal ulcer,
6. Corneal abscess,
7. Intra-ocular sepsis (panophthalmitis), and
8. Endophthalmitis, etc.

Out of these conditions I intend to take up the subject of corneal ulcer and its treatment with Penicillin.

I must say without much exaggeration that Penicillin has greatly revolutionised the treatment of corneal ulcer. Penicillin has been found not only to shorten the period of recovery but has been found to prevent

* Specially contributed to THE ANTISEPTIC.

and combat any complications arising therefrom. In short it is a great advance in the line of treatment of corneal ulcers as compared to the other lines of treatment given in text books.

Method employed.—The method employed in the treatment of corneal ulcers with Penicillin is as follows:—

Penicillin is prepared in solution in distilled water in strength varying from 1 in 500 or 1 in 1,000, and two drops of this solution is instilled in the affected eye every 2 hourly for a period varying from 1 to 4 weeks depending upon the severity of the condition.

For simple corneal ulcers this is all that is required. In fact within 48 hours or so, a considerable improvement is noticed. The conjestion becomes less and in some cases the eye becomes white.

Photophobia and laceymation disappear and the patient feels considerable relief. The instillation of the drops has to be continued for a week more to prevent a relapse. So for a simple corneal ulcer it does not take more than a week or ten days for complete recovery.

In ulcers complicated with hypopyon, the following additional treatment is found necessary, besides the instillation of drops as suggested above.

Penicillin solution 1 in 1,000 1 c.c. is injected sub-conjunctivally every day for about a week or so depending upon the severity of the complication. The hypopyon gets absorbed after the second or the third injection.

In addition to this, Redoxon or Vitamin C 500 mgm. given intravenously has been found to be of great value in bringing about an early recovery.

So, in short, the treatment of corneal ulcer resolves itself into—instillation of Penicillin drops 1 in 500 every 2 hourly, injection of Penicillin 1 in 1,000 sol. 1 c.c. sub-conjunctivally, injection of Vitamin C intravenously and Sulpha drugs by mouth in appropriate doses.

I would like to cite a few cases treated on the lines suggested above so that the reader may be able to judge for himself the efficacy of Penicillin treatment over other lines of treatment adopted so far.

Case 1.—A boy aged 10 came to me with trachoma both eyes and a corneal ulcer in right eye at 4 o'clock position.

There was much photophobia, laceymation and mucopurulent discharge from both the eyes with a circum-corneal conjestion in the right eye.

In the beginning, he was given the following treatment: 1. His eyes were irrigated daily with a simple Boric lotion 3%. Silver Nitrate 2% sol. was applied to both the eyes twice a week followed by Hydrargyri per Chlor lotion 1 in 4,000 wash.

On rest of the days in the week, Silver Nitrate 1/2% sol. drops were put in both the eyes. Atropin 1% per cent. sol. drops was put in the right eye, and Yellow Oxide of Mercury 1% ointment was applied to both the eyes.

Sulphanilamide tablets were given by mouth.

This treatment was given for two weeks at the end of which it was found that though the trachoma showed signs of healing, the corneal ulcer did not. The patient still complained of photophobia and laceymation, and circum-corneal conjestion was still present.

Then for another week Atropin 1% sol. drops, Sulphacetamide 30% sol. drops and Sulphonamide ointment 5% applied every day to right eye and it was kept bandaged.

Even then at the end of a week no appreciable change was noticed.

Then finally I ordered for Penicillin and after preparing it in solution 1 in 500, asked the patient's relatives to instill 2 drops at 2-hourly intervals in the right eye. The solution was stored in ice chamber to prevent its deterioration. To my surprise, I found that after 48 hours, a considerable amount of improvement was noticed. The circum-corneal congestion was much less. There was practically no photophobia and very little of laceymation. The instillation of drops was continued for a week more and the patient made an uneventful recovery without any recurrence of symptoms.

Case II.—An adult male aged 35 came to me with a corneal ulcer at the centre of the cornea right eye, and a hypopyon with marked congestion of the whole of the eye. There was marked photophobia, pain and laceymation. The following treatment was given:—

Penicillin solution 1 in 1,000 was ordered to be instilled in the affected eye every 2-hourly.

1 c.c. of the same solution was injected sub-conjunctivally every day for a period of one week.

Redoxon 500 mgm. was given intravenously every day for 10 days and Cibazol 1 Tab. four times a day was given internally for three weeks.

In addition to Penicillin drops Atropin 1% solution was also instilled in the affected eye.

With this treatment, the relief of symptoms was prompt. The hypopyon got absorbed after the third injection. Other symptoms like photophobia, laceymation and pain subsided within a week and the whole ulcer healed up within a period of less than 6 weeks.

Case No. III.—An adult male aged 46 came to me with a chronic serpigenous ulcer extending transversely from one cornea to the other in the right eye. The whole eye was deeply congested and there was laceymation and photophobia.

In the beginning the following treatment was given:—

The eye was irrigated daily with Boric lotion 3% and Silver Nitrate 1/2% sol. drops, Atropin 1%, Dionin 3% and Yellow Oxide of Mercury 1% ointment was applied. The eye was kept bandaged.

Cibazol tablets were given internally. This treatment was continued for about two weeks, but at the end of this period no improvement was noticed. The congestion in the eye was of the same intensity and the ulcer showed no sign of healing.

Then I ordered for Penicillin and after preparing it in strength 1 in 500, asked the patient to put 2 drops of this solution in the eye every 2-hourly. The patient took the solution home and was putting the drops as per instruction. He returned to me after a week, when I was surprised to see that the congestion in the eye had completely disappeared and the eye looked white. The ulcer was showing signs of healing and the patient felt much relief. The patient was asked to continue the same drops for a week more.

But unfortunately when he came to me after 10 days I found that the eye was quite red and congested as before and this time there was hypopyon in the anterior chamber. The constitutional symptoms were marked. I could not understand the recurrence of these symptoms, but later on I learnt that the patient finding his eye quite normal, had discontinued putting the drops any further.

This time I gave him one injection of 1 c.c. of Penicillin sol. 1 in 1,000 sub-conjunctivally and asked the patient to come next day for the injection. Unfortunately the patient never returned thereafter for the treatment. I am however quite sure that had he continued the treatment and the injections, it would have been possible to bring about a speedy recovery.

In contrast to these cases cited above, I would like to cite the following case of corneal ulcer in both the eyes, who came to me for treatment some 2 years ago, when Penicillin was not available for civilian use. The patient was an adult male aged 25 who had corneal ulcer in both the eyes. Both the eyes were deeply congested and constitutional symptoms were marked. Following treatment was given:—

- (1) Both the ulcers were caterised with caubolic acid.
- (2) Both the eyes were washed with Boric Lotion 3% and the following drops were instilled:—

Atropin 1% Solution,
Silver Nitrate 1/2% Sol.,
Sulphacetamide 30% Sol., and
Sulphamide ointment 5% applied.

- (3) Two injections of T.A.B. Vaccine 25 million diluted in 2 c.c. distilled water were given intravenously at 15 days interval.

- (4) Six injections of Milk 5 c.c. were given intramuscularly.

- (5) Cibazol tablets 6 per day were given internally.

- (6) At one time it was found necessary to do paracentesis.

With this treatment it took nearly three months to bring about a complete recovery. I am sure that had Penicillin treatment been given a trial, that period of recovery would have been much shorter.

Conclusion.—1. Four cases are cited, out of which three received the Penicillin treatment, while the fourth did not, thereby showing the differences in the period of recovery between those that received the Penicillin treatment and the one that did not.

It therefore definitely proves that Penicillin brings about an early cure of corneal ulcers. It is found to be more efficacious than any other treatment tried so far.

2. Instillation of Penicillin drops has to be continued for some time after the congestion in the eye has disappeared, otherwise there is a possibility of recurrences of the symptoms.

3. Chances of developing any complications are few.

4. Complications like hypopyon, yield very quickly to Penicillin treatment.

Tropical Eosinophilia*

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THIS condition was originally described by FRIMODT—MOLLER and BARTON (1940). Now more and more cases are being recognized and observed from almost every part of India. Such conditions are also being recorded from Ceylon, Tongking in China, America, Havana, Tanganyika in Africa, Samoa in the South Pacific, Egypt and Singapore.

Ætiology.—The ætiology is not yet known and various theories have been advanced.

WEINGARTEN suggests some correlation with the sea-side but cases have been recorded from places which are far off from sea-sides. No age seems to be exempt.

Symptoms and signs.—The symptoms are always varied and it is dangerous to clinch the diagnosis only on mere shaky signs and symptoms. There are many cases which are not examples of this condition but have been included as such and the treatment in such cases have always been unsatisfactory. Some of the common symptoms are being enumerated below.

This is characterised by low fever. Some cases seem to have acute onset with fever going upto 101–102°F. followed by troublesome cough.

Cough is a constant symptom—which is irritating and annoying at times. At night it becomes worse. This irritating cough gets worse as time passes and is often frankly spasmodic in character resembling a typical bronchial asthma.

Sputum is scanty and mucoid and at times frankly blood-stained.

The patient gradually loses weight inspite of his taking a most nutritious diet. Gradual loss of appetite and increasing weakness are observed. Pulse is rather rapid.

X-ray examination.—Suggests infiltration of the lungs which confuses the diagnosis with tuberculosis of lung (miliary), or pneumokoniosis.

Examination of chest.—V. F. No change.

Percussion note.—hyper-resonant.

Auscultation.—reveals harsh breath sounds with prolonged expiration—many bronchi in the early stages which are later replaced by crepitations which persist after coughing. Vocal resonance shows no change.

Examination of sputum.—Always found negative for tubercle bacilli—contains usual bacterial flora—contains large number of eosinophil cells—Charcot-Leydon crystals and Curshmann's spirals have not been reported.

Examination of blood-film.—Marked leucocytosis—From 15,000 to over 100,000 per cmm.

Eosinophils.—Over 2 per cent. and may be as high as 80 per cent. of the differential count.

Absolute numbers of the neutrophils and the lymphocytes are usually unaltered.

As regards eosinophils.—Lt. Col. P. N. BARDHAN and Capt. N. TYAGI suggest and have proved by a number of experiments that there are some cells in the granulocyte series of leucocytes which stain so peculiarly that they cause great confusion in differentiating eosinophils from polymorphonuclear leucocytes. This stained (peculiarly) cell is a polymorphonuclear leucocyte with eosinophil cytoplasm showing fine granules. It shows a similarity to

a polymorph in the nuclear pattern and a similarity to an eosinophil in having acidophilic cytoplasm. The granules vary from groundglass consistency to the typical coarse eosinophil granules. These peculiarly stained cells are present in health also but more so in illness. To consider these cells as a separate class of leucocytes is rather difficult at this stage, but it should be conceded that they are some atypical phases of granulocytes.

These slides were shown by Col. BARDHAN to other recognized pathologists. The different pathologists gave different opinions and they refused to recognize these peculiarly stained cells as a separate entity. Col. BARDHAN is of opinion that they should be classed as different cells. Such is the confusion as regards the recognition of eosinophil cells. Such confusion sounds a warning against a diagnosis of eosinophilia. To avoid confusion Col. BARDHAN puts the definition of eosinophil cells as follows:—“The eosinophil leucocyte is by definition a polymorphonuclear or a kidney-shaped unclear cell with coarse acidophilic granules and nothing else should be called an eosinophil.” The fact that these cells are found both in health and more in illness would suggest that they are perhaps effete cells.

Other conditions which produce eosinophilia are.—(1) Physiological. In infancy, upto 10 per cent. (in Indians.)

(2) Allergic—(a) Bronchial asthma, often slight, occasionally as high as 20 per cent. of 20,000; (b) Hay fever, upto 10 per cent. during the period of symptoms; (c) Migraine, inconstant and slight; (d) Urticaria, eczema and with positive tuberculin reaction.

(3) With parasitic infestations (a) Infection with intestinal tænia (not constant), ankylostomes (up to 15 per cent. of 10,000), ascariis, oxyuris, etc. (far less constant) amœbic dysentery (inconstant). (b) In tissues: trichina, (20 per cent. of 20,000 and higher), echinococcus (50 per cent. of normal total count), coccidioidal granuloma (occasionally high, 12 per cent. of 22,000), filaria (less marked, occasionally 15 per cent.) and bilharzia.

(4) Familial eosinophilia—is high and persisting but rare.

(5) In skin diseases.—Angioneurotic œdema (variable up to 85 per cent. of 44,000), pemphigus (upto 60 per cent.), Dühring's disease (upto 40 per cent. of 12,000), scabies (5 to 15 per cent.); herpes zoster (occasionally high), psoriasis, urticaria, eczema (less marked).

(6) In diseases of the hæmopoietic system, myelocytic leukaemia (1 to 5 per cent. of high total count), the so-called “eosinophilic leukaemia” (80 per cent. of 100,000), Hodgkin's disease (usually trifling, occasionally as high as 55 per cent. of 25,000).

(7) With certain infections (a) cholera (4 to 6, per cent. of normal total count, gonorrhœa (inconstant, may be upto 12 per cent.), scarlet fever (5 to 10 per cent. of 20,000) and active tuberculosis. (b) Post-febrile, e.g., after pneumonia (up to 13 per cent.), measles, varicella, rheumatic fever and malaria (only slight).

(8) In certain diseases of bones. Sarcoma, metastatic carcinoma, osteomyelitis, rachitis, osteomalacia, osteitis deformans.

(9) With various neoplasms. Rare but reported upto 30 per cent.

(10) In endocrine disorders. Addison's disease (6 to 10 per cent.), ovarian disease (non-suppurative, non-malignant), also reported with menstruation.

(11) From certain chemicals, etc. After administration of uncooked liver (up to 50 per cent.), following camphor, pilocarpin, phosphorus, chronic copper sulphate poisoning, etc.

(12) After irradiation. Two to three weeks later up to 20 per cent.).

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(13) After splenectomy. After one month (up to 15 per cent. for many months.)

(14) Inconstantly noted after Emetine injections and injections of Insulin, liver, etc.

Differential diagnosis.—*Bronchial asthma*:—In asthma the blood count is seldom over 15,000 and the eosinophils are under 20 per cent. There is no febrile reaction. Paroxysms are much severe.

2. *Miliary tuberculosis*:—Is a much more acute condition and is invariably fatal within a few weeks.

3. *Loeffler's syndrome*:—Its duration is only for a week and it is a self-curing condition.

A case note.—Recently I had occasion to treat a case of this condition. The patient, Deo Kumar Singh, a young intelligent man, had been suffering from cough and low fever since last 13 years. The cough used to be worst at nights and occasionally he was thrown into fits of most irritating cough which resembled asthma or whooping cough. The trouble was relieved by Adrenalin and Ephedrine. During these 13 years he became well and free of cough, etc., on two occasions, once some two years back and another time some seven years back. Since last two years he was in great agony and he had no hope of even being cured. He was labouring under a wrong notion that he had developed tuberculosis of lung. He spitted blood also on more than one occasion. He had taken 2 courses of Calcium injection, 30 injections of Calcium Gluconate at an interval of 1 month. He had also taken 10 injections of Soamin. He had also taken some time back some few injections of Sulfarsenol. He had numerous other treatments e.g., Ayurvedic and Vaidya etc., etc. When he first saw me he insisted upon my giving some injections. When I looked to the list of treatments he had, I found that doctors have left nothing for me to inject this poor man. I took his blood on two slides and sent him to Arrah to get the blood report (differential and total leucocyte count) as early as possible.

When the result came I found that his total leucocytic count was 49,000 per c.mm. and the eosinophils was 71%. This blood report was enough to clinch to my provisional diagnosis of tropical eosinophilia.

I examined his urine also for albumen etc., and then started with N.A.B. (M. & B.) injection. I started with 15 gm. and after a lapse of six days increased the dose and injected the next higher doses 30, 45, 60, 750 and finished with 9 gm.

The interval between the two injections were 6 days. With the first two injections the symptoms were aggravated but the action of subsequent injections were really marvellous. The patient is now free from all troubles and is putting on weight.

Treatment and discussion.—It has now been confirmed that in this condition Arsenicals are the drugs of choice. These arsenicals act best when given by the intravenous route. I have found that N. A. B. given intravenously gives brilliant results. The action of Soamin and Acetylarsen are rather slow. Soamin when given should be nothing less than 20 grains in all and it should be given by the intravenous route. For anaemia, Iron should be given. *Plastules hæmatinæ* with liver extract, I have found doing a great deal towards combating anaemia which is of secondary origin. Appetite to be stimulated with the help of stomachics and vitamins, especially vitamin B₁.

Note:—I have not seen cases of tropical eosinophilia relapsing. The

cases I have treated have not relapsed. I would like to know the views of my colleagues through this most popular journal THE ANTISEPTIC. "Neosalvarsan, if given in a course of six injections intravenously, commencing with 0.3 gm. and then continuing with 0.45 gm. at weekly intervals, establishes a practical cure which may last over 3 years and in some cases even much longer" (S. K. VAIDYA, *Ibid* 365). I hope readers would enlighten me on this subject of relapsing eosinophilia. What should be the treatment in relapsing cases?

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Typhoid or Enteric Fever*

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School Health Officer, Agra.

TYPHOID is very common among school children. I do not wish to go into the details of its aetiology or pathology. I will mention only so much, that its cause is *typhoid bacillus*.

Clinical symptoms and signs and diagnoses.—(1) *Fever*:—In most cases fever is the only symptom to begin with. (a) In a majority of cases physicians are apt to consider that fever as malaria fever. On the first day of rise of temperature most of the doctors cannot make any diagnosis as to what kind of fever it is, and if the doctor is wise, he should never give out his diagnosis for 5 days as to what kind of fever it is, for the diagnosis can be correct only after 5 days. One is apt to consider it to be malaria fever from which it can be distinguished by not coming to normal i.e. 98.4°F during the first 3 days at any time during the 24 hours. One should make it his rule that if the temperature does not come to normal during the first five days he should declare it to be typhoid fever. (b) This fever is so severe that it makes the child exhausted, the look of the child is very anxious.

