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January, 1971
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The Pakistan Journal of Health

A Quarterly Devoted to
Hygiene, Preventive Medicine,
Medical Sciences and
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6 —Birdwood Road, Lahore—(West Pakistan)

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INFORMATION

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INNOVATION AND HEALTH IN THE DEVELOPING COUNTRIES

by

Professor A. H. Awan, M. D., T. D. D., M. P. H. and*
*Professor (Mrs.) A. K. Awan, M. B. B. S., M. P. H., F. P. H. A. P.***

Health of a community is a part of its culture, and abundant empirical evidence exists to show the relationship of socio-cultural practices and behaviour patterns with the occurrence, control, management and treatment of diseases. Sociological theory of rank differentiation based on functional explanation relates social s'atus with life styles and life chances, and with the resulting sub-cultures. The life styles have an impact on the needed behaviour for health, and life chances also incorporate impact of disease and death. Relating disease and death with status oriented sub-cultures is therefore in consonance with sociological thought. It is in this framework of status oriented sub-cultures that we should study the impact of health inventries and innovations. The upper classes not only are the first to get affected by innovations, but draw maximum benefit from its wide spread utilization.

No human society lives in a state of nature. Each Society has a more or less elaborate and integrated social system, inherited from ancesstors and transmitted to the descendants. This heritage maintains the spatial and temporal solidarity and perspective of the group. The social heritage basically determines our culture. Culture is the cause and effect of the qualities that make life human. "Culture includes all that man has made in the form of tools, weapons, shelter and other material goods and processes, all that he has elaborated in the way of attitudes and beliefs, ideas and judgments, codes and institutions, arts and science, philosophy and social organization"[1].

Culture also includes health and medicinel, i.e., health oriented practices and the scientific means and mechanism of preventing and treating ill health.

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"*Pak. Jour. Hlth.* 1970-71"

Everything, material or non-material, created by man in the process of living, comes with in the concept of culture. Culture undergoes gradual changes due to the impact of environment and needs.

Culture originates from associative life and is elaborated in response to human need. The beginning of new culture facts, at any given time or place, is either the result of invention or innovation. These influence social organization through social change, and have economic effects[2] All culture facts begin as inventions. Once invented, culture facts spread. It is easier to copy and use things than to invent them. Within a group, innovational cultural practices come from outside sources. Wherever there is communication of the group with outside groups, and within the group, the knowledge of outgroup inventions would be acquired and would be introduced as innovation within the group. But there is always selection.

Social evaluation determines the social uses to which innovations may be put[3]. Only those things are accepted from outside culture for which there is obvious felt need and which can be adjusted to the existing culture and the social patterns of the group. Though simple people readily accept metal implements of the civilized group because af their obvious superiority over stone and bone tools, in the realm of social organization and idea systems innovations are resisted and change is slow. A long time may elapse after the introduction of an innovation or after an invention before it is accepted and widely used. Innovational influences leading to social change spread through dispersion, succession and convergence Multiple factors may collectively operate to produce a social change. Diverging social effects may result because of the introduction of inventions or innovations.

There are two philosophies of medicine : the primitive or superstitious and the modern or rational. They are in complete disonance to one another. The overwhelming majority of human race, mostly occupying the developing countries, have held belief in Demonic Theory of Disease Causation, i.e., disease is caused by supernatural powers may be exercised by living persons (witches), by spirits of disembodied dead or by super-human entities. Such a doctorine associates disease with sin, and attempts to control disease by ceremonial and superstitious measures. On the other hand rational medicine, as nurtured and matured by developed societies, is based on the concept that disease arises from

natural causes and associates ill-health with ignorance. Civilized men try to control the forces causing disease by material and not spirtualistic means, as they recognize that knowledge and its application are the sole means of preventing ill-health. Introduction of innovations in primitive superstitious societies produces social change and modernization through a chain reaction.

Man's exalted position in the world is found upon his mental rather than physical attributes ; physically he is ill equipped for survival. He realized this fact long ago and has been acquiring and using knowledge for gaining more and more supermacy over his environment.

Modern medical science has made this world of ours a far more convenient place to live in than it was a generation or two before. Many of the perils which beset primitive men have been made to disappear from earth, and many others are confined within narrow bounds. Maximum benefit of medical science has been drawn by developed nations where death has been greatly controlled and life has been greatly prolonged. However, developing nations have been left behind and due to inadequate diffusion, benefits of medical innovations are not fully available to the majority of masses. The innovations which have been accepted and integrated in our culture have resulted already in the rarity of past epidemics and in decline of morbidity and mortality rates. Life expectancies have considerably increased as compared to what these were previously. Yet as compared to developed nations, where adequate social machinery was developed to utilize new knowledge and where the impact of the inventions and innovations has been the maximum, we still have a large number of health problems leading to collossal death and disease rates and loss of human potential and happiness. Innovations became available to us, but due to poor dispersion and diffusion and due to the lack of development of adequate social machinery and political system, we have not yet been able to take maximum benefits from them. World has become smaller and communications have improved. Knowledge of the better lot of people in developed societies leads to feelings of discontentment, frustration and rebellion against the social and political order amongst the people of developing nations. This in turn makes the social and political systems unstable, thus retarding progress.

In a nation-wide study, recently conducted by the Public Health Association of Pakistan, at the request of the Planning Commission of Pakistan, it was found that :

"*Pak. Jour. Hlth.* 1970-71"

1. Large majority of Union Council leaderships were not familiar with the health responsibilities as envisaged under the law.

2. Record keeping of vital data, a basic public health tool for health development, was being imperfectly carried out. Only 12.5 per cent of Unions were recording live births to an extent which could be considered as correct. Further not more than 11.8 per cent Unions were correctly recording their crude death rate.

3. Infant mortality rate in rural Pakistan was found to be 80.9 per 1000 live births as against 20-30 per thousand in developed nations.

4. Maternal mortality rate in rural Pakistan was found to be 16.92 per thousand live births as compared to 0.1– 0.5 in the developed countries.

5. Majority of communicable diseases remained uncontrolled in the Union areas, inspite of the fact that they were recognized as health problems needing urgent attention. Even Smallpox, which can be easily controlled through preventive vaccinations, was present in 67.1 per cent of the Unions.

6. Seventy eight per cent of the Unions had not even an elementary health organization for performing their health functions, though 98.3 per cent were conscious of the need for its existence.

7. Most of the health functions were, by large not being performed. Those that were being attempted were being imperfectly attended.

8. For water supply for drinking purposes, 10.5 per cent Unions depended upon raw river water, 11.8 per cent on canal or stream water, 1.3 per cent on rain water, 36.8 per cent on ponds, 2.6 per cent on springs, 53.9 per cent on wells, 44'7 pes cent on hand pumps, 1.3 per cent on karez, 52.6 per cent on tubewells, but only 6.6 per cent of unions had arrangements for community water supply. Thus 93.4 per cent unions permitted individuals to arrange for their own water supply at an individuals private level.

9. Fifty per cent Unions had no latrines at the homes of the people. Varying percentage of Union's population had latrines at home in the remaining 50 per cent of the Unions. Amongst 65.8 per cent Unions people went to the fields or jungle for answering the call of nature, and in 1.3 per cent they went to public latrines.

(10) Seventy per cent Unions had not even a small dispensary to take care of their sick. Unions by large did not have any MCH services which are universally considered as most basic, most vital. and most economical.

(11) Economic potentiality of the Union Councils for carrying out their health functions was ridiculously low, and with the amount of public money available they cannot carry out even a few of the elementary health functions[4].

We can thus see that inspite of the available knowledge of medical science, imported from developed nations, rural Pakistan has neither been able to develop an adequate mechanism for health functioning nor has been able to prevent, control or deal with the health problems which confront the people.

Cultural changes occur with the introduction of new elements viz., inventions, but social changes are proportional to the extent of their adoption. Extensive adoption does not only depend upon the introduction of the new element but also on the diffusion of the innovational influences. These influences not only have impact on the attitudes and behaviour patterns of the individuals but also affect the nature of politics and activities of the government, Spiralling of social change may produce a cummulative accelerated effect leading to faster development. Delay in wide-spread diffusion of innovations, thus impedes social change and social progress. A social change takes place through the medium of ideas. Introduction of innovations to stimulate factual and observational ideas produces maximum impact and leads to faster social change.

The conquest of death has made its victories. In its means of advancement it has run ahead of civilization. It now awaits for the culture lag to disappear before fullest benefit of this victory over death can be employed by mankind[5]. The prevalence of diseases like tuberculosis, typhoid, dysentery, whooping cough, tetanus, puerperal sepsis, etc., and the resultant deaths are no

fault of medical science. Medical science has led the conquest, but it can give no more than what the communities can accept. It cannot of itself overcome true inertia of civilization, or say what value shall be placed on the lives of the people by a nation.

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1. Alfred McClung Lee (1965). *Principles of Sociology*, New York: Barnes & Noble, Inc., p. 140.

2. William F. Ogburn and Meyer F. Nimkoff (1958). *Sociology*, 3rd ed Boston : Houghton mifflin Company, p. 670.

3. *Ibid.*, p. 678.

4. Akhtar Hussain Awan (1969). *The system of Local Health Services in Rural Pakistan and Planned Administrative and Technical Support*, Lahore : Public Health Association of Pakistan.

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THE PATTERN AND MORBIDITY OF ABNORMAL UTERINE BLEEDING

Professor M. A. Farooki, D. Sc., M.B.B.S.. M.P.H.

The frequency of abnormal uterine bleeding and the morbidity of it has attracted the attention of the author for the last 10 years, for several reasons, including the etiology, the pattern, the age features and the prognostic outlook.

It can at once be granted that abnormal uterine bleeding which can be excessive or irregular or both (abnormal in amount, duration, frequency or nature of flow) can be a symptom and manifestation of many diseases and states, functional. dysfunctional, organic or elusive.

The understanding of the nature of uterine bleeding and the management of it is the most important and time consuming problem in the practice of gynecology. The present state of our knowledge is neither comprehensive nor definitive, as the factors spread beyond the realms of uterus and even the ovaries (Jeffcoate 1969).

Methodology and Materials

This presentation is an analytical study of the gross and histological features of the specimens received alongwith requisition slips of the patients containing brief pertinent clinical data on uterine bleeding. Patients with (non-uterine) vaginal bleeding only are excluded. Materials for histopathology were composed of endometrial curettings, cervical biopsies, tuboovarian materials or hysterectomy specimens received in the following institutions.