At the disposal of the general practitioners in private practice there is nothing but this clinical symptom: (i) there are other symptoms such as strawberry coloured tongue, (ii) constipation, (iii) slow pulse, (iv) tenderness in the region of spleen, (v) severe headache, but they are such symptoms that a private practitioner can never be definite about his diagnosis and one should never give his diagnosis until the 5th day. However much the guardians of the child may insist. He must tell them it is a waiting game, he cannot give his diagnosis. I remember that when I was called to see one Raj Kumar, a son of a Maharaja, he had all of a sudden a rise of temperature. His father was very anxious to know the kind of fever. I could not give him my diagnosis. He sent for other doctors to consult, but no one could give definite diagnosis. The whole history of the case was sent to Dr. B. C. Roy of Calcutta, he too could not give his diagnosis and wrote in reply that it is a waiting game. The diagnosis cleared after five days when temperature did not come to normal. There was another case of typhoid in a royal family, the Raj Kumar had sudden rise of temperature, his blood was examined and some germs of malaria were found under microscope, but I did not give my diagnosis as the temperature did not come to normal during the first five days. After the 5th day I gave the diagnosis of typhoid fever of typho-malaria type. When a case of typhoid occurs in a royal family, the Chief Medical Officer is in great danger of his

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reputation and of keeping the patience of the Maharaja. He insists on giving diagnosis and if the Chief Medical Officer gives a diagnosis without being definite he loses his reputation, if the diagnosis comes out to be wrong. Therefore one should never give his diagnosis till after the 5th day. By that time the clinical picture becomes clear and if the temperature does not come to normal and pulse and temperature ratio is disturbed the diagnosis becomes definite. It is to be distinguished from pneumonia by symptoms and signs and rate of respirations. Typhoid sometimes is complicated with bronchitis, sometimes with pneumonia, sometimes with mumps. So one should be very careful in giving diagnosis.

Treatment.— If the doctor is called to see the case on the 1st day of rise of temperature he should give only diaphoretic mixture (i.e., Liq. Ammonia Acet. Dil. Pot. Nitrate, Spt. A ethers, Nitruse, etc., and aqua pure according to age of child). If after the 5th day the temperature does not come to normal then he should give typhoid mixture i.e., the following:—

Spt. Ammonia Arom	℥ x
Pot. Citrate	gr. ii
Liq. Ammonia Acetate Dil	℥ 30 to 60
Oil Cinamomi	℥ 4
Tr. Digt.	℥ iii
Syrup Aurantii	℥ 30
Hexamini	gr. v
Aq.	oz. 4 t.d.s.
For a child of 8 or 10 years.	

The following powders should also be given:—

Cal Lactate	gr. iii
Salol	gr. ii
Equinine	gr. ii

One powder to be given twice a day morning and evening.

The above mixture and powders should be continued till temperature remains above the normal all the 24 hours. It comes down to normal after 7 days, 14 days, 21 days, or 28 days. Then stimulant mixture should be given and the following is the best:—

Spt. Amonite Arom	℥ vii
Spt. Aetheris	℥ iv
Tr. Cardiom Co.	℥ vii
Spt. Vini Galicii	℥ xx
Aq. Chloroformi	4 oz. t.d.s.

If bowels do not move give glycerine suppository or glycerine enema; no other purgative should be given under any circumstances till temperature comes down to normal and then only liquid paraffin or castor oil.

During convalescent Easton syrup 3 i should be given.

Nursing.—The most important thing is the nursing of the patient. He should remain in absolute rest in bed. He should not get out of his bed even for passing urine or motion. When temperature is high, cold water pads should be applied on forehead and cold sponging should be done on arms and legs only.

Diet.—Nothing should be given by mouth except Glucose water or fruit juice or honey or albumen water till temperature comes down and then only milk for 10 days after the temperature becomes normal. These ten days after the temperature is normal are very critical. child is very hungry. He cries for food, and sometimes attempts to steal food, and sometimes mothers who are very sympathetic give some kind of food, and the child gets a relapse. Therefore strict orders should be given and absolutely nothing but fruit juice and milk should be given for 10 days. After 10 days food should be increased gradually. First liquids, the semi-solids, and then solids. No relapse will occur under this regime.

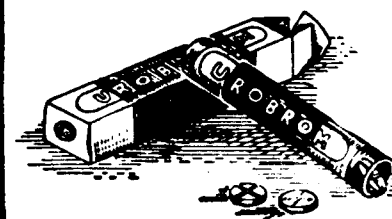
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Enemata*

M. P. RANJAN, I.M.P.,

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Hospital Road, Laheria-Sarai.

Introductory.—Enemata or clysters play an important role in the treatment of the sick. The main idea is to introduce them high up in the pelvic colon to induce peristalsis of the gut causing thereby softening of the contents of the bowels and rapid expulsion. The rectum is distended with the enemata fluid which is retained and absorbed. Water, salts, alcohol, glucose are absorbed fairly well from the colon but drugs which are chiefly absorbed through the small intestines are less slowly absorbed from colon.

Classification.—Enemata used generally for intestinal diseases are classified as under :—

1. **HIGH ENEMATA** :—This is better known as colonic lavage or irrigation. It is used much less commonly owing to the difficulties that it presents. The fluid injected per rectum in these enemata reaches the cæcum and by gradual distension of the colon induces peristalsis and thus helps evacuation. With a view to empty the bowels of the irritating and toxic contents contained therein, this method is frequently employed but sufficient time must be allowed for the fluid to find its way along the bowels.

Despite its so many advantages, it has a few drawbacks consisting of the chances of habit formation and the disturbances in the entire normal physiological activity of the colon—nervous, muscular or secretory (mucus).

(a) **Technique.**—The patient lies on the back with the hips raised by a pillow. A soft rubber tube or catheter is introduced 4 to 5 inches in the rectum and the funnel held some 18 inches above the bed. The fluid warmed to body temperature is so allowed to run in slowly and when sufficient amount of fluid has run in, it may be syphoned off by lowering the funnel and the process repeated till the returning fluid is quite clear.

(b) **Amount of fluid.**—For inflammatory lesions or ulcerative cases, one pint in acute cases and more in chronic cases. To empty the colon, one to two pints of plain warm water may be injected very slowly.

2. **LOW ENEMATA** :—They act mechanically as chemical irritants or softeners of hard faeces or by a combination of both these effects. As heat does not play any important part, enema given at a higher temperature than that of body is useless, hence better avoided.

(a) **Technique.** :—The patient lies on the left side with the knees drawn up. The anus is opened up by placing the finger and the thumb on each side. The apparatus (douche can) consists of a glass or enamelled funnel and a rubber tubing about two feet long to which is attached the nozzle. The nozzle well-vaselined is introduced gently in the direction of the anal canal (with a slight inclination to the left hip bone following the canal) and the fluid at a temperature of 100°F. to be allowed to run in slowly.

(b) **Amount** :—(i) Infant at breast—1 to 2 oz.; (ii) Infant one year 3 oz.; (iii) Child 5 years—4 oz.; (iv) Child 8 years—8 to 12 oz.; and (v) Adult—one pint or more.

(c) **Precautions** :—(i) Solution should be properly warmed at 75°F. to 85°F.; (ii) Tube or nozzle to be warmed and well-oiled before use—

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avoid air to be pumped in. (Lynch's safety enema syringe or Higginson's syringe preferred); (iii) Protect bed and clothings by oil cloth; and (iv) Inject very slowly.

(d) *Enema rashes*.—Urticarial wheels—previous history of having been given enema-irregular distribution and polymorphic character of the rash.

Classification.—To facilitate description, low enemata may be classified as under:—

1. *Purgative*.—To expel the contents of the bowels by dislodging the faeces and flatus from the intestines.

(a) *Enema Saponis*:

℞ Saponis Mollis (B. P.) ... 3 i
Aqua destillata ... ad one pint

(b) *Enema Glycerini*:

℞ Glycerine ... 3 i
Castor Oil ... 3 i
Aqua ... One pint

(c) *Water enema*: According to MCKENNEY, a pint of plain cold water used as enema daily for a week followed by douching on alternate days for the next week to regulate the bowels to normal motions; 2 pints of warm water with soft soap is also used but when there is much congestion in the lower gut, Normal Saline or 1% Sodii Bicarb solution should be employed.

(d) According to LEVY, two tea-spoonfuls of the following mixture in 4 oz. of water to be given as enema:—

℞ Rect. Spirit of Turpentine ... 5 c.c.
Cotton Seed Oil ... 25 c.c.
Glycerine ... 45 c.c.
Soft liquid soap ... 45 c.c.

2. *Astringent*.—To offer local treatment of the inflamed states of in rectum and colon.

(a) *Solution of Alum*.

(b) *Iced solution of Hamamelis*.

(c) *Infusum Quassia*.

(d) *Solution of Silver Nitrate 1 in 1,000 or 1 in 500*.

3. *Digested*.—To supply food when the patient is unable to swallow:—

℞ Beef tea ... 8 oz
Yolk of egg ... one
Raw Arrow root—one tea-spoonful.

Mix and warm in covered jar up to 100°F., then add separately.

℞ Pepsin ... gr xv
Strong Hydrochloric acid ... ℥ x

Allow them to remain at the same temperature—Stir the mixture and add small quantities of Sodii Bicarb so long it effervesces. Inject slowly to be absorbed in due course.

Take care that:—(i) Natural motion had lately passed; (ii) Bowels were previously washed; (iii) Amount does not exceed 4 oz. at a time; (iv) Process repeated every 4 hours; and (v) A towel is placed against the anal orifice to prevent escape of enema.

Nutrient.—To supply food to the patient when unable to take orally. When swallowing is rendered difficult in the diseased states of the intestines or after serious operations—essential in post-anæsthetic vomiting.

(a) ℞ Glucose ... 3 p
Aqua ... 3 x

To be given by continuous drip method to facilitate retention and slow absorption. Repeat every 4 to 6 hours.

(b) ℞ Yolk of an egg ... 3 p
Beef tea or fresh milk ... 3 p
Brandy ... 3 p
Liq. Pancreaticus ... 3 i p

Inject after bowel wash and repeat every 4 hours.

(c) ℞ Egg ... one
Fresh milk or beef tea ... 3 iii
Sodii Bicarb ... gr xx
Pancreatic solutions ... 3 ii

Switch the egg and milk—add the remaining and allow to stand in a jar of hot water for 20 minutes before use.

(d) ℞ Peptonised beef ... 3 i
Ext. of malt ... 3 i
Gruel ... ad 3 ii (obsolete)

(5) *Stimulant*.—To combat shock and collapse, to relieve severe shock and bodily decompression—

℞ Brandy or strong coffee ... 3 i
Normal Saline ... one pint every 4 hours

'Special Technique'.—The patient is propped up in Fowler's position (which assists in easy introduction of the fluid and its retention) and a glass tube (bent to a right angle 3 inches from the rounded end with several perforations) is introduced into the rectum. The nozzle remains *in situ* while enema is being given and which is connected to an irrigator containing the fluid at a temperature of 103 to 110°F. Take care that the calibre of the tube is small, else all the fluid will be rejected due to irritation of the sphincter. The fluid is allowed to run in slowly wherefrom it is absorbed and carried to circulation. The rate of flow must be regulated by the height of the reservoir. 7 to 8 pints in total may be allowed to be absorbed in 12 hours but flow must be regulated to avoid distention of bowels. This has proven very useful in cases of collapse, hæmorrhage and toxæmia.

(6) *Sedative*.—To afford soothing effects to bowels, bladder, uterus, etc., in some inflammatory conditions:

℞ Chloral Hydras ... 3 p
Pot. Bromide ... 3 p
Aqua ... one pint

(7) *Diagnostic*.—Simple soap water enema—very useful and helpful test in diagnosis of intestinal obstruction. If first enema fails, repeat after 2 hours. If no result, diagnosis is obvious. Presence of mucus (in considerable amounts) in stools with absence of flatus are very much suggestive of the disease. In doubtful cases, half an ounce of liquid Paraffin to be given twice daily to ensure correct diagnosis.

(2) *Opaque enema*.—This is the best method for the examination of the lower intestines, especially the motor functions of the colon. It yields earlier and more correct idea as to the presence of any stricture in the iliac or pelvic colon which is not so satisfactorily obtained by the opaque meal. The fluid when introduced outlines the colon and the palpatory examination done under screen. Each portion of the gut can be separated by the fluid that has run in and inspected at different angles.

(a) *Preparation of the patient*.—(i) To avoid errors in diagnosis due to remaining faeces, open the bowels with suitable aperient and lavage some hours before the examination. (ii) Restricted fluid intake but not complete fasting.

(b) *Opaque fluid* :—

$\mathcal{R}$ Barium sulphate	... 3	vin
Kaolin	... 3	iv
Mucilage Tragacanth	... Q. S.	
Aqua destillata	... 1	pint

Absolutely fluid—there should be no lumpiness, else it will block the tube.

(c) *Technique* :—The patient lies on his back and the fluid allowed to flow from the reservoir kept at the height of 2 to 3 feet connected with a valveless Higgison's syringe. The tube is introduced into the rectum and the fluid introduced by the syringe at light pressure. Ordinarily, the fluid reaches the cæcum in 2 to 5 minutes time. The patient does not feel any inconvenience in retaining the fluid till the examination is done and skiagram taken. During the filling process, the head of the fluid is constantly watched by intermittent screening. To obtain further information about Polypii, injection of air into the colon helps a good deal.

(d) *Results* :—(i) Organic stricture :—(a) Fluid obstructed at the site of the stricture; (b) X-ray shadow ends at the seat of obstruction; and (c) to avoid errors, wait for an additional 10 minutes, increase or decrease the pressure, massage at the site to relax the spasm of the gut by the change of posture.

(ii) Carcinoma of the colon :—Commonest sites are at the lower sigmoid, junction of descending colon with sigmoid, cæcum, hepatic and splenic flexures. In malignant strictures, wide dilatation of the gut below in advanced cases leading to complete obstruction of the fluid. In early stages, only filling defect is outlined in the bowels. After having filled the colon as far as practicable, skiagram is taken. To avoid doubt, skiagram of the doubtful areas caused by the folds of the gut should be taken separately. Remember that a small area of narrowing in sigmoid is normally present and should not, therefore, be mistaken for complete obstruction. Similarly, a lax sphincter does not allow the patient to retain enema fluid and hence the colon beyond sigmoid is not filled. Complete obstruction of the colon caused by an acute diverticulitis, however, cannot be differentiated from neoplasm.

(iii) Neoplasm :—By this method, the presence of a filling defect in the colon caused by a growth is determined giving information as to the exact length and fibrous strictures. The flow of the enema is retarded due to the new growth but still the bowels function normally although diarrhoea sets in due to the spasmodic contraction of the bowel at the seat of obstruction; the enema reaching and distending the parts immediately below it.

(iv) Chronic colitis :—Normal pouching and the flexures of the gut disappears. The gut appears to be a series of straight patent tubes joining the various bands together owing to the inflammation of the area and the adhesion caused thereby.

(v) Diverticulitis :—Due to localised thickening, true picture of that produced by the neoplasm except more jagged and irregular appearance of the gut to whose margins small diverticulæ usually project.

(vi) Appendix :—Filling defect is the diagnostic sign. By palpation, the cæcum is moved for a better view and skiagram reveals the lumen, definite fixation, sometimes in an abnormal position, retention when the cæcum is empty, definite localised tenderness on palpation, stenosis or kinking of the appendix.

Clinical usage.—1. Severe diarrhoea and tenesmus :

$\mathcal{R}$ Tr. Opii	... m	xx
Starch Mucilage	... 3	iv

2. Tetanus :

$\mathcal{R}$ Chloral Hydras	... 3	p
Pot. Bromide	... 3	p
Aqua ad	... 1	pint

3. Apoplexy :

$\mathcal{R}$ Castor oil	... 3	p
Spt. Turpentine	... 3	p
Croton Oil	... m	iv
Soap	... 3	p
Warm water ad	... 3	viii

4. Restlessness :

$\mathcal{R}$ Paraldehyde	... 3	p
Olive Oil	... 3	p
Normal Saline	... 3	ii

5. Post-operative pain and restlessness (gynaecological) :

$\mathcal{R}$ Sodii Bromide	... 3	i
Lithii Acetyl Salicylatis	... gr.	x
Aqua destillata ad	... 3	v

Mix with equal amount of warm water and inject just after the operation.

6. 'Whites' and womb diseases :

$\mathcal{R}$ Zinc Sulph.	... gr.	xx
Tr. Opii	... 3	p
Warm water	... 3	viii

7. Post-operative ileus and intestinal obstruction :

$\mathcal{R}$ Ext. Fellis Bovini	... 3	i
Oleo Perbeuthinae	... 3	iv
Glycerin	... 3	iv
Enema Saponis ad	... one	pint

8. Meteorism (flatulent distension) :

(a) $\mathcal{R}$ Oil of Turpentine	... 3	i
Olive oil	... 3	ii
Soft Soap	... 3	iv
Aqua ad	... 3	xx
(b) $\mathcal{R}$ Asafoetida	... 3	i
Soft Soap	... 3	i
Castor Oil	... 3	i
Warm Water ad	... 3	viii

9. Thread worms :—

(a) $\mathcal{R}$ Sodii Chloride	... 3	i
Inf. Quassia Recentum	... One	pint
(b) $\mathcal{R}$ Quassia Chips	... 3	iv
Tepid Water	... One	pint

10. Ulcerative colitis :—Colonic lavage (irrigation) on alternate days for 10 to 14 days with Sodii Bicarb solution (one drachm to a pint) or Normal Saline to remove faeces and mucus; followed by Yatren solution 1% or Tannic acid (1 in 500) or Protargol or Albargin (1 in 1000) or Potassium Permanganate (1 in 5000) for astringent and antiseptic effects.

11. Chronic dysentery :—

(a) 5% solution of Bismuth subgallate in 4 to 8 oz. of Olive oil.

(b) Eusol solution (1 to 9 oz. of sterile water) injected and retained.

Strength of Eusol increased up to 8 oz. in equal quantity of water.

(c) Washing out of large bowels on alternate days with Normal Saline or warm Boric lotion or with two pints of Eusol diluted one and half times in warm water. Later, injection into lower bowel of 6 to 8 oz. of Silver Protein (B.P.) $\frac{1}{2}$ to 1% or Silver Nitrate solution ($\frac{1}{2}$ gr. to 1 oz.) or half per cent. solution of Chiniofon (B.P.).

12. Obstinate constipation :—

(i) $\mathcal{R}$	Rutæ Folium	6 parts
	Carni Seminum	6 parts
	Lauri Nobilis Seminum	6 parts
	Piperis Nigri	1 part
	In pulverem reduce, Misce.	
$\mathcal{R}$	Asafoetidae	2 parts
	Mellis	44 parts
	Aqua destillate Q. S.	
	Solve Calorē Modico M. Paulatim.	

Sig: 180 grs. to be used to 20 fluid ounces of Infusion of Chamomile

(ii) $\mathcal{R}$	Olive or Liquid Paraffin	3 v
	Tr. Belladonna	m xx

Warm and infect at the body temperature very slowly at night and allowed to retain. Place a pad of cotton wool to avoid soaking of bed clothes. Belladonna helps to relieve the painful and spasmodic effects of the bowels.

13. Amœbic dysentery :—

(a) Yatren enema—2% solution of Yatren in 10 to 20 oz. of water as a bowel wash for a week daily and repeated according to the results after a week. This should be retained for 8 hours at least.

(b) Entero-vioform enema :—8 to 10 oz. of $\frac{1}{2}$ to 1% suspension of the drug warmed at body temperature in chronic cases given for 5 days with a period of rest and repeated according to the results and other indications.

(c) Carbarsonc enema :

$\mathcal{R}$	Carbarsonc	3 p
	Sodii Bicarb	gr. xxx
	Aqua destillata add	3 vi

To be given on five alternate nights preceded by a bowel wash—better given 2 hours after meals at night. To facilitate perfect retention and absorption, some quickly acting hypnotic; e.g. Sodium Amytal or Dial or Luminal in 1 to 2 gr. doses should be given previously to ensure sleep. Stop oral administration of the drug to prevent over dosage.

(d) Bowel irrigation with 2% Allargin, 1% Argyrol or 1% Copper Sulphate solution.

Conclusion.—From the above, it is quite evident how do the enemata help in relieving some of the most distressing conditions especially when the oral administration of the drugs are rendered impossible. In suitable cases, it is of immense value, although temporary, to combat the ill effects. Although they play very important part in special circumstances, still they should be used sparingly and only when occasion demands. Generally, enemata are employed in cases of removal of the hard, scabulous masses preventing normal motions but also of great service in absolute constipation with debility, infection and inanition etc. For very weak patients who are not allowed to undergo any strain or exercise or in cases of invalids or when no aperient is possible through oral route, the method is frequently employed with good results. Very strong solution of Magnesium Sulphate is

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Common Colds—Its Prevention and Treatment

VALLABHDAS N. MEHTA, L.M. & S.

Morvi.

THE common cold is a universal destroyer of comfort and efficiency and the cause of vast economic losses, and while it is not a definite disease entity it is a condition which, in most cases, can be prevented by hygienic measures.

If you scatter a handful of corn on the surface of a metalled road, it will not grow, because the soil is too hard and the conditions are generally unsuitable. Plow up a bit of the same road, water it, and add a little fertilizer, and your corn will grow there as well as anywhere.

The upper respiratory tract constantly harbours a multitude of bacteria of many kinds, but in perfectly healthy persons, these produce no pathologic symptoms whatever because the soil is not suitable for their growth. Let the body resistance be lowered by toxæmia or other causes or the local conditions of the nasal mucous membranes rendered favourable for bacterial growth, infection promptly ensues.

There are many conditions which lower the body's resistance, among which are overwork, too little sleep, underfeeding or improper feeding, worry, lack of exercise, focal infections of various kinds (teeth, tonsils, prostate etc.) and probably the most common of all—toxæmia due to deficient elimination of wastes by the bowels, kidneys, skin and lungs.

Ordinary colds and acute bronchitis almost always have a basis in digestive disturbances. If one regulates the diet carefully using a maximum of fruits and vegetables and a minimum of pastries and condiments, secures one or two bowel movements a day, takes a cold bath every morning and a walk of at least three miles every day, and sleeps eight hours out of every 24 hours, in a well ventilated room, he will be practically cold-proof.

There are also a number of conditions which cause hyperæmia, and turgescence of nasal mucous membranes and irritation of those of the throat, larynx and trachea. Among the former the same old auto-toxæmia from deficient elimination is one of the worst offenders. Sexual overactivity sometimes causes it and so does local chilling of some areas of the skin. Dust, smoke, fumes pollens and other like things sometimes cause irritation all the way down.—Protein sensitization or anaphylaxis is sometimes the etiological condition.

Individuals who indulge too freely in affectionate embraces with persons of the opposite sex, but who do not obtain complete gratification, are prone to suffer from congestion of the nasal mucosa as a reflex accompaniment of the resulting hyperæmia of the sexual organs—many persons suffer from a low grade rhinitis during the engagement period, which disappears soon after marriage.

The following suggestions indicate the rational methods for preventing colds under ordinary conditions: (1) Avoid overwork and loss of sleep whenever possible and it usually is possible, if we develop a sound and proper sense of relative values. (2) Eat enough but not too much. Take plenty of milk, fresh fruits, and green leafy vegetables. (3) Take a reasonable amount of exercise, preferably out of doors and in the sunshine. (4) Have all sources of focal infection cleared up. (5) Secure adequate activity of all the organs of elimination. The bowels should move two or three times a day—enough water should be drunk (4 to 6 quarts daily) to flush the kidneys—the skin should be kept active by daily bathing and the lungs by plenty of deep breathing.

So much for general hygiene. Now for the local factors: (1) Accustom the skin to changes of temperature by exposing it to warm and cold air and water daily and avoid local chilling—as by a draft on some circumscribed part of the body. General chilling of the surface rarely results in colds. (2) If sensitive to certain pollens or other proteins, have the condition treated by anti-allergic vaccines or avoid the offending substances or both. (3) Keep out of dust, smoke and irritating gases as much as possible.

This advice may sound like a council of perfection, but the nearer you come to following it, the freer you will be from colds, winter and summer.

Treatment.—The treatment should be prompt to be effective and consists first of a good cleaning out with Calomel 1 gr. in divided doses, followed three hours later with a full dose of Magnesium Sulphate in a glass of hot water.

As a local treatment 1% solution of Chlorazene applied high up in the nose, and also used as a gargle in the throat gives great relief and adds to the hastening of cure.

Another local treatment which is highly efficacious is as follows—Prepare a 1 in 5,000 solution of Metaphen, a new synthetic organic Mercury compound and with an ordinary pipette, run a few drops in both the nostrils, letting it trickle back through the nose, until the whole nasopharyngeal mucous membrane is well covered. The patient is directed not to blow the nose, but to absorb any excess discharge with a handkerchief, the purpose being to keep the remedy in contact with the membrane as long as possible. These applications may be made several times a day. This treatment gives immediate relief from the congestive symptoms, i.e., the sense of fullness, difficulty of breathing, and general discomfort, which is so characteristic of rhinitis, acute or chronic. In about three days the symptoms entirely disappear.

Medicinal.—Atropine Sulph. gr. 1/500 every 2 hours for 5 doses, and then every 3 hours, for 5 more doses. Mostly this will abort, but if it progresses, and the congestion becomes severe, this will not abate until the secretion of mucous is free. Atropine should be stopped and Ammonium Chloride may be given. If there is an irritable cough Codeine may be added to the mixture.

If expectoration is profuse, Terpin Hydrate may be substituted for Ammonium Chloride. It should always be given in powder or in capsule. If the patient has difficulty in raising the muco-purulent secretion from the bronchial tubes and if it is sticky and hard to expectorate, Sodium Iodide in small doses is the best treatment.

One should not underestimate the importance of colds since these never leave the patients in perfect condition, but always necessitate a period of recuperation and further more, they predispose to repeated attacks.

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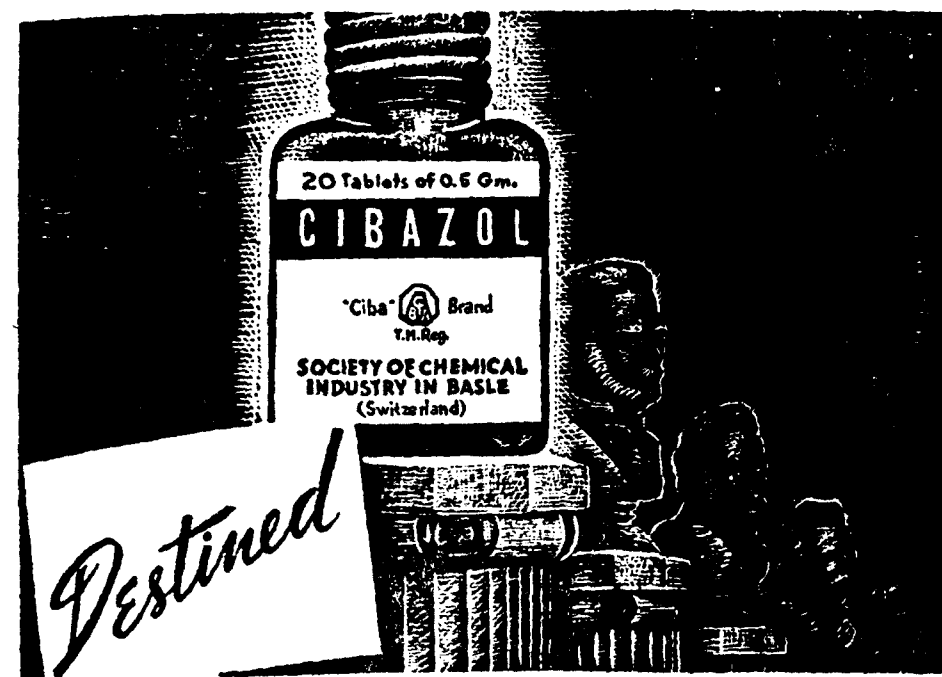
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Cases and Comments

A Case of Chorea Gravis

N. R. MERCHANT, L.M. & S.,

Mahuva, Kathiawar.

K., a Bania girl, aged about 16, came under my treatment in December 1944 for a severe type of chorea in the fifth week of her illness, after having tried all sorts of treatment at the hands of vaides and doctors. On examination it was found that she had lost even night's rest and it was getting extremely difficult to administer food or nourishment. A big soft bed on the floor with many cushions to prevent her from bruising herself with some contrivance to restrict the movements, if possible, had to be provided, so violent were the involuntary movements. She was put on Mag Phos. 6x a biochemic remedy, 5 grains every two hours, with a sedative draught of Potas Bromide 15 grs. with Chloral grs. x at night. B₁ Injections, 25 mgrs. for a dose, were immediately started. This treatment showed improvement even from the 2nd day. She began to take proper rest at night and take her food. Movements were the same as before. From some experience of these cases in the past I thought Mag. Phos. with Bromide Chloral was chiefly responsible for the improvement. I however continued B₁ injections.

I write this from memory and at this distant date I do not remember how the case actually progressed, but after about eight days she developed an abscess at the site of one of the injections. There were small boils and bruises all over the body and I don't exactly know the cause of this abscess. The patient would not let me open it and it burst by itself in a couple of days and closed spontaneously. Thereafter the treatment rapidly controlled all the symptoms and B₁ was stopped. About 12 injections were given in all. She made rapid and uneventful recovery after that and went away to her village.

She again came for the same condition in September of this year, this time at the end of the 3rd week of her illness. She was put on the same treatment except that Mag Sulph. 25% injections was substituted for B₁ injection. This gave her some much needed rest at night but otherwise she was getting worse. The treatment was not found satisfactory and was, therefore, discarded. Sodii Salicylas grs. xv, Potas Bromide grs. x, Liq. Arsenicalis m. iii were prescribed as a mixture every 4 hours with vitamin B comp. 2 c.c. as injections. She was getting some relief from this treatment when on the 4th day she developed high temperature with all the symptoms greatly aggravated. I examined her for rheumatism, endocarditis and malaria which was prevalent at the time but found nothing positive to guide me. Under the circumstances I was content to wait and see, but those who try anything that comes to hand whether there are indications or not might have scored here. But the patient stuck to me through thick and thin because, perhaps, I had helped her through the first attack. What with restlessness and want of sleep and want of nourishment the condition of the patient was miserable and serious indeed. Cardinal and other sedatives were pushed to their extreme limit but had no effect on the symptoms but made her more stupid and exhausted in the bargain. I wonder how Morphia and Hyoscine should have acted here but they did not occur to me then.

The patient continued in this condition for 3 days when at one of my visits the patient's mother drew down the drawers, which the girl was wearing, and showed me a big red patch covering almost the entire left buttock. I could not at the time differentiate cellulitis from erysipelas, but here, at any rate, was the cause of high fever. I promptly injected Soluseptasine and advised to give one Protalbin (Bayer's) every 3 hours which was in the house at the time. Next day the mother came for medicine at 10 a.m., and reported that temperature had gone down about 8 hours after the injection and the patient had gone to sleep thereafter and had not been awake till then. I visited the patient at my usual time—1 p.m. What I found to my surprise was that the patient was very calm, comparatively speaking. That Sulphanilamide often acts in a dramatic way in erysipelas or cellulitis is one's common experience, but here it seemed to stay the original diseased process itself. This continued for a couple of days when from the 3rd day the movements began to increase and in a short time assumed their former severity and violence. Sulphonamide was discontinued. I changed over to B₁ injections. In the meantime an abscess had formed on the left ant. sup. iliac spine, burst by itself and closed without any treatment. B₁ injections rapidly controlled the symptoms thereafter and after 4 or 5 injections they were stopped as the patient refused to have any more injections. The case now looked like a mild form of chorea. I prescribed vitamin B₁ comp. and stopped all other treatment. She came to my dispensary after about 12 days and was well except for a few jerky movements on the left side. She again came in a few days with no symptoms at all. The latest I hear about her is that she is weak but otherwise well.

Summary and conclusion.—(1) B₁ injections have a decidedly beneficial effect in chorea gravis. In this particular case I gave 25 mg. per dose but I believe 50 to 100 mg. per dose would have acted better.

Chorea being allied to rheumatism, Aspirin or Salicylate, preferably the former, is always worth trying. Aspirin or Salicylate with Mag. Sulph. 25%, 5 to 10 c.c. is another good treatment in moderately severe cases. It is not to be supposed from this that B₁ will rapidly cure a mild case because it acts so well in a major case. My experience is that drugs have not much effect in this. The man who treats the case last perhaps gets the credit. I believe it ends spontaneously. Time factor is important in all cases of chorea. The later in the course a case comes the more amenable it is to treatment. I am, however, open to correction in this matter.

(2) Sulphonamide simply acted on the complication and the case again took a serious turn even while it was being continued.

(3) Whether abscess formation had anything to do with the improvement? I am led to believe that it had some effect. B₁ began to have rapid action both the times, after the formation of the abscess. If a similar case came to me again and B₁ failed to hold out good prospects, I should seriously think of exciting leucocytosis either by drugs or an artificial abscess preferably of the latter method.

A case of the Depressed Fracture of the Skull

B. L. SHARMA, M.B.,
Sitamau (C.I.)

SOMETIME ago, just on the Holi-festival night, at about 8 p.m. scuffling took place between two persons—Bhil by caste, both being under the influence of coloured water.

One Bhil struck the other with an axe on the back, inflicting a deep incised wound. The younger brother of the wounded man with the aim of revenge ran to the residence of the culprit, and just at the entrance met the poor son of the culprit aged 8 years. Without any hesitation the poor child received a heavy blow from a thick club on the right side of his head and became unconscious on the spot. The child was brought to me with his wounded father by the police at about 11 p.m. and depressed fracture of the skull of the child was diagnosed. Immediate operation was advised to which the parents of the child did not agree and hence nothing was done in the night.

Next morning an influential man of the place made the parents agree to operation and preparations were started.

Now the most puzzling problem before me was that neither there was a Trephine, in the dispensary, nor any compounder to help me and sterilize the articles, nor there was any nearby well equipped hospital less than 200 miles (Ajmere) where to send the little patient nor the condition of the child and the father was fit to take such long journey. Hence I alone had to fight the occasion single handed with the poor equipments at my disposal.

After making the necessary preparations and sterilizations of the dressings and the available instruments I incised the fluctuating and throbbing spot on the head. The child was so unconscious that no anaesthesia—general or local was needed and used. On opening the skull I found the upper part of the right parietal bone crushed into pieces—some of which were embedded into the surface of the brain. With the aid of the point of a short pointed scissors pushed between two small crushed pieces of the bone, I could remove one piece and thus created a space for the others to be picked out with the aid of a hook and forceps one by one. With the aid of a magnifying lens I cleared the brain surface of all the embedded pieces. The exposed and injured portion of the brain was heavily throbbing.