1.	Pathology Department, Khyber Medical College, Peshawar. (Pakistan) 1-5-1966 to 27-5-1968	1054 specimens
2.	Pathology Department, Nishtar Medical College, Multan (West Pakistan) 28-6-1955 to 7-1 -1969	2693 ,,
3.	Pathology Department, Khyber Medical College, Peshawar 1-11-1960 to 30-4-1966	537 ,,

Head of Department of Pathology, King Edward Medical College, Lahore.

4. Pathology Department, Philadelphia General Hospital, Philadelphia, U. S. A. 1957-1958 2614 ,,

The assessment of the amount of flow is arbitrary and judged by the patients description of the amount. Women vary in their criteria for abnormal bleeding. A menstrual cycle shorter then 23 days and longer than 35 days is always abnormal (Goldzieheret at 1947). Artefacts are to be excluded in the study of uterine bleeding.

Definitions of Patterns of Bleeding

As one goes through the clinical notes, the description of the pattern of abnormal uterine bleeding is often incomplete, unrevealing and inconclusive. In general, the terms menorrhagia, metrorrhagia, menometrorrhagia, F. U. B., D. U. B., bleeding, continuous bleeding, irregular bleeding, excessive bleeding, intermenstrual bleeding, spotting, irregular spotting, amenorrhoea followed by bleeding, post-abortional bleeding, postnatal bleeding, post-coil bleeding, post-coital bleeding, polymenorrhoea, hypermenorrhoea, meno-polymenorrhoea, metro-polymenorrhoea and post-menopausal bleeding are met with.

Menorrhagia is the cyclical (Ovulatory) bleeding excessive in amount or duration or both, occuring at normal or reasonably normal regular intervals (Jeffcoate 1969). There is no innate ovarian disturbance. It is one form of functional uterine bleeding likely to be seen in uterine vascular conditions, congestions, systemic states, mechanical conditions and with increased thickness and retarded speed of repair of endometrium.

Hypermenorrhoea is the same thing as menorrhagia.

Metrorrhagia is totally irregular non-cyclical uterine bleeding of any amount occurring irregularly or continuously at indeterminate times. usually more frequently than every 20 days. There may be episode of daily spotting intermingled with episodes of moderate ot profuse bleeding. The cause can be an alteration in ovarian rhythm, a surface ulcerative lesion, growths of genital tract, aberrations of pregnancy, P. I. D. or anovulation.

Polymenorrhoea is the rare cyclic bleeding occurring at too frequent intervals. The follicular phase is shortened and regular ovulatory cycles occur more frequently than every 23 days. Hypermonorrhoea is also a frequent

accompaniment of polymenorrhoea (polymenorrhogia or epimenorrhagia) (Rogers 1963 ; Jeffcoate 1969). Most cases occur in early menstrual life and in premenopause (Collect et al 1954 ; Rogers 1963).

Epimenorrhoea is the same thing as polymenorrhoea. The causes can be functional or organic ovarian disease. congestion of ovaries or disturbance of pituitary ovarian relationship.

Polymenorrahgia (menorrhagia + polymenorrhoea) is excessive and too frequent cyclical bleeding (also called epimenorrhagia), The cause is a disturbance in ovary as well as uterus, especially in pelvic congestion of inflammatory and anxiety states (Jaffcoate 1969).

F. U. B. and D. U. B.

Functional uterine bleeding should mean the situations in which there is alteration or irregularity in the frequency of ovulatory cycles or the amount of flow in ovulatory cycles (Rock 1945 ; Fluhman 1956). Menstrual bleeding has always been preceded by ovulation. Examples are hypermenorrhoea (excessive or prolonged bleeding), polymenorrhoea (periods occurring more frequently than normal) and menorrhagia (prolonged or profuse periods ; same as hypermenerrhoea). Systemic disorders and organic diseases of reproductive system are to be excluded.

Dysfunctional uterine bleeding should mean disorders of flow associated with *failure of ovulation* (*metropathia hemorrhagica*) (Rogers 1963), the anovulatery cycles and abnormal bleeding being based on a disturbance in the rhythmical production of ovarian hormones (Jaffcoate 1969).

The terms functional and dysfunctional uterine bleeding are used to describe in clinical gynecology, abnormal uterine bleeding *not due to organic lesions*, but a lot of confusion and disagreement exists as to the use of terms.

One facet is academical and one technical. Several people use the term "Functional Uterine Bleeding" (F. U. B.) for both F. U. B. and D. U. B. while several other use the term "Dysfunctional Uterine Bleeding" for both F. U. B. and D. U. B. conditions (Jeffcoate 1966) without organic cause or palpalale lesions. In Practice the names functional or dysfunctional become unsatisfactory as they are bound to include a number of conditions that are not functional/

dysfunctional but are based on lesions of ovaries and uterus (Montgomery 1965) The process of exclusion of all organic lesions is laborious and time consuming and even a clear definition of an organic lesion is lacking. More than one cause of abnormal bleeding may be present. Sutherland (1953) analyzed 1000 cases of functional bleeding and found 139 instances of organic pathological lesions of endometruim.

Endometrial curetting only helps in diagnosing a local lesion such as incomplete abortion, uterine Polyp, tuberculous endometritis and carcinoma etc and reveals the tesponse of endomettrium to ovarian hormones, and sometimes curettage may even fail to reveal polyp, early carcinoma etc.

Age group (years)	Continuous bleeding	Total
16—20	11	
21—25	25	
26—30	60	
31—35	52	
36—40	36	
41—45	22	
46—50	10	
51—55	3	
56—60	0	
	219	

Table I

The pattern of bleeding symptom by age groups in the surgical material from Khyber Medical College Peshawar (1-5-66 to 27-6-68), Nishtar Medical College Multan (27-6-55 to 7-1I-69) & Philadelphia General Hospital (57-58).

Age group (years)	Menorr-hagia	Metrorr-hagia	Poly-menorrhoea	Polymeno-rrhagia	Meno-metro-rhagia	Just bleeding-
16—20	26	30	4	5	0	37
21—25	87	110	8	4	18	93
26—30	191	198	16	8	27	192
31—35	173	144	8	13	35	138
36—40	134	134	9	6	39	118
41—45	87	70	6	8	30	67
46—50	45	38	2	1	16	40
51—55	10	6	0	0	2	7
56—60+	1	0	0	0	0	1
Total :—	754	730	53	45	167	693

Continu-ous bleed-ing	Amenorr-hoea followed by bleed-ing	Postnatal or post abort-ional bleeding	D. U. B. unquali-fied	Post meno-pausal	Post I. U. D. bleeding	Post coital bleeding	Total
11	38	11	21	0	0	1	184
25	97	42	57	0	3	1	545
60	160	83	120	0	8	3	1066
52	77	37	85	0	4	3	769
36	54	20	69	1	I	1	622
22	19	8	39	8	1	0	365
10	7	1	21	18	0	0	199
3	2	0	1	21	0	0	52
0	0	0	0	43	0	0	45
219	454	202	413	91	17	9	3847

Table I records the pattern of abnormal uterine bleeding in 3847 patients in Pakistan (Peshawar and Multan) and some cases from Philadelphia General Hospital, U S A, from whom uterine curettings, biopsies and specimens were examined. The presenting or major bleeding symptoms fell into following categories and proportions :

Menorrhagia	754	19.6%
Metrorrhagia	730	18.9%
Polymenorrhoea	53	1.4%
Meno-metrorrhagia	167	4.3%
Epimenorrhagia	45	1.2%
Bleeding	693	18.0%
Continuous bleeding	219	5.7%
D. U. B. (unqualified)	413	10.7%
Amenorrhoea/bleeding	454	11.7%
Postpartal or post-abortional bleeding	202	5.3%
Post-intrauterine device insertion bleeding	17	0.4%
Post-coital bleeding	9	0.2%
Postmenopausal bleeding of any pattern	91	2.4%
Total	3847	100.0%

The most frequent pattern was menorrhagia (19.6%) followed by metrorrhagia (18.9%) etc.

The patients belonged to all age groups the maximum number being in age group 26-30 years :

Aged 16—30 years	184	4.8%	
,, 21—25	545	14.2%	
,, 26—30	1066	27.7%	
,, 31—35	769	20.0%	

,,	36—40	622	16.2%
,,	41—45	365	9.5%
,,	46—50	199	5.2%
,,	51—55	52	1.4%
,,	56—50	45	1.2%
	Total	3847	100%

CAUSES AND PATHAGENESIS OF ABNORMAL UTERINE BLEEDING

A. Cardioovascular abnormalities, blood dyscrasias & systemic causes

In this category are disorders of clotting mechanism (hemophilia ; pseudohemophilia; christmas disease), thrombocytopenic purpura, excessive capillary fragility states, leukemia, aplastic anemia, iron deficiency anemia (Taymor et al 1960), hypertension with uterine vascular arteriolosclerosis (especially significant in some cases of post-menopausal bleeding ; mechanism is excessive breaching tendency of superficial endometrial vessels) and congestive cardiac failure. The pattern of bleeding is usually menorrhagia. Anemia can be both a cause and effect of menorrhagia. In arteriolosclerosis of spiral arterioles, there is insufficient contractiltility leading to menorrhagia and irregular shedding from difficulty of detachment leading to prolonged menstrual bleeding,

Most of the above causes usually go undiscovered and unrecorded in studies of the present nature. Most of the cases in the present series in which no organic cause was discovered in the present series and in which the endometrial curettings, cervical biopsies, adnexal specimens and hysterectomies yielded negative results (only estrin phase, progestin phase or inactive endometria) could belong to this category of causes.

Systemic basis of uterine bleeding (blood dyscrasia) may exist in the background even in cases with obvious mechanical or other pelvic organic lesions apparently causing abnormal uterine bleeding. For unknown reasons, most women with blood disorders do not experience significant abnormal uterine bleeding and abnormalities of menstruation.

Table II

Histopathological diagnosis in 3847 cases with abnormal uterine bleeding (Peshawar and Multan, Pakistan).