Again the puzzling problem before me was that if any defect and shortcoming remained in all the preparations—sterilization and operation—done single handed, what the result will be in such a delicate case where the brain was exposed and handled, and if any sepsis took place in the brain will I be able to meet the serious condition which can easily prove dangerous and fatal? My intuition ordered me to be bold enough to do what conscience allowed. Hence without hesitation I painted the exposed part of the brain with pure Tr. of Iodine thoroughly together with the inner and outer surfaces of the wound and sutured the wound and dressed. The child came round after an hour. For about a week all went O.K. On the 8th day to my great disappointment I found that all the sutures had given way under the pressure of the throbbing of the brain which still was going on.

After sterilizing the wound with Tr. Iodine, I again sutured it with double silk and enough away from the margins, this time the sutures being continuous. To my great ecstasy the wound healed with first intention without any untoward happening and the child was discharged cured after ten days with all the stitches removed. Some pulsation

of the brain was still palpable. All through the period the child remained in the hospital well and jolly thriving well on the light nutrition.

For 15 months more I remained at that station during which period I had the chance of seeing the boy dozen of times and found him always O. K. in all respects.

The most wonderful thing to me was that the gap created by the removal of the bone pieces was no more soft but was as hard as other part of the skull as if the bone margins had grown and united so soon—(about four months)?

Will any kind reader oblige me with his experience and experiment that if a silver or gold leaf be placed on the gap created in the bone under such operations, what the result will be and can it be successfully done?

A Case of Severe and Persistent Hæmoptysis

S. N. DAS, L.M.F.,

Formerly M.O., M.I.M. Charitable Dispensary, Samra P.O.,
Hooghly Dist. (Bengal).

WHILE I was in Calcutta in May, 1941. I happened to treat a case of severe and persistent hæmoptysis (due to T.B.).

The patient was a robust young male of 20. When on the third day I was called in to see the case, the patient was coughing about an ounce of blood (mixed with slight frothy saliva) each time, every half an hour, inspite of usual treatment with Calcium (Sandoz), Haemostatic-sera (Neo Haemoplastin and Coagulen (Iba) and Morphine with a Calcium-Bromide mixture which his attending physician was doing. I added vitamin C injection (Cantan or Redoxon), Congo-red and Emetine Hcl (P.D.) and the above mixture with Glykeron (5p in each dose) with perfect rest of both body and mind. But unfortunately, inspite of all these the patient went downhill. Another doctor was called in (on the 4th day) for consultation, who advised A.P. which could not be done as X-ray at that moment was not possible to detect the bleeding lung. On the 5th day, with the help of another senior physician, we injected Anti Streptococcus serum $\frac{1}{2}$ Polyvalent, 25 c.c. (B.I.) (thinking it a case complicated with strepto-haemolytic infection) and prescribed the following:—

- R (a) Pot. Bromide gr. xx
Cal. Lactas gr. xx
Glykeron 5 p.
Hazeline gr. xx
Syr. Rose 5 i
Aqua Chloroform ad 5 viii such 4 i s.
(b) M. B. 693—4 tabs. daily 1 tab. every 4 hours.
(c) Isinglass (Nelson's)—3 i to drink frequently as directed.

The patient could not tolerate M.B. 693 tabs. (vomited and had intense nausea after the second dose) but yet we were glad to see the bleeding stopped completely from the 6th day—the day following. The patient had coughs (ineffectual) at times, yet he did not bleed again. He was given only the said mixture for a week more; thereafter, he made an uneventful recovery. Subsequently he was successfully treated for his lung condition with Calcium, Codliver oil, A. P. and Gold.

The point of interest in this case is: in cases of hæmoptysis which are severe and persistent and are not amenable to the usual line of treatment it should rouse suspicion of secondary streptococcus-haemolyticus infection and if present accordingly be dealt with promptly to save the life of the patient.

In conclusion, I express my grateful thanks to Drs. S. SAHA, M.R.C.P. (Edin.) and G. ROY of Calcutta and Drs. G. SANKAR, L.M. & S., D.P.H., D.T.M., and B. LAHARI of Sibpur, Howrah, whom I consulted regarding this case.

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Post Puerperal and Infantile Beri-beri

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BERI-BERI and multiple peripheral neuritis are often mixed up. Beri-beri is a specific form of multiple peripheral neuritis often associated with cardiac involvement. It is mostly due to a deficiency of vitamin B₁ and a toxin, the exact nature of which remains yet to be investigated. Multiple peripheral neuritis may follow as a sequela of any infectious disease, like enteric fever, pneumonia, influenza, malaria, or after dysentery, etc., with invariably no cardiac involvement. The cause of multiple peripheral neuritis is also a lack of vitamin B₁ in the body, perhaps due to the toxin of the disease of which neuritis is a direct sequela. Post puerperal neuritis is more of the nature of beri-beri; as there is invariably some cardiac involvement such as pain, precordial distress, palpitation, cyanosis, œdema of legs and anasarca. I am reporting below two cases amongst some of the type I had seen.

CASE I:—G. O. C., 22, F. came in with dyspnœa, orthopnœa, pain and precordial distress, puffiness of the face, œdema of legs, general anasarca, profuse sweating, cyanosis, tingling and numbness of extremities. She was unable to lie down and could just sit with her head resting on the bed side locker. She was very restless and could not lean back on a bed rest. The above signs and symptoms appeared gradually since 14 days. She delivered 3 months ago (the child reported as case No. 5 in the infantile beri-beri series below). Her pulse was scarcely perceptible and her heart was dilated. 30 m.g. of Berin (Glaxo) was injected at 10-30 a.m. and a Bromide, Digitalis, and Calcium mixture prescribed. The patient was much the same at 3-30 p.m. Another 30 mg. of Berin and 1/4 grain of Morphine were given hypodermically one after another. The patient was better and dozed fairly well at night. Second day, pulse rapid, restlessness and dyspnœa relapsed and the patient appeared to be drifting into the state in which she came in. 50 m.g. of Berin, Digitalin 1/100 gr., Morphine 1/4 gr. and Hyoscine Hydrobromide 1/200 gr. were given hypodermically. The patient was relieved, slept well and was sitting up towards evening. She could take some nourishment. The pulse and heart sounds were distinct. 30 m.g. Berin was again injected in the evening lest its effect should drop off at night. On the third day, 30 m.g. of Berin was given hypodermically and towards evening as there were signs of restlessness, Morphine 1/4 gr. was also given. This gave the desired effect, and the patient had a restful night. On the 4th day, 30 m.g. of Berin was repeated

hypodermically and from the 5th day onwards she was put on Berin by mouth, 3 m.g. tablets—six per day for a week. She was relieved of all the cardiac signs and symptoms and is now definitely convalescent. She is put on Yeast and Iron Strychnine mixture by mouth, and is now walking to the hospital to get her medicine and is almost alright. This woman was expected to die every day for 4 days and huge doses of Thiamin and Morphine undoubtedly saved her life.

CASE 2:—N., Female, aged 17 years, delivered 3 months ago, brought in a condition of breathlessness, palpitation, cyanosis, precordial distress, tingling and numbness, puffiness of face and slight general anasarca. 25 m.g. of Berin and 1/4 gr. Morphine given hypodermically. She is relieved of her distress and had a fairly good night's rest. Berin was injected in 25 m.g. doses daily for the next 4 days consecutively. She was able to walk about, relieved of all signs and symptoms with which she came in. She is now put on Bran tea, the cheapest form of Thiamin for the poor as she is unable to go in for Berin tablets or Yeast or Marmite. She made a good recovery and is now completely alright. There are many such cases I have come across, but have just given reports of a very severe case and a moderate one.

The pictures presented by these cases are not different from acute cardiac types of beri-beri seen in many places in the Northern Circars. In fact 35 years ago, when I was a medical student, one of my teachers used to wonder why these acute cases of beri-beri should die so very suddenly. The patients used to be seen in the morning in the hospital and their bodies in the anatomy department by the evening. Sometimes he used to do a partial post-mortem to find an acutely dilated heart with intense oedema of the heart muscle. The heart muscle was so waterlogged that fluid oozed out just as if from a cooked vegetable. Unfortunately synthetic vitamin B₁ was not known then and nitrites and diuretics were the rule.

INFANTILE BERI-BERI

Infantile beri-beri is claiming a high mortality in Narasapatnam and its neighbourhood. It is styled as 'Anasa' by the people and there are ever so many specifics by the village doctors, experienced grand-mothers, and neighbours from Masapatri leaf to Arka flower heads. Child after child is lost from persistent vomiting, diarrhoea, convulsions, or cardiac failure. Having seen a good many cases during the past one year, I made a short study. On enquiry, I learn there have been many deaths of children from the same disease in the same mother, some times even as many as four and five in succession at the same ages almost. The mothers all show unmistakable signs and symptoms of avitaminosis with preponderance of B₁ or B complex deficiency and sometimes of deficiency of Vitamin A too. Thus angular stomatitis, conjunctivitis, glossitis, and peripheral neuritis are the rule while xerosis and phrynodema are not uncommon. Many of the mothers also suffer from pregnancy anæmia. It is curious how some of them, if not all, have rightly believed that there is something wrong with their own milk which is the cause of their baby's illness. The position is made worse by the village doctors who are the first to handle them by a too rigorous dietetic regime for the mother avoiding all nutritious stuff and devoid of vitamins. Some of the mothers change to a wet nurse or to goat's or even ass's milk by past experience. There is no doubt that the diet of the mothers cases of infantile beri-beri has been grossly deficient in vitamin B

content, so that the child has been harbouring the deficiency even from intrauterine life and perhaps when its needs have grown about the second or the third month, signs and symptoms are apparent. Cases are more commonly seen in certain quarters and there are places where the women-folk, toil more than men and get the remnants of food left by the men-folk, for the men in these quarters come from a low grade society, addicted to drink and rule the women by the rod.

Of the many cases seen I give below short notes of 5 cases.

Case 1.—M. V., male, infant, aged 1 month and 20 days, was brought to me for bronchitis, inability to cry, loss of voice and greenish mucoid stools and vomitings. Mother lost a child from the same complaint an year ago. The mother suffered from pregnancy anæmia and had treatment for sometime with liver extract, Iron and vitamin B complex. The child was apparently alright for a month and after that developed the above signs and symptoms. He had also angular stomatitis and ulceration round the anus. He was rather drowsy and cyanosed, abdomen slightly distended, lungs full of rales and rhonchi. From the history it was taken to be a case of infantile beri-beri and 5 m.g. of Berin was at once injected and internally powders of M. & B. 693, Hyd. C. Creta Sacharata, Calcium Lactate and Soda Bicarb were prescribed. The child could not take any mixture and it was reported that he nearly suffocated to death when a diaphoretic mixture was attempted to be given. This had therefore to be stopped. The next day the child was better after the injection and the powders by mouth and Berin 5 m.g. hypodermically was again repeated. From the third day a 5 m.g. tablet of Berin was also added to the powders and a third injection of 5 m.g. of Berin was given on the 5th day and M. & B. 693 was stopped as the lungs were clear. The Berin powders with Calcium Lactate were continued for a week more by which time the voice cleared, the child's cry became normal, vomiting and diarrhoea stopped and he was taking feeds alright and gradually became quite well.

Case 2.—A Christian, male, infant, aged 1 month, brought in for intermittent crying, stretching and arching of the body, aphonia, vomiting after feeds, and slight cyanosis. Child put on Berin 5 m.g. with Hyd. c. Cret. and Calcium lactate by mouth for a week and he gradually recovered. The mother was advised diet rich in B₁ and Bran-tea freely. The child is since alright. The mother had signs and symptoms of B complex deficiency.

Case 3.—O. C. female child, 4 months, brought in with intermittent crying as if having a colicky pain, fretfulness, stretching of limbs and rigidity, occasional vomiting and inability to cry freely. The mother had signs and symptoms of B complex deficiency, viz., chelosis and neuritis. An injection of Thiamin 5 m.g. (Berin) was given with definite improvement next day. Berin was next continued by mouth for a week with recovery. The mother was advised to take Bran-tea and germinating pulses to get a sufficiency of vitamin B₁ both for herself as also for her child through her milk.

Case 4.—Twins, both girls, aged 4 months—one child was brought with ulcer cornea and aphonia. The child was marasmic with ulceration at the angles of the mouth and anus. The voice was hoarse and cry was feeble. The child was having diarrhoea and vomiting off and on, as also intermittent pain and stretching of limbs. The child first gave the impression of kera-tomalacia but on going into the history it was soon evident that it was a case of infantile beri-beri as the mother had lost 5 children from the same ailment year after year. The mother was thin and anæmic with signs and

symptoms of B. Complex deficiency. The other child was being fed on wet-nurse milk and she also showed signs and symptoms similar to the above but without an ulcer cornea. Both the children were treated with Berin internally 5 m.gr. daily for ten days and they improved satisfactorily. The mother was advised to take nourishing diet with a liberal amount of foods containing vitamin B₁. The children are doing well now.

Case 5.—This is a remarkable case of female infants aged 2 months—child of case No. 1 under post-puerperal beri-beri described above. This child was brought one afternoon cyanosed, sighing and gasping for breath. The child appeared to be dying. On enquiry it was gathered that the child was crying intermittently and vomiting persistently after feeds before she became suddenly cyanosed and gasping for breath. The mother was having signs and symptoms of avitaminosis B and A. Although much of hope could not be held out, Berin 10 m.g. was injected and it was marvellous. The child was normal by next morning. Another dose of 5 m.g. Berin was again injected and thereafter Berin powders by mouth for about 10 days 3 m.g. daily. The child is alright and healthy.

There are many more cases with similar signs and symptoms but I have not been able to keep notes of all of them amidst pressure of work. There are many mothers who give typical histories of loss of children with sudden attacks of sighing and gasping for breath and passing away before they could give them correct treatment. Infantile beri-beri in these parts is likely to be mistaken for bronchitis, acute gastro enteritis, marasmus, congenital syphilis, keratomalacia, rickets or infantile tetany. Improvement in the diet of the pregnant mother and education goes a long way to prevent it and reduce the infantile mortality. The cases are all from lower classes who are either too poor or do not know the method of correct living even though it lies within their means. Such cheap things like Bran-tea and germinating dhall can be adopted in their daily fare during pregnancy. I always preach to my patients the advantages and they follow my advice.

Summary and conclusions.—1. Some cases of post-puerperal and infantile beri-beri described.

2. The value of heroic doses of Thiamin as a life saving measures in both types is proved.

3. Morphine as a sedative to give rest to the exhausted heart is as much necessary as Thiamin in all cardiac types of beri-beri of which post puerperal is one. There need be no hesitation to push it till the patient is quite free from all restlessness.

4. Thiamin or better a B. Complex product like Yeast or Marmite should be continued orally with other tonics to maintain the effects of B₁ and prevent relapses.

5. All the mothers of infantile beri-beri had signs and symptoms of avitaminosis—marked B Complex deficiency with deficiency of vitamin A also in some.

6. A case of post puerperal beri-beri and her child with infantile beri-beri are described. Both of them were moribund and large doses of Thiamin and Morphine saved the mother and Thiamin the child.

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Protective Methods in Radiology

THE discovery of X-rays and radium opened up a new era in the treatment of diseases. But within a few years of the discovery of these, it was found that rays emanating from these were injurious to the health of the user. In the early days, many workers also, through ignorance or indifference, developed burns and dermatitis and some even lost their lives. As early as 1915, these accidents opened the eyes of the radiologists and following a discussion on protection for X-ray workers, the Roentgen Society formulated a set of suggestions regarding the safety measures. These, however, were not followed with the amount of strictness it deserved, with the result that there was a large increase in the above-mentioned accidents. From 1921 onwards, Committees were appointed both in Great Britain and in other countries to study the harmful effects of X-rays and radium and to devise protective measures. These Committees indicated the ways to ensure efficient protection against X-ray and radium gamma rays and also drew attention to the necessity for suitable working conditions. They condemned darkness and cramped space, and insisted upon natural lighting and ventilation. They also insisted that for all X-ray work, it was very necessary to surround the X-ray tube as completely as possible with adequate protective material. As lead had a high absorptive value and was easily procurable and workable, it became the common practice to place the tubes in lead-lined boxes. These were, however, heavy and clumsy, and hindered the radiologists in their work. Hence, efforts were made to reduce the size and weight, without sacrificing any of the protection.

These efforts led to the introduction of the so-called "self-protected" tube. Here, the main body of the tube was a chrome-iron cylinder to which glass was sealed directly. Surrounding the cylinder was a lead sheath of sufficient thickness to absorb practically all the primary radiation from the target, with the exception of the useful X-ray beam. Another unsatisfactory feature common to early X-ray tubes and high tension generators was the risk of electrical shock associated with their operation, since various parts of the equipment, working at several thousand volts, were often exposed. Accidents occurred to both workers and patients. Precautionary measures, such as the introduction of earthed metal guards, the reduction of the high-tension conduit system to a

minimum and the mounting of the overhead conductors as high as possible, were emphasised by the British Committees. A further advance was made in regard to high-tension protection by enclosing the tube and transformer in a single container and immersing them in oil. These combinations, however, were limited in regard to movement. The introduction of shock-proof equipment overcame this disadvantage. The tube was mounted in an earthed case and connected to the high-tension generator by means of shock-proof cables. This permitted the tube to be freely moved with respect to the generator. The shielding of high-tension parts has led to improvements in another aspect of safeguarding the health of X-ray workers. It had been observed that workers in X-ray departments complained of headaches and exhaustion and of irritable coughs and other inflammatory conditions of the respiratory tract. These effects were supposed to be due to nitrous fumes and ozone generated on points along with the high-tension system.