Pattern of abnormal uterine bleeding	Estrin phase	Pro-gestin phase	Irregular matura-tion	Cystic endomet rial hyper-plasia	Endomet-rial hyper-plasia + chronic endometr-itis	Endomet-rial hyper-plasia + chronic cervicitis
Menorrhagia	230 (1 with salping-itis 5 with ovarian cyst)	124	6	114	4	5 (1 with follicular cyst)
Metrorrhagia	229 (4 with ovarian cyst)	122	7	115 (1 adeno-matous glandular	3	2
Polymenorrhoea	25	15	1	2	—	—
Menometrorrhagia	43 (1 with cancer ovary)	27	—	9	—	—
Epimenorrhagia	17	9	1 (1 with endometr-itis)	3	—	—
Just bleeding	191 (3 with tumour ovary)	78	1	144	5	4 (1 with cervical polyp)
Continuous bleeding	47	25	4 (1 with endomet-itis)	45	4	—
Just "D. U. B."	132	77	2	163	1	2
Amenorrhoea followed by bleeding	71	48	3	27	—	—
Post-partal or Post abortional bleeding	30	22	—	8	2	—
Post I. U. D. bleeding	5	1	—	—	—	—
Post coital bleeding	3	1	—	—	—	—
Postmenopausal bleeding	3	0	—	13 (1 adeno-matous glandular)	—	1
Total :	1026	549	25	643	19	14

Pattern of abnormal uterine bleeding	Cystic endomc-trial hyperplasia + fibroid	Cystic endome-trial hyperplasia + adeno-myosis	Endometrial polyp	Endometrial hyperplasia + fibroid + Adenomyosis	Fibroids., Fibroid, polyp.	Fibroid, + Adeno myosis	Adeno-myosis
Menorrhagia	3 (1 with endometritis)	2	8	—	57 (1 with RPP) (1 with endom polyp) (2with salpingitis (1 with Brenner (1 with foll. cyst)	2 (1 with cervicitis	14 (1 with polyp) (2 with cervicitis 1 with cervicitis endometritis& salpingitis
Metrorrhagia	1	3	1	—	22 (1 with salpingitis)	2	17 (4 with cervicitis) (1 with endometritis
Polymenorrhoea	—	—	—	—	1	—	—
Meno-metrorrhagia	1 (with endometrial polyp)	—	—	—	18 (1 with foll. cyst) (1 with R.P.P. (1 with salpingitis & simplecyst ovary	—	2
Epimenorrhagia	—	1	—	—	—	—	—
Just bleeding	3 (1 with cervicitis	3	6	—	28	—	8 (2 with cervicitus & cervical polyp)
Continuous bleeding	1	—	1	—	4	—	1 (polyp)
Just "D.U.B."	2	—	—	—	1(with simple cyst ovary)	—	2 (with) cervicitis)
Amenorrhoea followed by bleeding	—	—	—	1	3	—	—
Post-abortion or post-partal bleeding	—	—	—	—	—	—	—
Post-IUD bleeding	—	—	—	—	—	—	—
Post-coital bleeding	—	—	—	—	1	—	—
Post-menopausal bleeding	—	1	1	—	3 (1 with salpingitis)	2	1 (with cervcitis)
Total :	11	10	17	1	138	6	45

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Pattern of bleeding	Incomplete abortion RPP	Mole	Ectopic pregnancy	Cervical polyp	Endometritis (mostly chronic)	Cervicitis with or without polyp	endometritis + cervicitis
Menorrhagia	20	—	—	7	76 (1 with endometrial polyp)	22	7
Metrorrhagia	42	1	—	12	66 (1 with salpingitis	26	3
Polymenorrhoea	—	—	—	—	8	—	—
Meno-metrorrhagia	11	—	—	2	11	2	1
Polymenotrhagia	2	—	—	—	9	—	—
Just bleeding	72(1 with cervical polyp)	3((mole) 2 (chorio adenoma destruens	3	1	54	33(1-with follicular cyst)	—
Continuous bleeding	30	—	1	2	36	5	2
Just "D.U.B."	8	1	—	1	10	—	1 (with cervical polyp
Amenorrhoea followed by bleeding	189	7	2	—	89	3	—
Postabortional or postpartal bleeding	104	3	—	—	28	1	—
Post IUD insertion bleeding	—	—	—	—	11	—	—
Postcoital bleeding	—	—	—	—	1	1	1
Postmenopausal bleeding	—	—	—	4	15	6	1
Total :—	478	17	6	29	414	99	16

Pattern of bleeding	Tuber culous endomet ritis	Inadequ- ate curet ting of NAD	Ademo- myosis + cervicitis endomet ritis	Ca-in-situ	Fibroid + endo- metritis	Fibroid + cervicitis
Menorrhagia	9	23	2	—	3 (1 with salpingitis 1 (with cervicitis	8
Metrorrhagia	5	27	—	1 (with simple cyst); 1 (with dermoid cyst)	1 (with salpingitis)	—
Polymenorrhoea	—	1	—	—	—	—
Meno-metrorrhagia	—	5	—	—	1	1 with salping itis)
Epimenorrhagia	—	3	—	—	—	—
Just bleeding	4	32	—	1 (with endometrial polyp & salping itis)	—	4
Continuous bleeding	—	3	—	—	—	—
Just "DUB"	1	9	—	—	—	—
Amenorrhoea followed by bleeding	—	6	—	—	1	—
Post abortional or post partal bleeding	—	4	—	—	—	—
Post-IUD insertion bleeding	—	—	—	—	—	—
Post coital bleeding	—	—	—	—	—	—
Postmenopausal Bleeding	—	8	—	—	1 (with cervicitis & salpingitis)	1
Total : —	19	121	2	3	7	16

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Pattern of bleeding	Adenocarcinoma corpus + adenomyosis	Epidermoid carcinoma cervix with adenomyosis	Adenoacanthoma Corpus	Epidermoid carcinoma cervix	Adenocarcinoma cervix	Adenocarcinoma corpus	Chorion carcinoma uterus	Leiomyosarcoma uterus	Total
Menorrhagia	—	—	—	3	—	2	—	3	754
Metrorrhagia	—	1	—	10	—	7	3	—	730
Polymenorrhoea	—	—	—	—	—	—	—	—	53
Metro-menorrhagia	—	—	—	—	—	1	—	—	167
Polymeno	—	—	—	—	—	—	—	—	45
Just bleeding	1	—	—	30	1	9	2	—	693
Continuous bleeding	—	—	—	4	1	2	1	—	219
D. U. B.	—	—	—	—	—	—	—	—	413
Amen/bleeding	—	—	—	1	—	3	—	—	454
Post-abortional postpartal	—	—	—	—	—	—	—	—	202
Post-IUD	—	—	—	—	—	—	—	—	17
Post coital	—	—	—	—	—	—	—	—	9
Post menopausal	—	—	1	19	1	10	—	—	91
Total :—	1	1	1	67	3	34	6	3	3847

(Elghamar et al 1949) suggested that anexcess of haparin-like substances in the blood of some women may cause monorrhagia.

B. Aberrations of pregnancy Abortions, Moles and ectopics

A woman with previous regular menses who experiences amenorrhoea followed by a sudden episode of uterine bleeding is said to be a likely subject of abortion or ectopic pregnancy. In the present series (table II) there were 454 cases with this history but only 196 (43%) proved to be cases of incomplete abortion. *Uterine bleeding in tubal Pregnancy* occurs in most (but not all) cases (Novak and Woodruff 1962). The source of external bleeding in tubal pregnancy is the endometrium (Sampson 1913) and perhaps not to any significant degree on a significant number of cases the tubal placental site. Endometrial curetting showing definite decidual tissue without chorionic villi suggests such a situation, but the occasional pronounced premenstrual hypertrophy of nonpregnant edometrium simulating decidua has to be excluded. Since uterine curettage in most cases of tubal pregnancy with uterine bleeding is performed late (embryo has died ; decidua has been cast off) histology of curettings commonly shows normal endometrium (and no decidual change) in postmenstrual or interval phase (52% of cases) Decidual change is identified only in 33% (Novak and Woodruff 1962) In the present series of 3847 cases there were 6 cascs of tubal pregnancy.

Hydatidiform mole (15 cases), chorionepithelioma (6 cases), retained secundines (478) and chorioadenoma destruens (2 cases) were associated with uterine bleeding.

After abortion and labour, the uterus is *sometimen* slow to involute (subinvolution) and therefore, the *first few* menstrual periods tend to be heavy. Very rarely, the *delayed* involution can cause severe menorrhagia if the large vassels in the placental site fail to become obliterated (Jeffcoate 1969).

In the present series of 3847 cases of abnormal uterine bleeding (Table II) there were 202 with post-abortional and post-partal bleeding of various patterns 10 of these (5%) showed cystic endometrial hyperplasia 107 (53%) showed retained products of pregnancy, 30 (15%) showed only endometritis which could be consistent with previous abortion and 62 (31%) showed unremarkable endometrium.

Amongst the 454 cases with history of amenorrhoea followed by bleeding, 43% were of incomplete abortion as mentioned above, 28% had unremarkable endometrium and 6% had cystic endometrial hyperplasia.

C. Uterine infections, traumas and Congestions

Intrauterine contraceptive appliances and foreign bodies for inducing abortion etc and uterine trauma for various reasons, can cause either menorrhagia or metrorrhagia. In the present series, stone was found in the uterus in one case of endometritis and hysterectomy was performed in 2 post-partal cases for severe uterine trauma and hemorrhage. Bicornuate uterus can cause menorrhagia due to increased bleeding surface In the present series there was one such case causing metrorrhagia. Retroversion and prolapse can cause abnormal uterine hemorrhage (menorrhagia) due to chronic congestion of uterus and ovaries from interference of venous return. In the present series, there were some cases with retroversion and two cases with prolapse.

Psychological upsets as in emotional and nervous disorders found in nervous tension, anxiety states, sexual tension and overwork can be important cause of excessive uterine bleeding usually menorrhagia. Their presence is often overlooked. Modus operandi is both hormonal (hypothalamico-pituitary-endocrine axis) and neurogenic (nervous system which controls the pelvic vessels) by emotional stimulation of hypothalamus. The vascular congestion, active or passive can causes endometrial and myometrial hypertroply and cystic ovaries.

Menorrhagia can be caused by endometritis. *Nonspecific chronic endometritis* is seen commonly. (Hitschmann & Adler 1907).

Jeffcoate (1969) has stated that chronic endometritis used to be blamed for excessive uterine bleeding but this condition is rarely found in the young adult. I believe (Farooki 1966, 1967, 1968) that nonspecific chronic endometritis is a common (20%) condition in total curettings. Sutherland (1953) recorded chronic endometritis in 11% of cases with uterine hemorrhage.

It is a common but mistaken belief that most of the endometrium is shed every month thus rendering a persistent primary endometritis impossible. Therefore, the inferrence that persistence of endometritis must be due to associated facter that maintains irritation or infection of endometrium is erroneous (Hughesdon and Symmers 1966).

The commonest of the factors that may be associated (in 90% of cases) with persistent endometritis, are P. I. D., retained products of conception chronic salpingitis, recent abortion, recent delivery (Sharman 1955) with post-partum streptocoual infection, intrauterine foreign body. sloughing submucosal tumour and submucous leiomyomas that may become infected from ulcerated surface (Montgomery 1965). Acute endometritis is often due to retained products of conception following delivary and miscarriage. Robbins (1967) has stressed that the entity of a primary chronic endometritis has to accepted (even though we do not understand the cause) when plasma cells are present.

Senile form of endometritis and senile pyometra can occur in later life (De Voe and Randall 1949) and cause postmenopausal bleeding. Most of these cases are associated with narrowing of cervical canal.

In the present series of 3847 cases of abnormal uterine bleeding, there were 411 (11%) cases of endometritis, mostly chronic. 49 (1%) were in association with other lesions (fibroid, cervicitis, adenomyosis etc). The overall incidence of chronic endometritis alone or associated was near 12%. In the 414 cases with independent endometritis history of recent or past pregnancy was present in 117 cases. Others should menorrhagia (18%), metrorrhagia (16%), menometrorrhagia (2.7%), epimenorrhagia (2.3%), polymenorrhoea (2%), bleeding, continuous bleeding or D. U. B. 24.2%), post-I U. coil insertion bleeding (2.7%) post-coital bleeding (0.2%) and post-menopausal bleeding (3.6%)

I believe that chronic endometritis is an entity frequently met with in uterine curettings. It is not a myth apart from a previous abortion or labour. It may well cause several symptoms, including menstrual disturbances and abnormal uterine bleeding. Table III summarizes and restates my previously published findings on chronic endometritis in materials of Peshawar, Pakistan and compares it with my unpublished findings at the Philadelphia General Hospital, U S A, only to reemphasize that there are other workers and institutions which diagnose chronic endometritis frequently. While in Peshawar material, the frequency of chronic endometritis was 19%, in P.G.H. U.S.A. it was 16%. The figures are very similar.

Table III

Total endometrial curettings P. G. H. (1957-1958) : Histologic diagnosis compared with my previous report from Peshawar.

	Farooki (unpublished) Philadelphia General Hospital (1957, 1958)	Farooki (1968) Khyber Medical College, Peshawar
Estrin phase endometrium	76	393 (14.6%)
Progestin phase ,,	106	713 (26.5%)
Irregular maturation	1	8
Cystic endometrial hyperplasia	2	141 (5.2%)
Cystic endometrial hyperplasia + chronic endometritis	1	60 (2.2%)
Incomplete abortion ; retained secundines	1284	565 (21%)
Endometrial polyp	18	—
Endocervical polyp	19	—
Fibroid polyp	7	2 (0.1%)
Mole	3	17 (0.6%)
Inactive/atrophic endometrium	37	19
Endometritis, acute subacute	19	81 (3%)
Chronic endometritis	312 (16%)	508 (18.9%)
Tuberculous endometritis	3	30 (1.1%)
Adenocarcinoma corpus uteri	20	31 (1.2%) includes cancer CX & corpus
Carcinoma-in-situ CX	2	—
Adenomyosis	1	—
Inadequate curettings or NAD	28	122 (4.5%)
Total :—	1957	2690

The next point which arises is that if chronic endometritis is an established and frequent entity, what is its role, if any, in the causation of abnormal uterine bleeding. Table IV records this aspect.

Table IV

Bleeding symptoms observed in association with Endometritis (mostly chronic)

	Khyber Medical College Peshawar 1-11-60 —30-4-66	Philadelphia General Hospital USA 1957 —1958	N. M. C. Multan 27-6-55 to 7-11-69 K.M.C. Pesh 1-5-66 to 27-5-68	Total
Menorrhagia	94	23	76	193
Metrorrhagia	106	54	66	226
Polymenorrhoea	10	9	8	27
Menometrorrhagia	23	39	11	73
Polymenorrhagia	12	3	9	24
Just bleeding continuous bleeding	8	73	90	171
Dysfunctional uterine bleeding	—	21	10	31
Amenorrhoea/bleeding	7	11	89	107
Post-partal or post-natal	18	13	28	59
Post coital bleeding	2	5	1	8
Metrorrhagia + post coital bleeding	—	2	—	2
Menorrhagia + postcoital bleeding	—	1	—	1
Postmenopausal bleeding (any pattern)	8	8	15	31
Post I.U.D. bleeding	2	—	11	13
Total :—	290	262	414	866

In about 80% cases of endometritis, the complaint of abnormal uterine

bleeding is observed. For example at the P. G. Hospital, USA. out of 331 cases of endometritis diagnosed during 1957-58, 262 showed bleeding. Only 69 cases (20%) were the exceptions.

Table V gives the breakdown of bleeding symptoms according to age groups in 262 cases of endometritis :

Table V

Bleeding complaints of 262 cases of endometritis by age groups

(P.G.H. ; U. S. A.)

Age years	Meno	Metro	Poly	Meno + metro	Poly + meno	Bleeding	Ame ı bleeding	Post-natal Post-abortal	"D.U.B."	Post-coital	Post-menopusal	Total
16—20	1	4	—	1	1	5	—	4	2	—	—	18
21—25	4	7	1	7	1	17	5	4	3	3 (1 with metro)	—	52
26—30	1	16	1	12	—	18	3	1	6	2 (1 with metro)	—	60
31—35	9	9	2	10	—	8	2	4	4	1		49
36—40	4	7	3	4	—	10	—	—	5	2 (1 with metro)	—	33
41—45	2	7	1	3	—	9	1	—	1	—	1	25
46—50	2	3	1	2	1	3	—	—	—	—	—	12
51—55	—	1	—	—	—	3	—	—	—	—	1	5
56—60	—	—	—	—	—	—	—		—	—	6	6
Total :	23	54	9	39	3	73	11	13	21	8	8	262

There where 19 cases (0.5%) of tuberculous endometritis leading to abnormal uterine bleeding. Tuberculous endometritis can cause bleeding of any type at any age (besides amenorrhoea and Oligomenorrhoea), Dommisse (1955) recorded menometrorrhagia or postmenopausal bleeding in 10% of cases of primary pelvic tuberculosis. Sutherland (1953) recorded its incidence as 1% in cases of uterine hemorrhage.

Nonspecific *cervicitis* (endocervicitis) can frequently cause irregular bleeding, contact bleeding and off and on spotting. When chronic cervicitis gives rise to cervical polyps, the tendency to metrorrhagia and intermenstrual bleeding etc. is greatly increased. This, my observation, is at variance with that of others (Jeffcoate 1969) who think that in chronic cervicitis excessive bleeding is rare except when cellulitis is present as well.

In the present study at Philadelphia General Hospital USA, (1957-58) there were a total of 558 cervical biopsies. 441 of these showed cervicitis with or without cervical polyp. The bleeding history of these 441 cases is given in table VI. This abnormal uterine bleeding may be due to cervicitis and/or other associated lesions.

Table VI

Pattern of bleeding in cervicitis with or without cervical polyp

Pattern of bleeding	P.G.H., USA (441 cases)	Present series (131 cases)	Total
Menorrhagia	40	29 (1 case of actinomycosis CX	69
Metrorrhagia or spotting	95 (1 with cervical polyp) (1 with Ca-in-situ)	40 (2 with Ca-in-Situ)	135
Polymenorrhoea	8	—	8
Menometrorrhagia	58 (3 with Ca-in-Situ) (1 with Ca-in-situ and leukoplakia)	4	62
Epimenorrhagia	3	—	3
Bleeding	92 (1 case of Tbc cervicitis (1 with Ca-in-situ)	42 (1 with Ca-in-Situ)	134
D. U. B.	26 (1 with cervical polyp)	1	27
Ameno/bleeding	10	3	13
Post abortion/Partum	9	1	10
Post-coital	32	1	33
Post coital + metro	2	—	2
Post-menopausal	66 (1 with leukeplakia) (1 with cervical polyp)	10	76

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Pelvic inflammations including salpingo-Ophoritis can cause abnormal uterine hemorrhage. There is menorrhagia (heavy and prolonged period), metrorrhagia and epimenorrhagia from change in cycle because ovaries are involved in pelvic congestion and infection (Jeffcoate 1969), the latter also interfering with ovulation from thickened ovarian capsule, as well as from associated ? endometritis and cervicitis.

In the present series, 15 cases of chronic salpingitis/tuboovarian abscess were identified, mostly in association with other lesions.

D. Uterine neoplasias, polyps and mechanical causes. *Adenomyosis* uteri frequently causes menorrhagia and metrorrhagia because of increased vascularity of the myometrium and endometrium, and perhaps ? Hyperestrinism (Hertig and Gore 1966 ; Jeffcoate 1969) and increasing bleeding surface.

In the present series, adenomyosis was found in the following combinations, in association with abnormal uterine bleeding :

Adenomyosis alone	45	Menorrhagia	20
With endometrial hyperplasia	10	Metrorrhagia	23
With endometrial fibroids	1	Menometrorrhagia	2
With fibroids	6	Epimenorrhagia	1
With endometritis & cervicitis	2	Bleeding	15
With cancer corpus	1	Ameno/bleeding	1
With cancer cervix	1	Postmenopausal bleeding	4
Total :—	65		65

Leiomyoma of uterus can frequently (in over 50% cases of fibroids) produce menorrhagia especially when these are submucosal (Jacobson and Enzer 1956) due to the *enlargment* of the surface (Haines and Taylor 1962) area of uterine mucosa, the *venous congestion* (resulting from myometrila pressure) delaying the occurrence of physiological thrombosis, prevented contractily of

uterine wall, hyperestrinism (fibroids and menorrhagia are both result of excessive or unopposed secretion of estrogen), higher incidence of cystic endometrial hyperplasia (Witherspoon 1935), increased vascularity of endometrium and rarely ? Ulceration of the overlying mucosa.

Leiomyomas can cause polymenorrhoea only when the tumour mass determines an increased blood supply to the ovary and metrorrhagia when submucous fibroids become polypoidal and ulcerated.