The First International Congress of Radiology held in London in 1925, and the Second Congress held at Stockholm and the International Commission appointed in 1937, have all been unanimous in their recommendations that it was necessary to have protection to the X-ray worker—the question of details being left to each country to elaborate. Most countries have, up to now, preferred not to take legislative measures. We should strongly urge on the various Governments to see that legislative safeguards are introduced to give protection to the X-ray worker. The recommendations have, however, been taken into consideration by manufacturers of X-ray equipment who have played an important part in the progressive improvement in conditions by designing equipment and departments in conformity with the Committee's proposals.

It will be important to know what quantity of ionizing radiations of all types can be tolerated without ill effects. It will be necessary to know what are the ill effects that such radiation can produce. It may be necessary also to know what quantity of each type of radiation, a person can receive continuously without suffering any ill effects. This quantity is called the "tolerance dose," and this must be known before any protective schemes can be formulated on a sound basis. A subsequent task in formulating the scheme is to try to express the particular tolerance dose in terms of a specifiable and reproducible biological standard, which in turn can be measured in terms of a physical unit.

In 1928, the roentgen (r) was accepted internationally as the unit of X-ray quantity. At present 0.2 r per day is accepted as the "tolerance dose." The American Advisory Committee, however, takes the view that 0.1 r per day is the "tolerance dose." At the Fifth International Congress of Radiology, held at Chicago in 1937, the definition of the roentgen was modified in such a way that it became a unit of gamma rays as well as X-rays. As regards the tolerance dose of radium gamma rays, the early evidence indicated that it was likely to be of the same order of magnitude as that for X-rays. Accordingly the International and British Committees state that "the evidence at present available suggests that a person in normal health can tolerate with impunity exposure to X-rays and radium gamma rays to an extent of about 0.2 international roentgen (r) per day or 1 r per week." In this respect, the American Advisory Committee have again chosen the lower tolerance dose of 0.1 r per day.

The present tolerance doses are expressed in terms of the radiation falling upon the surface of the body. MAYNEORD and others emphasised

that the total quantity of energy absorbed throughout the body of an irradiated person of "integral dose" as it is called, is of considerable importance, both physically and clinically. For a given dosage-rate of radiation (expressed in roentgens per unit time) incident upon the surface of the body, the dosage rates at various depths in the body will be greater, the more penetrating the radiation. It follows, therefore, that the integral dose per unit surface-dose will depend on the quality of the x-radiation. This integral dosage will naturally, therefore, vary according to the power of the machine and the quality of the radiation. It may therefore be necessary in the future to express the tolerance dose of x or gamma radiation in terms of the integral dose, measured in gramme-roentgens, rather than in terms of the surface dose, measured in roentgens. Alternatively, since in practice it will be the surface-dose which is likely to be measured, it may be necessary to adopt different values of the tolerance dose, expressed in roentgens, of various qualities of radiation.

When the tolerance dose for a particular type of radiation, say, X-radiation, has been established and is measurable in terms of a physical unit, the subsequent procedure in determining the protection in any instance is to measure the dosage rate of the radiation received at a specified point in terms of the unit adopted, to determine the transmission values of the radiation through various thicknesses of various absorbing materials, and finally to calculate the thickness of the chosen absorbent which is required to reduce the transmitted radiation received at the point in question to the tolerance dosage-rate. It is well known that x-rays and radium gamma rays are absorbed more effectively by lead than by any other common material. Hence lead or lead-impregnated materials, such as rubber and glass, have generally been used to secure protection.

When using X-ray equipment, steps must be taken to safeguard the operator against three types of radiation. In the first place, the tube itself must be protected in all directions other than that of the useful beam. Secondly, if the direct beam is pointed at the operator, as is often the case in screening a patient or object, a protective barrier must be placed in front of the operator. Thirdly, since all the objects which are placed in the path of the direct beam scatter the radiation in all directions, the operator must be protected against this secondary radiation, either by means of a protective barrier or by relying on remoteness from the scattering objects.

Much research work is being done in connection with: (1) the intensities of the direct beams in X-ray and gamma-ray work; (2) absorption of direct and scattered X-rays and gamma-rays in various materials; (3) thickness of lead required to reduce the radiation at any point to the tolerance amount etc.; and (4) the practical methods of diagnosing over-dosage with the help of film tests.

When answers to many of these questions are available, the lot of the present worker in radium and X-ray and the lot of the persons exposed to these radiations, will probably be much better and safer.

For a more detailed study, we would refer our readers to the very excellent article in the *British Medical Bulletin*—Vol. 4, No. 1, 1946, by W. BINKS, M.Sc., F. Inst. P., who has made a thorough study of the subject and deserves to be congratulated and from which we have taken the matter for this article. This Bulletin is devoted entirely to Radiobiology—Experimental and Applied—and we would ask our readers to go through this very valuable Monogram.

Gleanings From Medical Press

MEDICINE and THERAPEUTICS

Mercurial diuretics.—Intravenous injections of the mercurial diuretics may be given over a period of many months, even two or three years, without producing undesirable side-effects in the kidneys or elsewhere. In general it is best not to repeat the injections more frequently than every five days. Restriction of the fluid intake, avoidance of salt in the diet, and the administration of ammonium chloride 20 gr. in capsules three daily for forty-eight hours before each injection may enhance the effect and allow less frequent dosage. Patients may at any time develop idiosyncrasy to the drug; the lesser grades of sensitivity are manifested by skin disorders, such as urticaria or erythema, more severe reactions include purpura and enfoliative dermatitis. A positive patch test will prove the relationship of the drug to the eruption and indicate withdrawal. Allergy as indicated by cutaneous lesions, often passes off after withdrawal for a week or two, and the injections can then be resumed in smaller dosage or at longer intervals, without further sensitivity developing.

Accumulation of mercury in the body may occur if the dosage is too great or too frequent; it is to be suspected if poor diuresis is obtained in spite of the existence of edema. Digestive disturbances, such as vomiting or diarrhea, may arise, and occasionally hematuria or stomatitis. These disorders will generally preclude further use of mercurial diuretics. If mercurials have to be withdrawn, a trial may be made of diuretin 10 gr. three daily or of theophylline ethylenediamine 7½ gr. by intramuscular injection daily for three days. Failing these measures, mechanical drainage by Southey's tube may be undertaken.—*B.M.J.*

Erythema multiforme.—Erythema multiforme claims numerous and different aetiological factors, such as focal infection, or an association with inflammatory ringworm, or as representing part of the symptom complex of paradenitis venerea. The occasional pain and joint swelling have suggested a rheumatic or streptococcal origin. Most attacks run their course in three to four weeks. Routine treatment includes rest in bed and a sodium salicylate mixture. In the recurrent form, which is uncommon, a variety of dermatitis perpetiformis may be suspected. A careful search for an underlying cause, such as chronic focal sepsis, or gastro-intestinal or even renal disease, may be required.—*B.M.J.*

Cœliac disease.—There is usually no difficulty in deciding the diagnosis of cœliac disease. However, in some cases of steatorrhœa in infancy or early childhood it is important to exclude the possibility of congenital pancreatic deficiency (fibrocystic disease of the pancreas). The stools in the

latter disease are of a peculiar waxy appearance, and it may be necessary to determine the enzyme (lipase, etc.) content of the duodenal juice. In cœliac disease the enzymes are present.

The cause of cœliac disease is still unknown. In fact the whole problem of normal fat absorption is under close and elaborate reconsideration. One of the remarkable facts in cœliac disease is that quite a good percentage of fat is absorbed, and it is a strange thing that the loss of, say, 8 to 10 gr. of fat out of an intake of, say, 50 to 60 gr. a day should be related to such severe clinical disorder. Clearly some deeper processes are at fault in the intestinal tract, as suggested by the peculiar radiological appearances in the active disease—but in most cases the abnormality is not irreversible. Since the disease usually begins in late infancy or early childhood it suggests an early partial failure or exhaustion of one or more of the complex enzyme factors which probably contribute to fat absorption. Whether this is interlocked with abnormal requirements in vitamins of the B group is at the moment a matter of considerable interest and importance, and especially so in the light of the alleged clinical and biochemical success of folic acid in the treatment of the possibly allied disease, sprue. A clinical trial of folic acid in adequate dosage in cœliac disease is awaited with keen interest.

May et al. consider that the defect in the intestinal absorption of fat and carbohydrate in cœliac disease lies in the mucosa, and that the absorption of fat, intestinal motility, absorption of glucose, and the clinical course are favourably influenced by crude extract of liver and the vitamin B complex given parenterally. Paterson et al. while admitting that the malabsorption of fat and carbohydrate remains unexplained, claim satisfactory gains in height, weight, and general health in the early weeks on a similar course of therapy; and that if the treatment is sufficiently intensive and prolonged, there is a diminished loss of fat in the stools. A less dramatic improvement is noted in cases receiving only parenteral liver extract and B complex orally. However, in some cases, even with intensive therapy, the high fat content of the stools may persist, although there is a gain in weight and general improvement; some cases are refractory and fail to improve on the treatment. It appears from the study of these reports that no specific remedy has yet been discovered. Be it noted, however, that the folic acid content of the material used was not known, or it may have been present in insignificant amounts.

It should be stated that Ross obtained significant evidence that injection of liver extract increased dextrose absorption and

utilization, and it may well be that the therapeutic response is in some degree due to changes in this function rather than to a direct check on the steatorrhœa. Personal observations on the newer forms of treatment have not yielded the anticipated dramatic results, and we believe that it is most desirable, at present, to continue the low-fat diet regime as a basis, add adequate amounts of ascorbic acid, carotene, and calciferol, and give oral or parenteral B group and concentrated crude liver extract, 2.4 ml. intramuscularly each week. In the more severe cases it may be desirable to proceed to an 'intensive course' of parenteral therapy, but even so dietetic control is still considered necessary. Slow progress is often good progress in this disease. Inter-current infections, especially the inhibitory and possibly devastating upper respiratory infections, must be attacked urgently to avoid an acute intensification of the cœliac syndrome.—*B.M.J.*

Intramuscular injection.—Neuritis and paralysis of the greater sciatic nerve with such syndromes as drop foot, parasthesia, anæsthesia, and gluteal paresis, are frequently caused by careless intramuscular injection into the *gluteus maximus* muscles.

There are occasional sloughing and massive hemorrhages as a result of fluids being introduced into the superior gluteal artery.

If it is desired to use the gluteals as the site of introduction of intramuscular therapy the following technique is recommended:

1. Circumscribe the buttock of choice with an imaginary circle including the largest parts.

2. Bisect this circle vertically and transversally into four quadrants.

3. Prepare and inject into the upper and outer quadrant thus selected, well away from the midlines.

This practice is desirable for two reasons. First, the site so chosen is comparatively free from nerves and vessels, hence there is but little danger of the above sequelae. Second, this area is not a "pressure area" for the bed patient as it is well above the prominence and toward the side. An alternate (and preferred) site is the outer aspect of the thigh. The *vastus externus* muscle is large and well protected by its sheath, the *fascia lata*. It is free of any important nerves or vessels so that even the inexperienced person may perform any necessary injection with little fear of sequelae.

There is sufficient vascularity to permit absorption of any drug normally introduced intramuscularly. Because this is a comparatively large muscle, as much as 500 cubic centimeters of fluid may be (slowly) instilled.

The technique is simple. After the usual skin "prep," inject the needle into the middle of the outer thigh at right angles to the surface. A noticeable lessening of resistance at about 1 inch below the skin indicates the entrance in the muscle through the fascia.

If, by chance, the needle should strike the bone, little damage is done but the needle should be withdrawn about one-half inch before injecting the fluid.

One of the advantages of this method is that frequently repeated injections may be made into the muscle. By alternating from one side to the other and varying the sites on each side a few inches, both muscles may be used from just below the top of the thighs to within approximately 3 inches above the knee.

Only healthy muscles should be used no matter what site is chosen. Other sites suitable for intramuscular injecting include any large muscle away from nerves or vessels. The most often used in addition to the above are the pectorals and the deltoids.

—*Na. Med. Bull.*

Superior vena caval syndrome.—Hussey and his associates present 35 cases of the superior vena caval syndrome observed at the Gallinger Municipal Hospital and the Georgetown University Hospital. Aneurysm of the ascending aorta, bronchogenic carcinoma, malignant lymphoma and carcinoma with metastasis to the mediastinum are the main causes of the syndrome. Simple thrombosis of the superior vena cava is very rare. The principal symptoms and signs of the syndrome are edema and cyanosis in the upper part of the body, dyspnea and dilated veins indicative of the collateral circulation. Comparison of measurements of the venous pressure in the upper and lower extremities is the best method for establishing the diagnosis of obstruction of the superior vena cava. This may be the only practical method for recognizing that the vena cava is partially obstructed. The venous pressure in the arms is significantly higher than the femoral venous pressure. The arm-to-tongue circulation time may be normal in spite of the fact that the venous pressure is quite high. If prolonged, it is usually less than might be anticipated from the height of the venous pressure. Phlebography demonstrates that obstruction of the superior vena cava is present and, at times, localizes the point of obstruction. In 2 cases of aortic aneurysm the superior vena caval syndrome was produced by communication between the aneurysm and the superior vena cava. This condition should be suspected when the syndrome develops explosively in a patient with aneurysm of the aorta. The usual features of arterio-venous communication, such as thrill, bruit and an increase in pulse pressure, may not be present. X-ray therapy of radio-sensitive mediastinal tumours may decrease obstruction of the superior vena cava. Surgical exploration of mediastinum may be of value in cases in which lack of evidence of the cause of the obstruction holds promise of a remediable condition such as obstruction by adhesions.—*J.A.M.A.*

Treatment of hyperthyroidism by aminothiazole.—Thiourea and its related substance thiouracil are at present considered

to be the most active agents in the treatment of hyperthyroidism. Experiments have been made in France with aminothiazole, otherwise called Abadol. The efficacy of this agent was discovered by chance. On workers handling aminothiazole, a substance which was used to prepare sulphathiazole. Dr. Jeantet, of Lyons, noticed an abnormal development of goitres, but without ill effects and with signs of hyperthyroidism and lowering of the basal metabolism. It was sufficient to transfer the worker from one department to another to verify that the thyroid body resumed its normal size. On the contrary, the condition of hyperthyroid subjects working in the same factory was improved.

Marcel Perrault, Bovet, Progniet, Cournel and others explained their experimental and clinical studies on this substance at the meeting of the *Société médicale des hôpitaux de Paris*. Functional and subjective signs improve rapidly. Tremor is quickly suppressed. Tachycardia disappears and the pulse becomes normal. The weight increases by several kilograms. The basal metabolism is lowered to normal. The amount of cholesterol increases by 40 per cent., but the size of the goitre and exophthalmos does not appear to be influenced by this treatment. Aminothiazole has been administered to hyperthyroid patients in tablets of 0.1 gm. at a rate of four a day during three to four weeks. Improvement persists in some patients after suspension of the treatment; in others, relapses take place after some weeks. Aminothiazole has not to the present time produced any toxic accidents, as mentioned by various authors in England and in the United States in the treatment of hyperthyroidism by thiourea and thioracil.—*General Practitioner*.

Neostigmine in spastic paralysis from cerebral lesions—Kabat and Jones review the results obtained with neostigmine in 32 cases of chronic spastic paralysis or related conditions from cerebral lesions. Intramuscular injections were given six times a week. The dosage varied from neostigmine methylsulfate 1 c.c. of 1:2,000 solution (0.5 mg.) with atropine sulfate 1/200 grain (0.32 mg.) to neostigmine methylsulfate 3 c.c. of 1:2,000 solution (1.5 mg.) with atropine sulfate 1/100 grain (0.65 mg.). In a few cases neostigmine bromide was given orally to supplement the injections in a dosage of from 15 mg. three times a day to 45 mg. of neostigmine bromide with atropine sulfate 1/150 grain (0.5 mg.) three times a day. Neostigmine was administered after meals to diminish toxic effects. It has been admini-

stered to 11 patients who had developed hemiplegia from one to thirty-one years before. Improvement was observed in all and was retained after therapy was discontinued. Eight patients who had had hemiplegia for months were treated with neostigmine, and accelerated recovery of motor function was observed in some. Of 6 patients with the spastic type of cerebral palsy who were given neostigmine 3 children all showed significant improvement, while of 3 adults only 1 showed slight improvement. Three patients with the athetoid type of cerebral palsy all showed significant improvement from neostigmine therapy. Two children with mild ataxia, 1 of whom had cerebral palsy with severe stuttering and the other of whom had had a subarachnoid hemorrhage at the age of 1½ years, improved greatly on neostigmine. Definite improvement from neostigmine occurred in ataxia and facial paralysis in a patient who had had an acoustic neuroma removed more than three years before. Moderate improvement was observed in a disability of the upper extremity in a patient who had had a prolapsed cervical disk removed. One patient with pseudo-hypertrophic muscular dystrophy derived no benefit from neostigmine. Three patients with lesions of the spinal cord obtained slight or no improvement. Neostigmine decreases spasticity, thereby decreasing resistance to passive motion, increasing range of joint motion, relieving muscular pain and decreasing deformity. It facilitates voluntary motion, resulting in restoration of motions which had long been absent, increasing strength, improving coordination and diminishing fatigue. In addition, neostigmine has resulted in improvement in athetosis, ataxia, dysphagia, dysarthria, facial paralysis, motor aphasia and conscious proprioception from a variety of types of lesions of the brain. Improvement has been retained after therapy was discontinued. For maximal functional recovery, neostigmine therapy should be accompanied by exercise and muscle training. Evidence is presented that the locus of the therapeutic action of neostigmine in these cases is on the central nervous system.

Histamine therapy in multiple sclerosis.