In the present series of 3847 cases, there were 179 (4.7%) cases of fibroid/ fibroids alone (3.3%) or in combination (1.4%) with other lesions (table II). The bleeding pattern of patients with fibroids with or without other lesions is restated in table VII.

Table VII

Fibroids and their bleeding pattern

Fibroids & their associations	Cases	Meno	Metro	Meno-metro	Bleeding	Amen bleeding	Polymenorrhoea	Postcoit	Postmenopausal
	126	51	21	15	32	3	1	1	2
Fibroid/fibroids/fibroid polyp, alone	1	1	—	—	—	—	—	—	—
Fibroid + end. hyperplasia + endometritis	8	2	1	—	5	—	—		—
Fibroid with cystic endometrial hyperplasia	1	—	—	1	—	—	—	—	—
,, + end. hyperplasia + end. polyp	1	—	—	—	1	—	—	—	—
,, + endom. hyperplasia + cervicitis	1	—	—	—	—	1	—	—	—
,, + endom. hyperplasia + adenomyosis	2	+	—	+	—	—	—	—	—
,, + R P. P.	1	1	—	—	—	—	—	—	—
,, + endomet. polyp	4	2	1	—	—	—	—	—	1
,, + salpingitis	1	—	—	1	—	—	—	—	—
,, + ,, + simple cyst.	2	1	—	1	—	—	—	—	—
,, + follicular cyst	1	1	—	—	—	—	—	—	—
,, + brenner's tumour	1	—	—	—	1	—	—	—	—
,, + simple cyst ovary	5	1	2	—	—	—	—	—	2
,, + adenomyosis	1	1	—	—	—	—	—	—	—
,, + ,, + cervicitis	3	1	—	1	—	1	—	—	—
,, + endometritis	1	1	—	—	—	—	—	—	—
,, + ,, + cervicitis	2	1	1	—		—	—	—	—
,, + salpingitis + endom.	1	—	—	—	—	—	—	—	1
,, + ,, + cervicitis	14	8	—	1	4	—	—	1	—
,, + cervicitis	2	—	—	2	—	—	—	—	—
,, + ,, + salpingitis	179	73	26	23	43	5	1	2	6
Total :—									

Other benign or malignant tumours of the uterus and cervix also cause abnormal uterine bleeding for similar reasons and mechanism, but there is an additional likelihood of extensive ulceration of erosion of a malignant tumour into a blood vessel. Cancer of the cervix is likely to cause intermittent spotting and postcoital bleeding. Cancer of the carpus often produces bloody discharge, intermittent spotting and then gross bleeding. Sutherland (1958) recorded meligaancy in 0.8% of curettings of patients with uterine hemorrhage.

Polypoidal benign tumours of the cervix and endometrium (endometrial polyps : cervical polyps) and malignant surface growths of uterus, cervix etc cause irregular bleeding or continuous bleeding when their tip is ulcerated.

Cervical polyps (localized heaping up of cervical mucosa ; usually pendunculated ; occasionally sessile ; single or multiple ; small or large, great majority arise fr. m endocervix) often cause frequent bleeding ("contact bleeding" of the type of early cervical carcinoma) or scanty metrorrhagia or scanty metrorrhagia or scanty intermenstrual bleeding or spotting on account of their vascularity, frequency of infection, vascular fragility and ulceration of surface mucosa (Novak & Woodruff 1962). The etiologic factor of endocervical polyps ls chronic endocervicitis in most cases. True adenomatous polyps are very rare (Saphir 1958). Cervica polyps occur in 2.5% of all women (Robbins 1967).

Endometrial polyps

Endometrial polyps are benign endometrial overgrowths which often arise by focal exaggeration of an endometrial hyperplasia. While small polyps are usually asymptomatic, larger ones become ulcerated and cause metrorrhagia and if the mural endometrium is hyperplastic, menorrhagia is usually present as well. While endometrial polyps first appear usually in the 4th decade (Hertig and Gore 1966) and achieve a peak incidence in 5th decade (Jeffcoate 1969) they may persist into and after menopause. Other uterine polypimet with are fibromyomatous polypi, adenomyomatous (protrusion) polypi and adenofibromatous polypi (Montgomery 1965 : Saphir 1958) and cause similar abnormal uterine bleeding. Some endomatrial polypi are ademomatoid overgrowths (true tumors) of lamina basalis, stalked or unstalked. Usually compression of the polyp by the uterine muscle may lead to severe engorgement of it and hemorrages (menorrhagia). In routine uterine curettings, the polyp may be missed to be brought out especially if located in cornual region Also the diagnosis may be missed if its distinctive histological features are unfamiliar to the histologist.

Hughesdon and Symmers (1966) are therefore, of the opinion that on this account many cases of menorrhagia may remain unexplained.

Sutherland (1950) recorded endometrial polyps in 1.1% of curettage specimens of patients with uterine hemorrhage.

Endometrial polyps can occur at any age but maximum incidence is at age 45—50 years. In about 20% of cases, they are multiple. The cause for these polyps which are local overgrowths is unclear and there is no relation to parity, infertility or estrogen excess. (The polyps which develop with cystic endometrial hyperplasia are related to estrogen excess).

In the present series (table II, VI), there were 72 cervical polyps and 3 adenomyomatous polyps with disposition and bleeding history as laid in table VIII. Fibroid polyps are excluded.

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Table VIII

Polyps and obnormal uterine bleeding
(Peshawar, Multan, Philadelphia)

Polyps	Nos	Menorrh-agia	Metrorrh-agia	Meno-metrorrh-agia	Bleeding	Postmeno-pausal	No bleed-ing record
Endometrial polyp	35	8	3	—	7	2	15
End. polyp + end. hyperplasia	3	—	1	1	1	—	—
End. poly + end hyp. + fibroid	1	—	—	1	—	—	—
,, ,, + fibroid	1	1	—	—	—	—	—
,, ,, + ch. endometritis	1	1	—	—	—	—	—
,, ,, + cervicitis	4	2	—	—	2	—	—
Adenomyomatous polyp	2	1	—	—	1	—	—
,, ,, + fibroid	1	—	—	—	1	—	—
Cervical polyp	51	7	12	2	4	7	19
,, ,, ÷ cervicitis	14	—	8	—	3	3	—
,, ,, + R. P. P.	1	—	—	—	1	—	—
,, ,, + cervicitis + endo-metritis	1	—	—	—	1	—	—
,, ,, + cervicitis + adeno-myosis	2	—	—	—	2	—	—
,, ,, + cervicitis + endo-met. hyperplasia	1	—	—	—	1	—	—
,, ,, + endometritis	1	—	—	—	1	—	—
,, ,, + cervicitis + Ca-in-situ + salpingitis	1	—	—	—	1	—	—
Total :—	110	20	22	4	26	11	27

There were 116 malignancies (Table II), 25 more not included already in P.G.H. cervical biopsy series (Table VI) and 15 more not already detailed (Table III) in endometria of patients who had history of bleeding, making, a total of 156. Their bleeding pattern is detailed in table IX.

Table IX

Uterine bleeding pattern in uterine malignancy (Peshawar, Multan, and Phil delphia)

Cancer	Number	Menorrhagia	Metrorrhagia	Meno metro	Bleeding	Amen bleeding	Postmenopausal bleeding	Postcoital
Fpidermoid Ca CX	90	4	12	1	40	1	27	5
Adenocarcinoma CX	4	—	—	—	3	—	1	
Epid. Ca CX + adenomyosis	1	—	1	—	—	—	—	—
Adenocarcinoma corpus	50	2	7	1	11	3	26	—
,, ,, + adenomyosis	1	—	1	—	—	—	—	—
Adenoacanthoma corpus	1	—	—	—	—	—	1	—
Chorion-carcinoma uterus	6	—	3	—	3	—	—	—
Leiomyosarcoma	3	3	—	—	—	—	—	—
Total :—	156	9	24	2	57	4	55	5

Myometrial hypertrophy (Robbins 1967) or Chronic symmetrical enlargement of the uterus may sometimes be associated with menorrhagia (excessive uterine bleeding) which is painless and ovular. There is generalized diffuse, symmetric enlargement of the myometrium and endometrium in the bulky uterus. Causes like "chronic inflammation" (chronic metritis), "fibrosis uteri" or "chronic subinvolution" have no proof and are discarded. The generalized uterine enlargement is now thought to be possible due to *normal largeness* of uterus, *idiopathic developmental hypertrophy* (causes ovular menorrhagia) or *excessive and prolonged estrogen stimulation* (bleeding as seen in metropathia hemorrhagia) or *chronic active or passive congestion from local* pelvic disease or anxiety states, psychological disturbances and unsatisfied sex urges etc. (the pelvic congestion fibrosis syndrome; ovular menorrhagia; ovular epimenorrhagia if ovaries share in pelvic congestion (Jeffcoate 1969).

The old view was that diffuse symenetrical uterine enlargement was caused by chronic subinvolution due to retroversion or infection etc. that there was a high collagen content of myometrium (Shoemaker and Kahler 1947) and corresponding regression of muscle leading to diminished contractility and that the consequent diminished ability to check menstrual bleeding lead to menorrhagia.

But Truemner and Kaump (1949) who studied 52 cases of chronic subinvolution found menorrhagia in less than 50%, increase in muscle cell diameter (work hypertrophy), unusually prominent vasculature of myometrium and no myometrial excessive fibrosis. The "Elastosis" is a natural accompaniment of parity (Shaw 1929). The condition has been found occasionally in women who have never borne children.

E. Ovarian lesions, neoplasias and hormonal conditions

Ovarin endometriosis can cause polymenorrhoea or polymenorrhagia. due to ovarian irritation (Jeffcoate 1969). Follicular cysts and estrogen producing ovarian tumours can cause acyclical-anovular bleeding from hyperestrinism, and myometrial hypertrophy and endometrial hyperplasia. Granulosa-theca cell tumours are usually functioning. 10% occur in children, 30% occur in reproductive life (cause amenorrhoea followed by profuse and continued bleeding) and 60% occur after menopause, especially in the 6th decade (cause resumption of uterine bleeding which is usually irregular and profuse) (Fallis 1964). Like the thecagranulosal tumours, *thecomas* secrete estrogens, which leads to cystic endometrial hyperplasia. Large tumours of the ovary with or without axial rotation can cause abnormal uterine bleeding from disturbed blood supply of uterus. Non-functional ovarian tumours may cause bleeding, especially postmanopausal, from mechanical stimulation of adjacent ovarian stroma to produce hormones.