Carter studied the effect of histamine therapy in 20 cases of multiple sclerosis. The technique of Horton (1944) was used, 2.75 mg. of histamine diphosphate in 250 c.c. of isotonic solution of sodium chloride being given intravenously at 30 to 90 drops per minute. Seventeen cases failed to show improvement. Three cases showed temporary improvement of less than six months followed by relapse. J.A.M.A.

SURGERY

Storage of skin autogenous grafts—N. N. Mathews, June 23, 1945.—Skin storage reduces the number of donor areas needed in multiple stage plastic repair. The patient is saved the pain of second or even third donor areas, which often cause more

pain than the field of operation during the first forty-eight hours. The duration of each operation, after the first, is also lessened by the time it would have taken to cut a fresh graft. A spare piece of skin is available in case the graft partially fails. Stored

skin can provide an experimental skin bank for investigating the problems of heterogenous grafting. A standard method has been developed for the storage of skin in a refrigerator which maintains a temperature of from 3 to 6 °C. The graft is folded with its raw surfaces opposed as far as its shape allows. It is wrapped in a piece of tulle-gras, and this in turn in a piece of gauze tightly wrung out of isotonic solution of sodium chloride. It is then placed in a sterile air-tight screw-topped glass bottle having a cubic capacity of 20 cc. It is held away from the small amount of fluid which collects in the bottle by insertion of a ring of rubber tubing or leadfoil for it to rest on. The power of growth of stored skin was proved by tissue culture of skin stored for eight days. Some of the refrigerated grafts were found to be sterile; others were infected with the normal contaminants of the skin, but despite the survival of these organisms on the grafts they had no harmful effects even after storage as long as seven months. The contact coagulum method of fixation has been employed to hold the stored grafts in position when their application at the bedside without an anesthetic has prohibited the insertion of stitches for pressure dressings. Mathews reviews the results of the first fifty grafts of stored skin. There is a possible relationship between refrigeration and increased resistance of the skin to infection.—*General Practitioner*.

The injection treatment of flat feet.—Injection treatment of flat feet and allied conditions involves: (1) injection of procaine hydrochloride through the sole of the foot into the painful site; and (2) continuation of moderate ambulatory activity while recovery occurs.

The value of this method first became apparent with the injection of procaine hydrochloride into the site of a simple sprain. Interruption of the vasomotor changes usually associated with the injury produced increased circulation of blood and lymph, and made immediate use of the part possible by eliminating the reflex arc of pain. The period of disability in sprains so treated was greatly decreased and patients were reported to have had much less pain during the entire period of recovery.

In those damaged bones and ligaments in which continued activity or weight bearing will not allow deformity to occur, this method of treatment definitely seems to accelerate healing time and does greatly shorten the period of recovery.

Most of the pain and soreness that persist in the long arch of a normal foot after excessive and unaccustomed exertion is due to overstretching and spraining of supporting ligamentous structures. This, of course, is doubly true in the foot that is structurally abnormal. It becomes painful more quickly than does a normal one and with less exertion, but the mere altered position of the bones in itself is not productive of pain.

Ligaments simply receive more body weight with each step than is normal and thus are more quickly and thoroughly stretched and sprained. This ligamentous stretching, then, may be considered as the basic and major cause of persistent pain in all over-used feet.

Ambulation during recovery does not imply continued over-use of the foot—that was the traumatizing force—but rather moderate activity which does not place enough strain on the ligaments to prevent their healing. Ligaments not only should heal, but should hypertrophy and become stronger during the process, a natural physiologic response to increased demands on the foot.

Treatment is indicated in cases of painful sore arches following exertion in: (1) structurally normal feet; (2) valgus or flat feet of mild or moderate degree; and (3) moderately relaxed feet.

Contraindications for this treatment are: (1) infectious metabolic and degenerative diseases of the feet; (2) gross trauma; (3) obvious deformities; (4) pes cavus (moderate or severe degree); and (5) pes valgus or planus of severe degree.

Most pain in pes cavus is not due to ligament stretch but to bruising of soft tissue under rigid metatarsal heads. Procaine injections, therefore, probably would be of no value. The valgus or planus foot condition of such degree as to be inconvertible by conservative orthopaedic measures is not a suitable case, since spraining of the supporting ligaments very rapidly will recur on resumption of heavy activity.

First injection is made just as soon after admission to the hospital as possible, regardless of the condition for which it is given. Using proper aseptic precautions, 15 c.c. of 2 per cent. solution of procaine hydrochloride is injected into the zones where the patient says he has pain and where examination has revealed tenderness on pressure. The tip of the needle reaches the bone and is withdrawn about 1/16 inch before injection is made. In most cases, the areas injected will be the plantar surfaces of the first and second metatarsocuneiform bones, the inter-cuneiform bone, the naviculo-cuneiform bone and talonavicular joints.

The patient, lying prone, now flexes his knees to a right angle while the bottoms of his feet are massaged with an alcohol sponge for 5 minutes. He then is required to walk up and down the ward 25 times, a distance of approximately one-fourth mile, after which he is free for the day.

Injections, followed by the walking period, are repeated every third day. Usually after the second injection the patient expresses a desire to return to duty. On discharge from hospital, the patient is recommended to have two weeks' light duty.

Valgus deformities are corrected the first day by the usual shoe alterations, supplemented with physiotherapy, muscle exercises

and instructions as to how to stand and walk. Adhesive strapping is not used because it limits natural movements of the foot.

Outpatients receive the same treatment except for omitting the walking period immediately following injection. They, too, are recommended for two or three weeks' light duty.

In actual trial on naval personnel, injection treatment as described has reduced, the number of hospital days per patient, decreased the number of cases medically discharged from service, increased the number of men returning to duty, decreased the number of recurrences and surprisingly enough even has decreased by about 50 per cent. the number of hospital admissions for foot pain.

Success of the injection treatment probably may be explained by actual beneficial effect on the foot and by psychologic effect on the patient.

It seems probable that the second factor is more important in those patients the authenticity of whose complaints might be questioned. This would account for the 50 per cent. decrease in number of admissions. Malingerers, misfits, dissatisfied men, and those having only a very minor degree of foot discomfort probably were the ones who did not come in when they heard of the new "needle" treatment. The remaining 50 per cent., then, must be those with genuine foot disability, men whose feet hurt badly enough so that the pain outweighed their fear of needles.—*General Practitioner*.

Cancer oesophagus.—(Ronald W. Ruven, F.R.C.S.). The age incidence varies from thirty to ninety years. The average age in a series studied by the author was 57.2 years. The disease is much more frequent in males than in females in the proportion of 4 or 5:1.

The early symptoms are often vague, slight, queer feeling in swallowing. A feeling of nervousness about starting to swallow, after starting food went down without any trouble; a feeling of nervousness in the neck, vague sensation about the neck as of something wrong; a feeling of cramp around the neck, food sticking in the throat while eating in a hurry, a feeling as of a lump rising in the throat. Sometimes cough is an early symptom. The late symptoms point to an advanced stage of the disease. There is dysphagia for solid food and later for liquids. Regurgitation of unaltered food occurs together with excessive salivation, pain in the chest and night cough. Hoarseness is usually present and there is progressive loss of weight leading to emaciation and cachexia.

A careful history including that of the present condition, past and family must be taken and a complete clinical examination made. Special attention is given to the lymph nodes of the neck, the condition of the chest

and liver. A plain X-ray followed by a barium swallow examination. In an X-ray picture there is marked irregularity of the affected segment with moderate dilatation of the oesophagus above the stricture. Spasmodic contractions are also seen. The types of growth are proliferative, ulcerative, stenosing plaque. The commonest sites are at the cricoid, bifurcation of the trachea and cardiac regions. There are two varieties seen on microscopical examination namely, squamous celled and columnar celled, the former being commoner than the latter.

One of the commonest complications to occur is perforation into an adjacent viscus. Other complications are mediastinitis, empyema thoracis, pericardium, oedema of the larynx, and abductor paralysis of the vocal cords.

In an early case surgical extirpation of the disease should be carried out. For other cases, high voltage X-ray therapy, intubation, dilatation with bougies, gastrostomy.—*Medical World*, June 21, 1946.

Testosterone propionate in mammary cancer.—Prudent (*Surg. Gynec. and Obstet.*, 80: 575, 1945) used testosterone propionate as a postoperative measure in treating 63 patients with mammary cancer who had previously been operated on. As controls, 64 cases are reported in which the breast had been removed by the same technique of radical surgery and in which testosterone propionate was not used. All 127 cases were operable. The results obtained, as gauged by the survival of three, four and five years, are about 100 per cent. better than those observed after operation only. This fact indicates that testosterone propionate exercises a protective or prophylactic action against recurrences of surgically treated mammary cancer. Very high doses of testosterone propionate, up to 175 mg. a week, may and should be used after operations for mammary cancer, and this treatment should be continued through many years. The principal criterion for the selection of the dosage is the grade of histologic malignancy of the tumour. The side effects following this therapy are of secondary importance. They consist of virilization and of disturbances of menstruation. In patients between 40 and 50 years of age who are still menstruating, early cessation of menstruation is often observed.—*General Practitioner*.

Treatment of causalgia.—The 100 patients with causalgia treated by Rasmussen and Freedman were Chinese soldiers who had been wounded in the Burma campaigns. The upper extremity was involved in 82 and the lower in 18 patients. The major nerves of the extremity were involved in 81 of the 100 patients and in 10 there was injury only to bones or joints. There was injury only to soft tissues in 5 patients and injury to cutaneous or digital nerves in 4. The characteristic pain appeared within six hours after the injury in 48 cases and within one to three days in another 23. In a few cases

its appearance was delayed up to eight weeks. Fifty-five of the patients described the pain as hot or burning in character, while the remaining 45 did not associate any sensation of temperature with the pain. Since the rest of the clinical picture of these 45 patients was so nearly identical with the others, it seemed obvious that the same pathologic process was involved. Diagnostic paravertebral injection of procaine was done in 91 of the patients, producing complete temporary relief of pain in 73, great relief in 16 and no relief in 2 of the patients. From the therapeutic standpoint, paravertebral injections of procaine produced complete and permanent relief of pain either immediately or within four weeks after the injections in 13 of the 91 patients. Moderate relief of pain was produced in 32 patients, while in the remaining 46 the relief of pain produced by the injections alone was inadequate. Postganglionic operations for causalgia of the arm resulted in immediate complete relief of pain in 5 patients and complete disappearance of all pain within one month after the operation in an additional 5. There was gradual disappearance of the pain several months after the operation in 4 patients, and in the remaining 7 patients in this group of 21 some residual pain persisted throughout the follow-up period. Preganglionic operations for causalgia of the arm were done in 14 patients with immediate complete and permanent relief of pain in 13. The remaining patient in this group obtained great immediate relief followed by complete disappearance of the residual pain during the first month after the operation. The results of sympathectomy for causalgia of the leg were not quite as satisfactory as for the arm. Four of the 5 patients in this group continued to have some residual pain.—*J.A.M.A.*

Actinomycosis.—With special reference to its pathogenesis, its treatment and its cure with penicillin.—ANDRE BRUWER, CAPT. S.A.M.C.

Lord defines actinomycosis as follows: "An infection, characterized by the usual presence of lesions composed of abundant granulation or connective tissue surrounding multiple areas of abscess formation and by the presence within the abscesses of the specific infecting agent in compact colonies composed of branching, Gram-staining, filamentous, anaerobic organisms with radially disposed, club-bearing filaments."

To the definition might also be added the fact that the disease is due to an organism called *Actinomyces*.

INCIDENCE.—The disease has a world-wide distribution.

No race is exempt.

The sex incidence seems to average about 80% in males and about 20% in females.

Age.—About 60% of cases appear to occur between the ages of 20 and 30 years i.e., young adults are chiefly affected.

COMMUNICABILITY.—Very few cases in the

literature suggest that the disease is directly communicable from one person to the other.

PATHOGENESIS.—The literature usually makes reference to two different theories regarding the etiology and pathogenesis of actinomycosis. The one is the *exogenous* theory propounded by Bollinger and the other is the *endogenous* theory attributed to Wolff and Israel. The former states that the infection originates from pathogenic organisms outside the body; the latter that the infection originates from pathogenic organisms inside the body usually from inside the oral cavity or the intestine.

COMBINED ENDOGENOUS AND EXOGENOUS THEORY.—The literature on the merits of the discussed theories suggests that the pathogenesis of the disease may lie in a combination of the two. The very pleomorphic attributes of the organism would tend to support this suggestion. Statements supporting such a contention have been made at various times, e.g., by Foulerton who says: "... when not living as parasites on man and other animals, the *Streptothrichæ* (*Actinomyces*) are essentially either saprophytes living in the soil, or parasites of higher forms of vegetable life."

CLASSIFICATION.—No ideal classification of the actinomycosis-like disease seems to have been arrived at. Colebrook points out that the term has been used for a number of diverse conditions. He "arbitrarily" divides these conditions into:—

(a) Those where there are granules that bear clubs.

(b) Those where there is infection due to mycelial organisms, either without any granules, or having granules but no clubs.

Under those conditions where there are granules with clubs, Colebrook groups the following conditions:—

(i) *Human actinomycosis*—due to *Actinomyces bovis* (Israel).

(ii) *Madura Foot or Mycetoma*—due in many cases to an aerobic *Actinomyces*.

(iii) *Actinobacillosis*—this condition used to be thought the same disease as actinomycosis in man; it occurs in cattle under the name "woody tongue." It was only when Ligniere and Spitz (1902) pointed out that the condition was due to a gram-negative bacterium, that the condition was differentiated from human actinomycosis. The organisms do not form mycelia, but in certain media they may take on filamentous growth.

(iv) Certain staphylococcal infections of cows' and pigs' udders, producing granules and clubs but no mycelial elements. The granules are dense aggregations of staphylococci. Colebrook knows of one case in a human being where a staphylococcal infection showed club-formation.

(v) The *B. pyogenes* infections of veterinary bacteriologists which Magnusson believes to be potential club formers.

The non-club-forming mycelial infections Colebrook calls the *Streptothricoses*.

SYMPTOMS.—There are generally three

types of the disease, named according to the anatomic distribution:—

- (i) Cervico-facial, 60% } These figures are
(ii) Thoracic, 14% } from Sanford
(iii) Abdominal, 18% } and Völker's
review of 670 cases.

The symptoms of the disease naturally depend on the functional importance of the part involved, and it is therefore expected that the prognosis gets progressively worse in the following order: cervico-facial, abdominal, thoracic. Again, in the cervico-facial type, the prognosis will also depend on what part of the cervico-facial distribution is involved, e.g., those cases involving the region of the angle of the mandible (not bony involvement) have a good prognosis, whereas those cases involving the orbit may have a very grave prognosis if spread occurs to the base of the brain.

Symptoms will also depend, to some extent, on the stage of the disease. Two stages are generally recognised, the first preceding the second, but the two at times being co-existent:—

(i) *Stage of induration*:—This usually lasts 3 to 4 months before softening occurs. But it may be much more rapid (few weeks—acute type) or much longer (a year or more—chronic type).

(ii) *Stage of softening*:—Softening and ulceration will take place in one area, then heal, and then occur in another area.

Symptoms may last for as short a time as a week, or as long a time as 15 years.

The symptoms of actinomycosis are confused with a great many conditions, as is seen in most case reports—in the vast majority of cases the condition is at first misdiagnosed, until the case is referred to someone who considers actinomycosis.

Cervico-facial cases:—These cases may occur in any of the separate regions or organs of the face and neck, e.g., the eye, the lacrimal apparatus, the middle ear, etc. But roughly these cases are about equally divided between the jaw, temporal region and neck.

The infected area becomes indurated and wooden in consistency; and the overlying skin becomes dark red or purplish in colour about two weeks before abscess-formation; the surface is lumpy and uneven. The colour of the skin indicates the great vascularity of the region at this time. The indurated area softens after a variable time, in some places, breaks down, and forms discharging, secondarily infected sinuses. These sinuses heal at times, only to form in other places where indurated tissue breaks down. The spread is in the subcutaneous tissues, and therefore healing scars pucker the skin deeply.

Pain in these cases is not usually a very prominent symptom, except sometimes when the disease attacks the parotid gland region, when they are in their "subacute" stage.

Swelling on the lingual side of the mandible is very rare and it is very un-

common for sinuses to open on the inside of the mouth. According to the endogenous theory, the cases usually start on the inside of the mouth, and then the condition works its way extra-orally. When the swelling is in the masseter region, its anterior border is most easily palpated through the mouth, when it will be felt as a definite ridge just anterior to the masseter. Swelling may be so great in the neck as to cause dyspnoea.

The lymph nodes are not involved, unless it be due to secondary infection. The lymphoid tissue seems to have a peculiar immunity to the disease.

The general health usually remains from fair to well. But many of these cases worry a great deal.

The main symptom in these cases is usually trismus, and this may be so severe that the patient cannot open the mouth at all.

Abdominal Cases. These cases usually commence in the appendix. The condition may start exactly like an ordinary acute appendicitis, or it may start in a chronic way as an indefinable lump in the right iliac fossa. If in either case an operation is performed, it will be found that the wound does not heal, but continues to discharge, until at some time the histological evidence of actinomycosis is found in the discharging pus.

From this area any one of the pelvic or abdominal viscera may be involved, either by continuity or by contiguity. In some cases the thorax may even be involved from the abdomen.

Thoracic Cases:—This is the most fatal form of the disease. There may be cough, sputum, dyspnoea, slight fever, loss of weight and strength, night sweats and emaciation. In about 50% of the cases there is blood in the sputum. Pleural effusions are fairly common, and may be massive as seen on X-ray. Extension through the pleura and the skin causes sinuses. Extension takes place by continuity and contiguity even through the diaphragm and into the abdomen. Sometimes the large vessels in the chest are involved and there may be a hamatogenous spread.

GENERAL AND HISTOLOGICAL PATHOLOGY.—The principle features of the disorder of actinomycosis are as follows:—

1. A granuloma is produced by an acute and chronic infectious process that progresses at the same time around the ray fungus.

2. The granulation tissue is composed of polymorphonuclear and mononuclear cells surrounded by a vascular zone of epithelial cells, multinucleated giant cells, eosinophiles and plasma cells. This zone is in turn bounded by dense fibrous connective tissue. The vascularity of this granulation tissue is of great importance from the standpoint of operative treatment. Wangensteen also stresses this vascularity which may sometimes be alarming, but which is easily controlled by gentle pressure with a gauze pack.

3. Along with the dense fibrous tissue

reaction, there is a simultaneous degenerative process due to the invasion of connective tissue by the fungus. The former has a tendency to wall off and to heal the infection, and the latter to advance the process by supuration, degeneration and liquefaction.

4. When softening takes place, there is an extensive lipid degeneration with production of abscesses, cavities and intercommunicating pus channels filled with purulent material containing yellow granules (sulphur granules).

5. Sinuses form and discharge purulent exudate.

6. The healing process is characterised by the formation of large nodules of cellular fibrous tissue.

7. Extension probably takes place by the mycelial filaments breaking off and being carried into adjacent tissue by wandering macrophages.

It seems that the virulence of the organism determines how the condition will commence i.e. a virulent organism will commence by the formation of soft vascular granulation tissue—the acute variety—and the less virulent organism will cause the condition to start by the laying down of dense fibrous tissue—the chronic variety.

The Sulphur Granule. This is the characteristic macroscopic finding often encountered in the uncontaminated actinomycotic pus. When stained with Gram's stain and counter-stained with dilute carbol fuchsin, the centre of the granule will show a mycelium of tangled and wavy filaments, many showing true branching. At the ends of some filaments, at the periphery of the granule, are the characteristic clubs. There is considerable variation in the manner in which the filaments and clubs retain their stain. "In some the protoplasm is not uniformly stained, but shows light and dark violet stained areas, while in others the protoplasm shows a gram-positive granular appearance."

The granules are lobulated or spherical, and they vary in size from minute particles to about 1 mm.

The Clubs:—Colebrook draws attention to the fact that 40 years ago it was shown that clubs were formed around a mass of dead tubercle bacilli implanted into living animal tissue.

Diagnosis:—Actinomycosis should be considered in every case where a sinus forms which does not respond to sufficient of the usual treatment. In every case of acute cervico-facial swelling actinomycosis should be considered, especially as it has often shown itself to present as acutely as an acute pyogenic abscess. In every case of chronic cervico-facial swelling, with induration, trismus, lack of particular pain, purplish discoloration of the skin, especially following tooth extraction, and without particular systemic symptoms, and not responding to the usual treatment for inflammatory swellings, one should become suspicious of actinomycosis. When this latter

process in the cervico-facial region forms abscesses which break down to form sinuses that discharge sulphur granules in their pus, and refuse to heal following routine treatment, the diagnosis of actinomycosis should be obvious.

Apart from the diagnosis being made from the clinical syndrome the following confirmatory aids have to be resorted to; the finding of macroscopic and microscopic sulphur granules containing the Gram positive mycelial core ringed in by clubs; cultivation of the organism; biopsy if necessary.

TREATMENT.—*Potassium Iodide*. It seems that few drugs have lived on their laurels as has potassium iodide. It has certainly been used more than anything else in the treatment of actinomycosis.

The drug has been said to act through its lysis of granulation tissue.

The dosage varies with the physician, but a usual method of oral administration is to start with 3 drops of the concentrated solution t.i.d., increasing the dosage gradually until about 120 daily are being taken. But some people give heroic doses of 300 to 400 drops per day, and even much more.

Vaccine Therapy.—Various authors have had favourable results at times in cases where vaccines had been used in conjunction with other methods. Either autogenous or stock vaccines are used. A dosage of 5 to 10 million mycelial fragments are given at 5-day intervals, after desensitization has been carried out.

Surgery.—Randal feels that surgical therapy should have precedence over all other forms of therapy. Colebrook considers surgery of paramount importance and attributes the good effect to the free flow of lymph which is obtained. He also quotes Sir Almroth Wright's method of packing the wound with hypertonic saline so as to promote a free flow of lymph. Colebrook prefers curetting the area with gauze as against the use of a sharp instrument.

Sulphonamides.—Many cases of cure by means of one of the sulphonamides have been reported in the literature. Billington, for instance, describes the case of cervico-facial actinomycosis involving the mandible, which showed triumphant improvement on sulphapyridine in 19 days. He used a small dosage method, which, he points out is in contradistinction to the Medical Research Council's Memorandum No. 10 on "The Medical use of Sulphonamides," which says that large doses should be used for actinomycosis.

The foregoing covers the main methods of treatment that have been used, but a great variety of less successful methods have been tried, of which a few will be mentioned:

Silver Nitrate.—This has been used locally, and per injection in abdominal cases.

Cinnamyl and Muriate of Cocaine in alcohol has been used twice weekly per local injection.

Alcohol.—Parenchymatous injections.
Ethyl Iodide.—Used by inhalation in thoracic cases.

Hydrogen Peroxide.—This is reasonable medication in local dressings in view of the anerobic nature of the organism.

Colloidal Gold.—This has been used by local injection.

Raising B.M.R.—Attempts to raise the B.M.R. by the intravenous administration of glucose with one unit of insulin for every 3 Gm. of sugar.

Colloidal Copper.—This has been given by the intravenous and intramuscular routes with apparent success in some cases.

Salvarsan.—This has been tried with doubtful result.

Formalin.—This has been used by local injection into the swelling but is so painful that it requires an anesthetic and has no obvious advantage over other methods of treatment.

"Yatren" or Chiniofon.—Gradually increasing doses of a 5% solution intravenously starting with 5 c.c. and increasing to 100 c.c. on successive days, has had some favourable results.

Phenol.—Swabbing with pure phenol and then neutralizing with alcohol.

Methylene Blue.—Has been given by local or intravenous injection.

Oil of Eucalyptus.

Potassium Permanganate.

Actinomycine.—A filtration product of broth cultures of the organism has been used but without any particular advantage.

Apart from any of the above methods of treatment, it is also essential to keep the patient's general health and morale up to the mark.—*Clinical Proceedings of the Jour. of Cape Town Post-Graduate Med. Assn.* (Vol. 5, No. 2, April 1946).

Cancer of the stomach.—Cancer of the stomach is one of the most frequent forms of the disease and accounts for a large percentage of the annual deaths from cancer. The stomach is the most frequent site for cancer in the male, and in the female it ranks third after the breast and reproductive organs. It is an unfortunate fact that many patients present themselves for treatment when the disease is inoperable. The only hope for the patient with cancer of the stomach lies in the early performance of either partial or total gastrectomy with removal of the regional lymph nodes.

Symptomatology.—The importance of recognising the early symptoms of carcinoma of the stomach is greatly stressed so that the prognosis may be improved. Early diagnosis is the key for successful treatment. In the past the symptoms described are those which supervene late in the disease and are mainly those occasioned by complications which occur, such as ulceration, pyloric obstruction or secondary infection.

Duration of Symptoms.—There are two main groups of patients which can be distinguished.

Group I.—Patients with a relatively short history.

Group II.—Patients with a relatively long history. These patients have suffered

for a long time with a gastric ulcer and the symptomatology changes in character with supervening malignancy developing in the ulcer. It is computed that about 10 per cent. of benign gastric ulcers later develop malignant characteristics.

The following are important early symptoms of carcinoma of the stomach and their presence demands a complete investigation of the patient.

Anorexia.—A gradual loss of appetite which is progressive until there is repugnance for food.

Malaise.—The patient complains of becoming easily fatigued and there is a lack of feeling of well-being. He is conscious that something is wrong although he is unable to describe his feelings with accuracy.

Abdominal discomfort.—An early symptom is the presence of vague abdominal discomfort which is experienced in the upper half of the abdomen. There may be remissions from discomfort for several months. Later, with the progress of the disease, constant abdominal pain supervenes.

Nausea.—A common early symptom and is frequently experienced early in the morning before food is taken. Sometimes it occurs after meals.

Change in bowel habit.—In a patient of cancer age this particular symptom should always be fully investigated and a complete examination of the gastro-intestinal tract carried out. In cancer of the stomach constipation may be an early symptom and later, in the presence of achlorhydria, diarrhoea develops.

Pallor.—A slight degree of pallor is often seen in the early stages of the disease. A pronounced degree of pallor or lemon tint indicates that the condition is far advanced.

Loss of weight.—In many patients this is gradual and persistent in the early phase.

The late symptoms may be described as follows:

Abdominal pain.—In a majority of patients there is pain of varying severity, dull aching in character and is usually constant. It may occur during the night. Frequently the pain is unrelated to the taking of food and is situated in the epigastrium, to the right or left of the mid-line according to the situation of the carcinoma in the stomach. The pain may simulate that experienced in peptic ulcer and sometimes it occurs in sharp paroxysms like biliary colic.

Vomiting.—This is usually seen when the neoplasm is situated in the region of the pylorus with consequent stenosis and obstruction. There may be varying degrees of dilatation of the stomach and the vomitus is composed of large quantities of stagnant food which has collected for several days in the stomach. Vomiting may recur at night in order to relieve the pain of gastric distension.

Hæmatemesis.—Erosion of blood vessels due to an ulcerating carcinoma causes hæmorrhage of varying degrees into the stomach. A severe hæmatemesis sufficient to cause the patient's death may occur. The vomiting has the well-known "coffee-ground" appearance.

Melæna.—This may be a late symptom giving the stools a tarry appearance.

Cachexia.—Always a late manifestation of cancer.

Diarrhoea.—Usually accompanies achlorhydria and is sometimes present as a late symptom.

Tumour. A palpable carcinoma of the stomach does not necessarily indicate that the disease is inoperable. It does mean that it is well established.

Investigations. After a careful history, including that of the present condition, family and past illness, has been elucidated a complete physical examination is made. Special attention is given to the left supraclavicular and left axillary region for enlarged lymph nodes. The abdominal examination is conducted with careful attention to the presence of an abnormal swelling or an enlarged liver. The presence or absence of ascites is noted. A rectal examination is never omitted to determine whether pelvic deposits of growth are present and in the female a vaginal examination should be performed with special reference to the ovaries.

Radiological examination. This is carried out after giving the patient a barium meal and valuable evidence is furnished by the screen examination. Sometimes a carcinoma at the cardiac end of the stomach may escape detection or a carcinoma with a patulous pylorus may lead to an error in diagnosis.

Gastroscopy. This form of investigation is valuable in all forms of abnormalities of the stomach and it may lead to the detection of an early neoplasm.

Gastric analysis. Achlorhydria is not pathognomonic of carcinoma of the stomach. It occurs, for example, in pernicious anemia and certain affections of the gall bladder. When the patient's vitality is poor, hypochlorhydria is present in certain benign lesions of the stomach. Further, hyperchlorhydria is not infrequently present in cases of carcinoma of the stomach. The presence of blood in the resting contents of the stomach is valuable evidence, but it must be remembered that this may also be found in chronic peptic ulceration.

Examination of the stools. The presence of occult blood is an important aid in the diagnosis.

Examination of the blood. With the progress of the disease marked secondary anemia develops; sometimes the anemia is so profound as to simulate pernicious anemia.

Treatment.—When a patient is considered to be suffering from cancer of the stomach an exploratory laparotomy should be performed without any delay, unless the condition is judged to be quite hopeless on clinical grounds.

Gastrectomy.—When the carcinoma involves the cardia a total or subtotal gastrectomy is required. In growths situated more distal in the stomach partial gastrectomy after the method of Polya should be performed. In patients who are in poor condi-

tion and possessing marked subnormal nutrition operation may be performed in two stages; a gastro-jejunostomy is carried out on the proximal side of the neoplasm and at a later date resection on the stomach is done.

Palliative treatment.—Palliative operations such as gastrojejunostomy, gastrostomy and jejunostomy are sometimes indicated. The usefulness of gastrojejunostomy is limited as the relief is only partial and the average expectation of life is not more than about seven months. A palliative partial gastrectomy may be of temporary benefit even though there are metastases present; the condition of the patient improves and life may be prolonged for a year or more. Ronald W. Raven, F.R.C.S.—*Medical World*, July 5, 1946.

Early rising after operation.—At a recent meeting of the Swedish Association of Surgeons, several speakers referred in appreciative terms to the growing practice in the Scandinavian countries of getting patients out of bed early after operations, even the most radical. In an address on "Three Years' Experiences of Early Rising after Operation," S. Lindvall admitted that the recent remarkable reduction in the thrombosis-embolism incidence after operation could not be exclusively attributed to early rising, as appreciable gains had been achieved also by other measures, such as chemotherapy, the exact control of the water balance in the body and advances in the prevention of shock. But with this reservation he put forward the following statistical evidence: In the period 1935-1944, previous to the introduction of systematic early rising after operation, 13,665 patients underwent various operations in his hospital. Among them there were 2, whose deaths were unmistakably due to post-operative embolism, and 6 others whose deaths were almost certainly due to this cause. Between the beginning of 1943 and Oct. 1945, 6,318 patients underwent operation. Not one of them died of embolism, and there was only one case of thromboembolism ending in recovery. In the first period the post-operative mortality was 1.79 per cent. (244 deaths) and in the second period it was 0.73 per cent. (46 deaths). When Lindvall first began to practise early post-operative rising he found his patients and his staff prejudiced against it, and only after 2 deaths from pulmonary embolism in rapid succession did opinion veer definitely round, and he was able at the beginning of 1943 to enjoy the co-operation of all concerned in his new measure.

The patient must not have been confined to bed before operation, and he gets out of bed a day or two after all abdominal operations, including operations for hernia and on the external reproductive organs. As a rule the patient sits up on the day after an operation on the prostate. In 1941 the average duration of hospital treatment was 15 days; in 1945 it was only 9.9 days. The general condition of the patient who has been in bed only for a day or two is much

better than when he has been confined to bed for several weeks. He is less liable to contract pneumonia and bronchitis, and his digestive system functions well as a rule without artificial aids. He is also less liable to atrophy of his muscles.

In Lindvall's experience it has become exceptional for a patient to remain longer in hospital than one week after a resection of the stomach or an operation on a myoma or the gall-bladder. The average duration of stay in hospital of 8 patients treated by primary resection for perforating ulcer during the past 3 years was 8.4 days. The average

stay in hospital after an operation for hernia was about 5 days, and it was about 9 days after a prostatectomy. It might have been expected that the increased turn-over of patients in hospital would give the staff more work. But not so, for the patients are better able to look after themselves, and do not require such measures as enemas to the same extent as before. It has been objected to this reform that it may increase the frequency of relapses after operations for hernia, but the experience of several Scandinavians does not bear out this forecast.

—*Clinical Journal, July—Aug., 1946.*

OPHTHALMOLOGY

The precursors of melanin as virulent nucleoproteins in the eye, with particular reference to sympathetic ophthalmia. Klin. M. f. Augenh., 1942, v. 108, Sept.-Oct., pp. 605-614. Spyratos, S.

The pigment in all pigment cells consists of protein granules to which the melanin is firmly bound. In the earliest stages of pigment formation these granules are colorless and consist of undifferentiated protein. Later they are transformed into black melanin granules under the influence of a pyrocatechin derivative in the presence of an oxidative ferment which is found in the immature protein granules. In the eye this transformation normally takes place only in a certain period of embryonic life. When the pigment genesis is complete, the melanoblasts become chromatophores and normally continue in this function for life. Under abnormal conditions, melanophores may react with degeneration and dissolution of the cells and with pathologic proliferation of pigmented epithelial cells. Atypical embryonic melanoblasts form, in which abundant immature atypical protein granules, the precursors of pigment, occur, not only within the cell itself but also in the surrounding tissue. The question arises whether these protein granules are able to interfere with the biologic balance of the melanophores in the healthy fellow eye, and eventually lead to the formation of the same immature protein bodies. The author found a direct relationship between these granules, on the one hand, and the epithelioid cells and infiltration nodules of sympathetic ophthalmia, on the other. Under pathologic conditions *in vivo* and in tissue culture *in vitro*, the pigment granules are engulfed by phagocytes as foreign bodies. The behaviour of pigmented epithelial cells and of the chromatophores of the choroid is different. The latter settle around the blood vessels in a characteristic way. Under pathologic conditions they are able to invade blood vessels. Free pigment granules are then found not only in the lumen but also in the endothelium of the vessel. Pigment epithelial cells may pass the barrier of the lamina vitrea and proliferate in the uvea and even cover organized masses of exudate. In histologic preparations impregnation with silver makes mature pigment granules as well as their colorless precursors visible. With this staining method a direct relation-

ship between free pigment granules and the epithelioid cells found in sympathetic ophthalmia could be established. The granules are found abundantly engulfed by the epithelioid cells. The author conducted numerous experiments with tissue cultures to clarify the reactions of the cells of the organism to free pigment granules. The pathogenic effect of the melanin granules and especially of the precursors of melanin in sympathetic ophthalmia seems to be firmly established. They act as virulent proteins like microbes or viruses. (6 photomicrographs, bibliography.)—A. J. Ophthalm.