Table X

Ovarian cysts, tumours and malignancies in association with uterine bleeding (Peshawar, Multan, Philadelphia)

	No.	Meno	Metro	Bleeding	Amen bleeding	Meno Metro rragia	Postmeno pausal bleeding
Follicular cyst ovary	1	1	—	—	—	—	—
Follicular cyst + cervicits + endom. hyperplasia	7	3	2	2	—	—	—
,, + endometritis + hyperplasia + fibroid	1	1	—	—	—	—	—
,, + salpingitis + fibroid + hyperplasia + adenomyosis	1	—	—	—	1	—	—
,, + cervicitis	3	2	—	1	—	—	—
,, + ,, + endometritis + adenomyosis + salpingitis	1	1	—	—	—	—	—
,, + estrin phase endometrium	1	1	—	—	—	—	—
,, endo. hyperplasia + fibroid + endometritis + cervicitis	1	1	—	—	—	—	—
,, + Adenomysis + fibroid +	1	1	—	—	—	—	—
,, + fibroid	2	1	—	—	—	1	—
Simple cyst + fibroid + salpingitis	1	1	—	—	—	—	—
simple cyst ovary + Ca-in-situ-cervix	1	—	1	—	—	—	—
Simple cyst	1	—	1	—	—	—	—
,, ,, + fibroid	1	—	1	—	—	—	—
Gronulosa cell tumour	2	—	1	—	—	—	1
Dysgerminoma + endometritis	1	1	—	—	—	—	—
Bremner tumour + fibroid	1	1	—	—	—	—	—
Mucinous cystoma	1	—	1	—	—	—	—
,, ,, + Adenomyosis + cercivitis	1	—	—	—	—	—	1
Serous cystoma	5	1	1	2	—	—	1
Dermoid cyst-ovary + Ca-in-situ CX	1	—	1	—	—	—	—
Papillary cystadenocarcinoma ovary	1	—	—	1	—	—	—
Serous cystadenoca ovary + Adenoca uterus + cervicitis	1	—	1	—	—	—	—
Mucinous cyst adenocarcinoma ovary	2	1	1	—	—	—	—
,, ,, + endometrial hyperplasia	1	—	—	1	—	—	—
Adenocarcinoma ovary	1	—	—	—	—	1	—
Total :—	41	17	11	7	1	2	3

Hyperestrogenism from exogenous estrogens administered fo. various therapeutic or other reasons are one of the commonest causes of menorrhagia and metrorrhagia (Jeffcoate 1969).

Injudicious use of estrogens about the time of menopause can cause excessive and prolonged bleeding due to estrogen induced cystic or adenomatous hyperplasia. Also estrogen withdrawal may produce bleeding in some cases (Tacchi 1958). Progesterone-breakthrough bleeding is a recognized entity (Masters and Atten 1948). Kahn et al (1965) reported severe uterine bleeding in two women following the prolonged use of Norethynodrel-Mestranol for contraceptive purposes. Luby (1965) noted troublesome post-partum or post-abortional uterine bleeding in a majority of patients after use of *depomedroxy progesterone* in treatment of threatened or habitual abortion, due to prolonged suppression of ovulation. *Hyperpituitarism* (e.g. early stages of acromegaly) can cause manorrhagia through overstimulation of ovaries with gonadotrophins.

Hypothyroidism causes menorrhagia or polymenorrhoea in 30-40% of cases.

Parenchymal liver disease and cirrhosis of liver can lead to menorrhagia or metrorrhagia through lack of inactivation of estrogrns in liver, disturbed estrogen-progesterone metabolism, pituitary depression (Pincus et al 1951) or nutritional deficiency.

F. Functional Uterine Bleeding

(*i*) *Functional epimenorrhoea and epimenorrhagia* is the commenest form of functional (Ovular) uterine bleeding and is due to a quicker ovarian cycle mostly from a *contraction of follicular phase* Most cases occur between 30 to 40 years of age. It is noted sometimes after *Pregnancy* due to failure of pituitary to return to normal in good time and this leading to excessive or abnormal stimulation of ovary. It is also noted during the few years *following menarche* and during tne few years *preceding the menopause.* Emotinal upsets and pelvic infections can cause this condition from congestion of ovary.

In the present series of 3847 cases of abnormal uterine bleeding, there were 98 (2.6%) with polymenorrhoea and polymenorrhagia, as clinically recorded The endometrium showed estrin phase in 42 cases, progestin phase in 24 cases (evidence of ovulation and possibility of functional bleeding, irregular ripening in 2 cases, endometrial hyperplasia in 6 cases, fibroids in one case, R.P.P. in 2 cases, chronic endometritis in 17 cases, and adenomyosis in 1 case.

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(*ii*) *Functional menorrhagia* (heavy and/or prolonged menses occuring at normal intarvals) of *irregular ripening* (*maturation*) *of the endometrium* is due to poor formation and poor function of the corpus luteum. Since the endometrium is without adequate hormonal support, slight bleeding or spotting occurs for several days before the proper flow starts. Menorrhagia results from more bleeding from stroma and vessels of endometrium. Endometrial curettings during menstruation reveal only patchy progestational changes (mixed endometrium) because the endometrium did not have normal uniform progestational effect after estrogenic stimulation, and unripe proliferative phase endometrium persists in some areas into the late 2nd half of the cycle. It is very rare (at the most 4% by serial sections of late progestin phase) apart from the normal mild variations just after ovulation (Noyes 1951) or except in case of admixture of relatively unresponsive endometrium of isthmic region among curettings of sercretary endometrium of corpus.

True mixture of secretary and proliferative endemetrium are sometimes noted in certain cases of abnormal bleeding occurring shortly after the menopause. Sutherland (1750) recorded irregular maturation in 2.6% of curettings of patients with uterine hemorrhoge. Most cases of "irregular ripening" are probably the unrecognized *endometrial polypi.* In the present series of 3847 cases, there were 25 cases (0.7%) with irregular maturation, two also showing chronic endometritis.

(*iii*) *Functional menorrhagia of irregular shedding* is said to be an important entity forming 15% of the menorrhagias (McLennan 1952). But Sutherland (1950) recorded irregular endometrial shedding in only 1.3% of cases of ulterine hemorrhage.

Slight bleeding continues intermittently for several days after the proper flow. Excessive prolonged bleeding is due to excessive vascularity and open areas of endometrium which do not heal quickly. In general there is *prolonged* and irregular menstruation (Hertig and Gore 1936). from piecemeal shedding during the course of getting rid of the whole. *Irregular shedding of endometrium* may arise from failure of secretary endometrium to desequestrate and regress or may be secondary to inadequate F. S. H. and estrogen production which is unable to suppress the corpus luteum and L. H. production. Bleeding and endometrial involution occur even though progesterome secretion continues. The condition can only be diagnosed from curettings obtained in late (5th day of bleeding) stages of menstruation.

The classical view is that the condition is caused through incomplete slow degeneration (delayed involution) of the corpus luteum (McKelvey and Samuels 1947 ; Brewer and Jones 1948), leading to abnormal persistence of functioning luteal tissue. (Shakhashir and Sommers 1956).

Recently, Sinykin et al (1956) have observed that the luteal phase of the cycle (as judged by temperature chart) is of normal length and have proposed the concept of "*breakthrough bleeding*" to explain this bleeding on basis of premature onset of menstruation. The menstruation *lasts longer* because it *begins earlier* in the cycle but ending at the same time as would normally have been the case. It is assumed that the cause is some *deficiency* of the corpus luteum.

The picture of the naturally occurring condition and the occurrence of bleeding from an actively progestational endometrium has been reproduced by giving luteotrophin (Holmstrom and Jones 1949) and by priming postmenopausal patients with estrogen and then administering decreasing doses of progesterone until "breakthrough bleeding" occurs (Masters and Magallon 1950).

Pathologically, there is a mosaic pattern of areas of variable degrees of retardation of shedding, involution, healing and maturity of endometrium (Hertig and Gore 1966). "*Chronic menstrual endometrium*" in which curettings show mingled fragments of endometrium in proliferative, secretory and menstrual phases, is one type of clinical irregular shedding described by Shakhashir and sommers (1956). In general, histologically, patchy progestational appearances persist in an endometrium which should be in early proliferative phase. Hughesdon and symmers (1966) describe the condition as "*irregular involution*" as there is extreme variation in the rate of involution of endometrial glands during menstruation, so that after a few days bleeding, glands in an early stage of involution are found side by side with glands in the late (secretary glands such as are normally found in the early stages of menstruation + darker simpler glands of the type ordinarily seen at a later stage in menstruation). Endometrial involution and endometrial shedding are associated phenomenon. Maximal cases are said to occur in the age group of 30-40 years with peak of incidence at age 40.

(*iv*) *Ovulation hemorrhage* is a common phenomenon in essentially normal cycles and may be mistaken for epimenorrhoea. It is usually small in amount and of short duration. It is the result of temporary estrogen deprivation (fall

in estrogen level) during the change over from the follicular to the luteal phase in the ovary.

G. Dyfunctional Uterine Bleeding

(*i*) *Threshold bleeding* This prolonged bleeding usually occurs in young girls or in woman aged 45-50. The ovary is producing enough estragen to reach the threshold for bleeding but not enough to produce a full proliferative phase. This underactivity marks the waxing and waning of ovarian activity. Histology reveals the endometrium to be thin and in poorly developed proliferative phase.

(*ii*) *Endometrial hyperphasia* (Metrophathia hemorrhagia of Schroder).

Dysfunctional uterine bleeding with irregular amovulatory cycles (*Metropathia hemorrhagica*) is a common problem of great frequency (Rogers 1963). The time and duration of bleeding is unpredicatable. It may last for days or 2-8 weeks. The bleeding may be heavy or light or a persistent staining. Episodes of bleeding are frequently interrupted by long or short periods of amenorrhoea (Rogers 1963). Although menorrhagia may occur but the usual pattern of this chronic anovulatory acyclical dysfunctional uterine bleeding is metrorrhagia or menometrorrhagia (Fallis 1964 ; Saphir 1958). In about 50% of cases the bleeding is preceded by a short period of amenorrhoea which coincides with a continuously high production of estrogen by the follicls (tc), but in contrast to abortion or ectopic pregnancy, the bleeding is painless (Jeffcoate 1969).