Nicotinic acid in ophthalmology and the principle of minimal elective vasodilation. Arch. d'Ophth., 1945, v. 3, No. 2, p. 197. Gollois, Jean

According to Gollois, the use of a vasodilator always carries a certain risk. Acetylcholine has frequently produced elevation in tension in mildly glaucomatous eyes. He has used nicotinic acid with satisfaction. It gives a vasodilation without modification of the arterial pressure and can be used either by mouth or by intramuscular or intravenous injection. He reports a series of 29 patients with chronic glaucoma in which the use of the drug resulted in significant lowering of the tension and increase in visual acuity and fields. In nine patients results were unfavourable. The favourable results were obtained in glaucoma at onset, glaucoma of slow evolution, and glaucoma with only slightly elevated tension. An individual oral dose of 30 mg. was usually satisfactory. General and local reactions could be expected within an hour. The treatment was given daily for three days of each week. Occasionally it was necessary to reduce the doses to 20 or even to 10 mg.—A. J. Ophthalm.

How do the hormones of the posterior lobe of the pituitary gland affect glaucoma. Klin. M. f. Augenh., 1942, v. 108, Nov.-Dec., p. 737.

Recent investigations have shown that the regulation of the intraocular pressure depends partly upon the function of certain endocrine glands. Many different endocrine preparations, among them meropitan, which is an extract of the posterior lobe of the pituitary, have been used successfully to

reduce the intraocular pressure in simple glaucoma. The author observed more than 100 cases of glaucoma, 45 of them simple glaucoma, 3 acute inflammatory glaucoma, 24 chronic inflammatory glaucoma, 15 absolute glaucoma, 4 juvenile glaucoma, and 5 postoperative glaucoma. Some of them had not been treated with any miotics previously, in others there had been continuously high tension in spite of the use of miotics. He used pituitan, which contains all the hormones of the posterior lobe, oxytocin, and vasopressin. The extracts were administered either sub-conjunctivally

or intramuscularly. Oxytocin and vasopressin appeared to be ineffective. In 24 cases decrease of the tension was obtained, in eight a very sudden drop. Absolute glaucoma, juvenile glaucoma, and acute inflammatory glaucoma did not respond to posterior-lobe extract. Simple and chronic inflammatory glaucoma responded best, but the response was usually of short duration and rarely permanent. When the glaucoma was probably a manifestation of an endocrine disturbance, results were most striking. (1 chart, bibliography.)—A. J. Ophthalm.

Book Reviews

A Manual of Tuberculosis—Clinical and Administrative—By E. ASHWORTH UNDERWOOD, M.A., B.Sc., M.D., D.P.H. (U. GLAS.), with an Introduction by Prof. J. R. CURRIE, M.A., M.D., F.R.C.P., D.P.H. Third Edition, largely rewritten. 524 pp., illustrated, (E. & S. Livingstone Ltd., Edinburgh), 15 sh. net.

Tuberculosis is an ever-increasing problem in this country. The student and the practitioner should be familiar with the causes of tuberculosis and its recognition in private and hospital practice and must be thorough with modern methods which are proving so successful in reducing infection, treating the active stages and in the end returning the patient to some effective role in the world. Dr. Ashworth Underwood, in his new Manual presents the student and practitioner with a discriminating discussion of those aspects of the pathology, clinical features and treatment which are most essential and deals with problems having a bearing on the Evolution of Pulmonary Tuberculosis, Allergy and Immunity as related to Tuberculosis; on X-rays and radiography as applied to tuberculosis work; on the mental aspects of this disease; on methods employed as a routine in the clinical laboratory; on social medicine and tuberculosis. He has written this book in a manner which will make it useful to the physician, surgeon, nursing staff, and the social worker. The book is well got up and profusely illustrated with photographs and line drawings. At the end of the book is a note giving the names of general works on diseases of the chest and on the problems of tuberculosis which may be consulted by those interested in the study of that disease. We congratulate the author on his work and recommend it to all who want to make a special study of the subject.

The Problem of Lupus Vulgaris — By ROBERT AITKEN, M.D., F.R.C.P.E., F.R.S.E., Lecturer in Diseases of the Skin, Edinburgh University, Physician to the Skin Department, Royal Infirmary, Edinburgh. 1946, 66 pp., illustrated. (E. & S. Livingstone Ltd., Edinburgh.) [15 sh. net.] Lupus Vulgaris is a serious social and economic disability. It is, however, not very common in this country. The present book embodies the experience of 30 years practice in the treatment of this disease. The first two chapters of the book deal with the clinical features and treatment. The third chapter deals with ultra-violet irradiation and gives a few case-reports. The whole of Chapter IV is devoted to Tuberculin treatment with a few case reports. The last chapter is devoted to a discussion of the problem of Lupus Vulgaris. The illustrations and the general get-up are very good and the book is an authoritative description of the disease.

Sterility—its causes and cure. By Dr. K. V. MATHIEW, Travancore Medical Service, 96 pages, illustrated—Published by Health Book Stall, 7, Krishnankoil Street, Madras Price Rs. 3-12-0.

This little handbook gives in a concise manner the causes and treatment of sterility. Beginning with a description of the anatomy of the male and female sex organs, Dr. Mathew proceeds to give causes of sterility in the male and then describes the methods of overcoming this trouble. He then discusses the causes of sterility in women and its cure. The subject of artificial insemination has been given in a very short paragraph. A chapter is devoted to hormones. On the whole, it is a very useful book.

Correspondence

Chronic Rhinitis

To The Editors, THE ANTISEPTIC, Madras.

Sirs,

I am suffering from Chr. Rhinitis since last 30 years. Both my middle and inferior turbins of both the sides have been removed during the course of treatment with absolutely no relief from the symptoms. Now about a couple of months back I got a severe frontal sinus infection and was

cured of it with S.U.P. 36, M & B 760: Polydin and cold vaccine and Penicillin. With the above treatment both the frontal sinusitis and chronic cold symptoms are completely relieved.

But eventually a new trouble has begun during the Penicillin treatment. After the first Penicillin injection of 15000 Units I.M. I suddenly lost the hearing of the rt. ear. The deafness was of the obstructive type.

I consulted a distinguished E.N.T. specialist of Ahmedabad and he was of the opinion that the deafness is simply due to the fungus infection of the external ear. After syringing the ear a plug of fungus was removed with a temporary cure of the deafness.

Now the fungus reappears every 3rd or 4th day since then inspite of the following treatment: (1) Glycerine Carbolic drops, (2) Boric-Alcohol Acriflavin drops, (3) Mercurochrome drops, (4) Cibazol dusting powder and (5) Gentian Violet 2% drops.

Now every 3rd or 4th day I have to syringe my ear and every time a plug of fungus gets out with a temporary cure of the symptoms. The idea in presenting this history before you is to get some useful suggestion regarding a permanent cure of this ailment.

I request the readers to make their suggestions in the columns of the next issue.

Kunghar Dispensary } Yours Faithfully,
Via. Patan (Gujarat) } C. C. PARIKH,
Kunghar, 3-7-1946. } L.C.P.S., (Bom.)

Preparations and Inventions

"Iodoquinol" manufactured by the Standard Pharmaceutical Works, Ltd., Calcutta. —On account of the protein character of Amœbiasis, the difficulties of eradicating the infection and eliminating relapse present a difficult problem. It is claimed that "Iodoquinol," unlike other products, can be used for 20 days in one course, thus exerting a beneficial influence on the residual infection. Further, with its higher iodine content (64%) the amœbicidal property of Iodoquinol is claimed to be much more than that of similar other products.

We are informed that in Iodoquinol one chlorine atom of vioform has been replaced by a second atom of iodine. This change in the chemical configuration has improved its therapeutic potency. The elimination of chlorine from the drug precludes the chances of gastrointestinal irritation met with after the administration of Iodochlorohydroxyquinoline compounds.

All the above factors go to make Iodoquinol an ideal drug for the treatment of Amœbiasis in acute or chronic form, balantidial infection and lambliasis.

Penicillin.—We are informed that Messrs. Glaxo Laboratories Limited, are now in a position to market British Penicillin in this country. We are also informed that although Penicillin was a British discovery, owing to the needs of war, British production has until now been earmarked exclusively for the fighting services. Now for the first time it is available in India.

We are further informed that Glaxo Laboratories, Limited run one of the two large scale penicillin plants in the United Kingdom, the other being a Government-owned plant at Speke in Cheshire.

News and Notes

F.R.C.S. (England)

Primary Examination in December

The Primary Examination for F.R.C.S. (England) will be held in Madras on December 23, 1946.

The Examination will be open to candidates (men or women) possessing a qualification which is registerable in the British

Medical Register, or degree in medicine and surgery of an Indian University recognized by the Council of the Royal College of Surgeons of England.

Regulations and application forms are obtainable from the Director of Examinations, Royal College of Surgeons of England, Examination Hall, Queen Square, London, W.C.1.

A Correction

In the article "Sulpha Drug in Samill-pox" by Dr. S. G. Sonavay, published in the July issue of the ANTISEPTIC in page 484, the 8th line from bottom should be Liq. Strychnine $\mathcal{M}$ ix. instead of Liq. Strychnine $\mathcal{M}$ ix.

School of Tropical Medicine, Calcutta

The six months' course of instruction for the Calcutta D.T.M. examination will begin from the 15th October 1946.

Applications for admission should be submitted by the 31st August to the Director, School of Tropical Medicine, Calcutta.

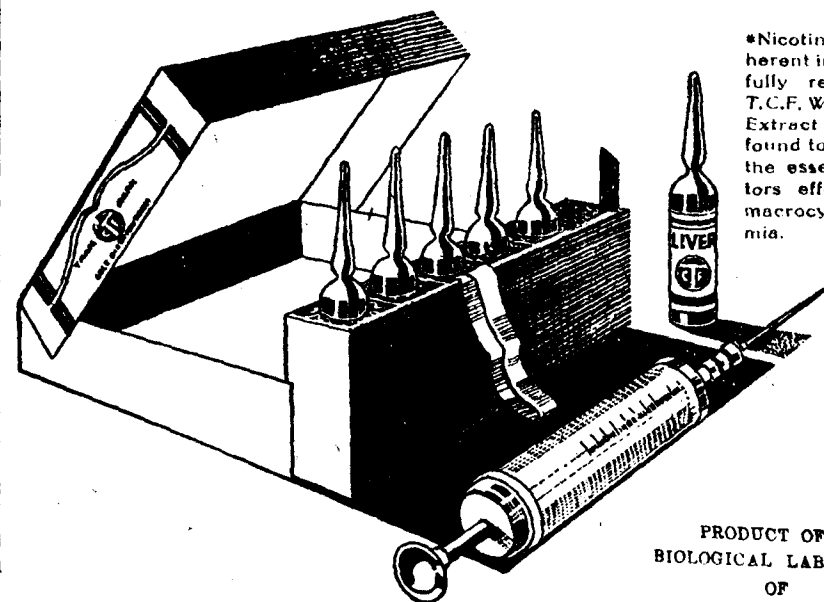
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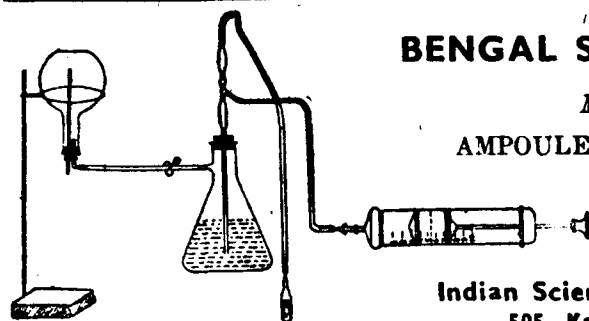
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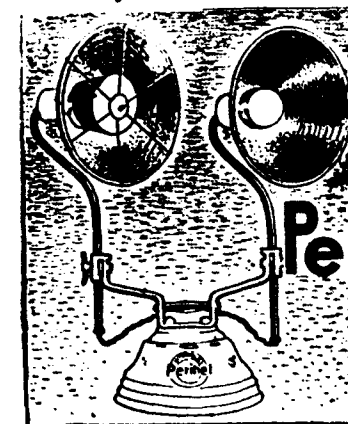
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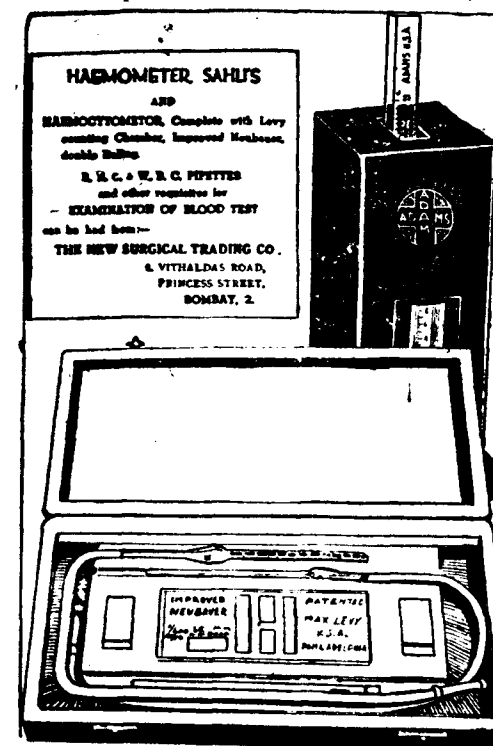
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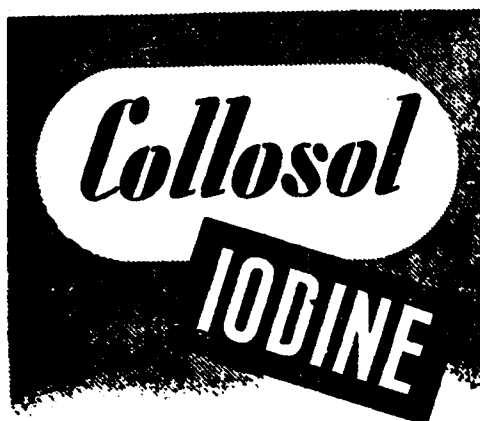
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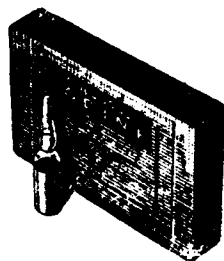
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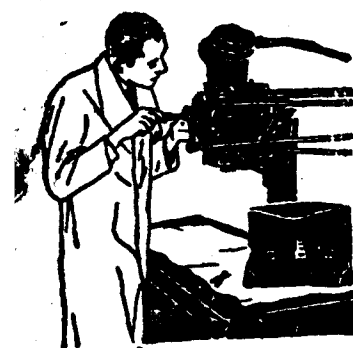
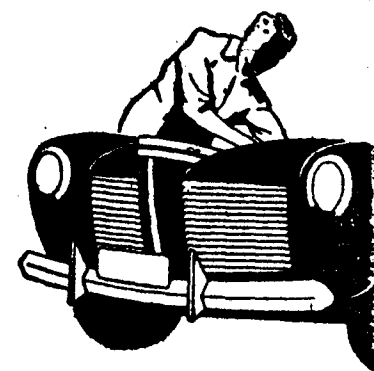
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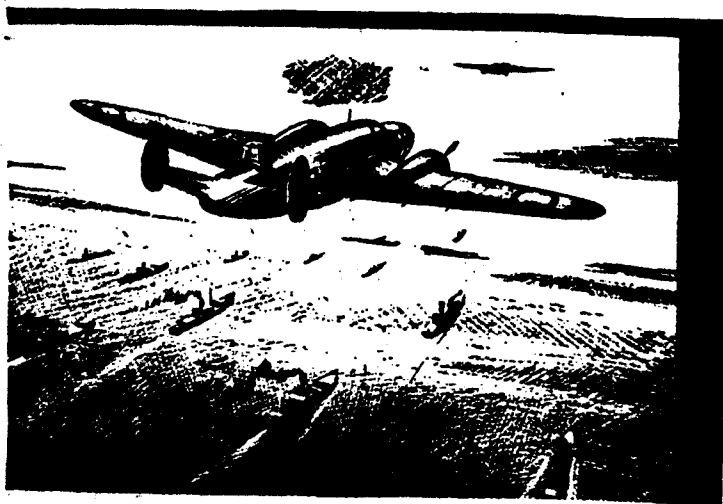
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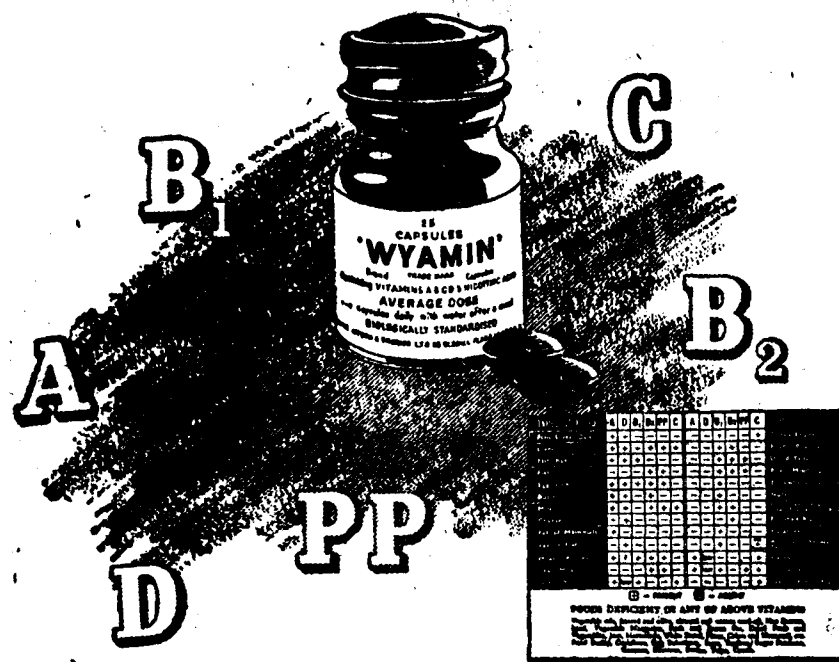
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