While sometimes the endometrial hyperplasia may be symptomless, Endometrial hyperplasia is found in 26.5% (Sutherland 1950) to 40% (Fluhmann 1956 ; Rogers 1963) of cases of abnormal uterine bleeding and the figures are higher (49%) for adolescent dysfunctional bleeding (Southam 1961). Salvatore (1958) recorded endometrial hyperplasia (cystic 50%, just hyperplasia 24% in 74% of cases of D. U. B. Commonest type of bleeding is a continuous loss and hence the name metropathia hemorrhagica.

Endometrial hyperplasia is caused by continuation of the follicular phase beyond the normal span, due to an abnormal persistence of unripened Graafian follicle/follicles due to failure of ovulation for a considerable time and the endometrium is subjected to an abnormally prolonged and excessive action of the estrogenic hormone.

There is absence of functioning corpus buteum because a ripened follicle

fails to rupture. The ovum dies and the follicle may go on to cyst formation. The follicle cystic or not, produces estrogen for a time. This acts on the uterus without being opposed by progesterone. The estrogen level may be continuous but moderate or intermittently high and low (Jeffocate 1969).

Anovulatory functional uterine bleeding is an exaggeration of the anovulatory cycle which is occasionally observed in most normal women, especially at puberty (Cooperman 1949), in association with infertility (Wong et al 1950), after partuitrition (Sharman 1951) and near monopause In the usual *anovulatory cycle, the ovarian follicle regresses* shortly after failure to rupture and ovulate and menses starts at approximately the expected time (Fallis 1964). But in some cases a succession of anovulatory cycles leads to eyptic transmormation of the mature unruptured ovarian follicles (Hughesdom and symmers 1966).

In patients of metropathia hemorrhagioa before the age of menopause, one or both ovaries show subcapsular cystic follicles or follicular cyst/cysts. Each ovary may contain mulliple small ½-2 cm dia cysts. Usually there is one large (often not more than 3 cm dia) follicular cyst in one ovary with several layers of cells of both the granulosa layer and theca interna layer present. Some cases show a feminizing tumour e.g. granulosa cell tumour or the coma and some only theca cell hyperplasia.

In postmenopausal cases of endometrial hyperplasia withuterine bleeding, the sources of estrogen responsible for the condition are granulosa cell tumour or theca granulosal tumour or thecoma or hilum cell hyperplasia or ovarian cortical hyperplasia (Roddick and Greene 1958) or adrenals (in case ovaries are normal)

The cause of failure of rupture of ripened follicle and ovulation may be a primary disorder of anterior pituitary-Ovarian-endometrial mechanism (Paschkis et al 1951) with abnormal production of gonadotrophin stimulus (Montgomery 1965 ; Jeffcoate 1969). According to Fallis (1964) there may be excessive gonadotrophic stimulation of the ovary from excessive production of F. S. H. (? why) (with development of multiple follicles) plus inadequate secretion of lutenizing hormone which normally induces ovulation.

Occasional cases Stein-Leventhal polcystic ovaries develop endometrial hyperplasia and menorrhagia (Jeffcoate 1969) and excessive bleeding (Jocobs and Friedman 1961). Goldzieher and Green (1962) recorded that 21% of cases of polycystic ovaries show functional bleeding. Sommers and Wadman (1956)

recorded that endometrial hyperplasia occurred in half the cases, while Jacobs and Friedman (1961) found endometrial hyperplasia in 66% (8/12).

Endometrial hyperplasia in the conventional histological sense is not a must in every case of D. U. B. Greenblatt (1953) and Southam (1961) recorded that 85-90% of patients with dysfunctional menorrhagia bleed from a *persistent* proliferative endometrium, which is not necessarily hyperplastic.

The reason for bleeding is also unclear. It is probably not due to hyperestrinism as bleeding may occur with even a normal or a decreased amount of estrogen acting for too long a period of time with absence of progesterone. So, just straight continued action of estrogens unopposed by progestin is not the answer, An insufficient or wavering titer of estrogen is an inadequate stimulus for the growth and maintenance of endometrium (Rogers 1963). When the level of estrogen declines sufficiently, the endometrium regresses and undergoes surface necrosis rather than full scale desquamation of normal endometrium (McArthur 1953). The resultant bleeding which varie. in intensity and duration persists until sufficient estrogen stimulus again produces endometrial growth (Zucherman 1941 ; Phelps 1946).

The later idea of a decreased estrogen stimulus has been refuted by the finding of urinary estrogen out put levels (fairly constant and high levels) by Brown et al (1959) (This may be true for ovarian tumour cases). Even then, it is vastly held that it is a *withdrawal bleeding* from a temporary sharp absolute drop in the blood levels of estrogens from feed-back mechanism acting on pituitary and resulting in decreased F. S. H. production (Novak and Woodruff 1962 ; Jeffcoate 1969) or a relative *inadequacy of estrogens* when endometrium grows so thick that the available supply of estrogens is relatively inadequate to maintain the hyperplastic endometrium (Fallis 1964 ; Jeffcoate 1969).

The estrogen-fall or estrogen-withdrawal theory of bleeding is supported by following observations :

(*i*) Normal menses is induced by reduction in progesterone due to degeneration of corpus luteum at a time when estrogen level is also low (Montgomery (1965).

(*ii*) Removal of ovaries of macaque is inevitably followed by a bout of uterine bleeding (Allen 1927).

(*iii*) Administration of estrogens immediately after above castration postpones the withdrawal bleeding until the estrogens are discontinued (Engle 1935).

(*iv*) If breakthrough bleeding occurs during the above estrogen therapy, arrest of bleeding is effected by doubling the dosage (Greenblatt 1959).

(*v*) If progesterone is given after cessation of estrogen administration above, bleeding will occur only after the progesterone is discontinued (Corner 1942 ; Zuckerman 1937).

(*vi*) Bleeding does not take place in ovariectomised women as long as injections of estrogens are continued but occurs on cassation of injections (Montgomery 1965).

Since bleeding occurs only when there is a *decreased level of estrogen*, prolonged nonbleeding phases occur when there is a sustained hormonal stimulus. If estrogen is being produced irregularly, estrogen-withdrawal bleeding may occur more frequently (polymenorrhoea). If renewed estrogen production is slow, the bleeding may be prolonged or excessive (*menorrhagia*). In some cases, bleeding may be irregular without definite pattern (*metrorrhagia*) (Paschkis et al 1954). *Grossly*, there is generalized increase in thickness and quantity of the endometrium which has a smooth and shiny surface.

In well deveioped cases, the mucosal surface is uneven and thrown into folds, producing sessile or/and pedunculated soft red moist polyps. In the most pronounced cases, endometrium is abundant and polypoid (Saphir 1958 ; Jeffcoate 1969). This thickened polypoid endometrium usually occurs in the upper three fourths of the uterine cavity. The myometrium is also hypertrophied (Hughesdon and Symmers 1966). Microscopically, there is hyperplasia of the surfaee and glandular epithelium as well as of the stroma of the endometrium. The endometrium is in proliferative phase. Almost always there is no evidence of progestin (secretary) effect, the endometium being in (non-secretary) estrin phase.

Very unusually, a few subnuclear vacuoles may be noted in some gland but these may be due to the earliest part of a random abortive luteal phase or iatrogenic. In any case there are rare, patchy and focal and Engle and Smith

(1938) could produce these in monkeys with prolonged overdosage of estrogens. The endometrial glands are bigger and irregular (disparity of size and shape) and the increased number of cells lining them are larger than normal. However, the changes are usually patchy in distribution (Haines and Taylor 1962).

The appearance of large, often cystic glands, lying said by said with smaller glands, has been termed "Swiss-Cheese pattern" (Novak and Martztlof 1924 ; Sharoder 1954) diagnostic of cystic glandular hyperplasia. The cystic glands are usually lined by single layer of cuboidal cells and the smaller glands by one or more layers (stratification) of epithelial cells. It is a true hyperplasia, i.e. more glands and more lining cells (Saphir 1958).

The lumen of the glands is usually empty but may contain a variable amount of an eosinophilic secretion (M. P. S. and glycogen) with or without red cells, monocytes (macrophages) and epithelial debris (Type I secretion of Bartelmez and Bensley 1932 in contrast to type II serretion of luteal phase : Bartelmaz & Bensley 1932).

The stromal proliferation is evidenced by patches of hyperplastic stroma of variable extent (separating the endometrial glands) composed of tightly packed large plump spindle shaped stromal cells with plump oval nuclei and frequent moitotic figures.

The stroma may show dilated capillaries and may contain fibrin thrombi. There are large thin walled stromal sinusoids from where bleeding occurs (Hertig and Gore 1966). Localized areas of necrobiosis (areas with marked degenerative change with round cellular infiltration and numerous thromboses of blood vessels) have been described by Schroder (1954).

Uterine bleeding is a prominent clinical feature of endometrial hyperplasia but the degree of observed endometrial hyperplasia is not necessarily related to the severity of uterine bleeding (Haines and Taylor 1962 ; Novak and Woodruff 1962). Hyperplasia itself is not a cause of bleeding but the hormonal abnormality is. The growth response is not necessarily parallel with bleeding propensities. Therefore, cases of anovulatory dyfunctional bleeding show hyperplasia of all grades and even an estrin phase endometrium.

Curettings may be scanty (in case in which the bulk of endometrium has just been shed) and may show a simple probiferative pattern (endometrium from basal zone). The degree of hyperplasia of the endometrium is dependent upon

the amount of estrogenic stimulus, the duration of it and on the degree of receptivity of the individual endometrium (Novak and Woodruff I962).

Atypical hyperplasia (adenomatous hyperplasia of Hertig's type ; glandular endometrial hyperplasia in contrast to cystic endometrial hyperplasia) is sometimes met with.

The changes (may be present in whole or part or polyp or patches of endometrium) consist of crowding of glands and irregularity of their shape (but cystic changes are absent or are minial) The glandular epithelial cells are large (lager than in cystic hyperplasia), reduplicated in irregular layers and posses hyperchrometic nuclei with freqnent mitotic figures. The cytoplasm is sometimes pale or clear in some of the glands.

Salvatore (1958) has tried to identify the physical basis in D. U. B. of this type. He *found segmental changes of* arteriosclerotic type in endometrial spiral arterioles, in greater percentage and degree as compared to normal endometria He thinks that such vessels do not contract significantly and therefore, menorrhagia occurs. Also the arterioles with thick walls detach more slowly, producing irregular shedding and prolonged menstrual bleeding.

The subject of coexistence of endometrial hyperplasia and uterine myomas is important but controversial. If there was a high correlation one would expect to believe in estrogenic causative factor of Leimyomatogenesis. While several author record a high coexistence, others do not.

Endometrial hyperplasia may occur in any age from menarche to menopause (Fallis 1964). It is quite common at or soon after puberty (age group 14-20 years) as ovulation is not yet well established at menarche (Rogers 1963 ; Jeffeoate 1969). According to Fallis (1964) 15% of cases occur at puberty.

Endometrial hyperplasia is the commenest lesion in "functional" bleeding in girls under the age of 20 years (To Linde 1950). According to Fallis (1964) 35% occur during reproductive life, especially following a pregnancy or during episodes of pelvic infiammatory disease. The highest incidence is found in premenopause (Shaw 1929), as before menopause ovulation has become less frequent. Schroder (1954) recorded a proportion equal to 36.8% (2202/3270) for age group 46-50 years. According to Fallis (1964) 50% of cases occur in the 5th decade.

While Schroder (1954) considered that endometrial hyperplasia is *rare after menopause*, Haines and Taylor (1962) and Novak and Yui (1936) record it not to be so infrequent after menopause. The latter authors recorded a proportion of 1.9% (15/804) in postmenopause of long standing.

In the present series of 3847 cases, there were 698 cases of endometrial hyperplasia (Table II,XI) giving an incidence of 18.1% in materials from cases of abnormal uterine bleeding. The bleeding pattern of these cases is detailed in Table XI. Previously 1966, 1967, 1968), I had reported incidence of endometrial hyperplasia in Peshawar material (bleeding or no bleeding) and curettings as 7.3% to 8.4%. Therefore, the presently reported incidence of 18.1% in Peshawar and Multan and some part of Philadelphia material and of 18.4% (690 cases out of 3747 curettings) in Peshawar and Multan material alone is interesting.

Table XI

Histologic diagnosis of cystic endometrial hyperplasia and clinical pattern of uterine bleeding in the present series of 3847 cases

Cystic endometrial hyperplasia	No.	Meno	Metro	Poly	Metromeno	Polymeno	Bleeding	Amen bleed	Post abortion or postpartal	Postmeno pausal
alone	643	114	115	2	39	3	322	27	8	13
with endometritis	19	4	3	—	—	—	10	—	2	—
with cervicitis	14	5	2	—	—	—	6	—	—	1
with fibroid	9	2	1	—	1	—	5	—	1	—
,, ,, and endometritis	1	1	—	—	—	—	—	—	—	—
,, fibroid and cervicitis	1	—	—	—	—	—	1	—	—	—
with adenomyosis	10	2	3	—	—	1	3	—	—	1
,, adenomyosis & fibroid	1	—	—	—	—	—	—	—	—	—
Total :—	698	128	124	2	40	4	347	28	10	15

While the overall incidence of endometrial hyperplasia in Pakistani material (Peshawar and Multan) is near 8% in endometrial curettings (Farooki 1966, 1967 1968) the frequency was (table III) only 1.1% in 1957, 1958 curettings at the Philadelphia General Hospital USA from patients of all types (bleeding or not bleeding). The frequency appears to be high in our patients.

H. Postmenopausal bleeding

Postmenopausal uterine bleeding (½ to 1 year after establishment of menopause) is somewhat of a class by ifself which has acquired special attention. The listed causes (Jeffcoate 1969) are indiscriminate use of estrogens, malignant tumours of uterus (3% of cases), benign tumours of uterus, ovarian tumours (estrogen producers ; large tumours affecting vascularity of uterus ; "misfit tumours"), senile endometritis and pyometra, metropathia hemorrhagica, hypertension, diseases of-blood and capillaries and foreign bodies.

Postmenopausal bleeding is generally considered to be due to gynecologic cancer but there are several in which no cancer is identified (Paloucek et al 1967) Antoine (1966) recorded 53.1% malignancies and 46.9% benign lesions.

Table XII compares the histopathological findings in 91 postmenopausal cases of the present series with those of other authors.

Table XII

Pathology of Postmenopausal bleeding

	Present series 91 cases	Adamson et all (1957) 745 cases	Benzi(196) 210 cases	Antoine (1966) 782 cases
N. A. D. Endometrium	11 (2 with prolapse)	134 150 (prolapse) 30 (estrogens)	18 12 (prolapse) 12 (estrogen withdrawal	—
endomet. hyperplasia	13	—	12	28
end. hyper + cervicitis	1	—	—	—
,, ,, + adenomyosis	1	—	—	—
Adenomyosis + cervicitis	1	—	—	—
Fibroids	3 (1 with salpingitis)	22	—	—
,, + adenomyosis	2	—	—	—
Endometrial polyp	1	—	—	—
Cervical polyp	4	79	48	—
Endometritis	15	8	48	—
Cervicitis with or without Polyp	6	66	—	—
Endometritis cervicitis	1	—	—	—
Fibroid + endometritis + cervicitis	1 with salping	—	—	—
Cancer corpus	11	82	21	107
Cancer cervix	20	174	17	260
Others or Miscellaneous unclassified		Caruncles (26); ovarian tumours (11) vaginitis (95); severe H.T. (21); Malig ovarian tumours (14) cancer vagina (5) cancer vulva (9); cancer rectum (3) & cancer urethra (1)	unknown (12) other cancers (10)	unspecified benign lesions (339)

Hysterectomies for Uterine bleeding

Table XIII recorded separately, the pattern of bleeding and lesions found in hysterectony cases at Peshawar, Multan and Philadelphia General Hospital, USA. Only those cases are naturally included in which history of bleeding was recorded in surgical pathology records. There must be many more which may have been missed. The bleeding in these cases was the major or minor symptom, occuring alone or alongwith other signs, symptoms and indications.

Table XIII

Bleeding pattern and histopathologic diagnosis in 263 hysterectomy cases at Peshawar, Multan and P. G. H. USA.

Pathologic diagnosis	Meno	Metro	Menometro polymeno	Bleeding or continuous bleeding	D. U. B.	Ameno-bleeding	Post natal or post abortional	Postcoital bleeding	Postmenopausal	Total
Cervicitis + endomet. polyp	1	1	1	—	—	—	—	—	—	3
,, + ,, + Ca-in-situ + T.O. abscess	—	—	—	1	—	—	—	—	—	1
,, + adenomyosis + endotritis	1	—	—	—	—	—	—	—	—	1
,, + ,, + ,, + salpingitis + follic cyst	1	—	—	—	—	—	—	—	—	1
,, + ch. endometritis	8	2	—	1	—	—	—	—	—	11
,, + Adenomyosis	3	—	—	2	2	—	—	—	—	12
,, + ,, + mucinous cystoma	—	—	—	—	—	—	—	—	1	1
,, + End. hyperplasia	2	2	—	3	1	—	—	—	1	9
,, + ,, ,, + follicular cyst	2	1	—	—	1	—	—	—	—	4
,, + ,, ,, + adenomysis	2	—	1	—	1	—	—	—	—	4

1	2	3	4	5	6	7	8	9	10	11
,, + Cervical polyp	—	—	—	1	—	—	—	—	—	1
,, + Fibroids	10	1	1	4	1	—	—	1	1	19
,, + ,, + T.O. abscess	—	—	2	—	—	—	—	—	—	2
,, + ,, + Cervical polyp	1	—	—	1	—	—	—	—	—	2
Cervicitis + fibroid + endomet. + endometrial hyperplasia + follicular cyst	1	—	—	—	—	—	—	—	—	1
Cervicitis + fibroid + end. hyperplasia	—	—	—	1	—	—	—	—	—	—
Cervicitis	2	1	—	1	—	—	—	—	—	4
,, + follicular cyst	1	—	—	1	1	—	—	—	—	3
,, + cervical polyp	—	—	—	—	—	—	—	—	1	1
,, + endometritis	8	1	—	1	—	—	—	—	—	10
,, + fibroid + ademomyosis	1	—	—	—	—	—	—	—	—	1
,, + fibroid + adenomyosis + follicular cyst	1	—	—	—	—	—	—	—	—	1
,, + fibroid + adenomyosis + salpingitis	—	—	—	—	—	—	—	—	1	1
,, + endometritis + fibroid	1	—	—	—	—	—	—	—	—	1
,, + Ca-in-situ + dermoid cyst	—	1	—	—	—	—	—	—	—	1
,, + ,, ,, + simple cyst	—	—	—	1	—	—	—	—	—	1
Endomet. hyperplasia	2	1	1	—	3	—	—	—	—	7
,, + endometritis	3	3	—	3	—	—	—	—	—	9
,, + Adenomyosis	—	3	—	7	1	—	—	—	—	11
,, + fibroids	2	—	—	3	—	—	—	—	—	5
,, + endomet. polyp	—	—	—	1	—	—	—	—	—	1
,, + ,, + fibroid	—	—	1	—	—	—	—	—	—	1

	2	3	4	5	6	7	8	9	10	11
Fibroids	24	7	16	18	—	1	—	—	2	68
Fibroids + adenomyosis	1	3	—	1	—	—	—	—	2	7
,, + endometrial polyp	—	—	—	—	—	—	—	—	1	1
,, + salpingitis	2	1	—	—	—	—	—	—	1	4
,, + endometritis	1	—	—	—	—	—	—	—	—	1
,, + simple cyst ovary	—	—	—	—	1	—	—	—	—	1
,, + follicular cyst ,,	1	—	1	—	—	—	—	—	—	2
,, + endometritis + salpingitis	1	1	—	—	—	—	—	—	—	2
,, + incomplete abortion	—	—	1	—	—	—	—	—	—	1
,, + salpingitis + simple cyst ovary	1	—	—	—	—	—	—	—	—	1
Fibroid + Brenner tumour	1	—	—	—	—	—	—	—	—	1
Adenomyosis	—	3	1	1	—	—	—	—	1	6
,, + fibroid + End. hyperplasia	1	—	—	—	—	—	—	—	—	1
Adenomyosis + fibroid + endometrial hyperplasia + salpingitis + follicular cyst	—	1	—	—	Nil	1	—	—	—	1
Endometritis	—	1	—	—	—	—	—	—	—	1
,, + myometritis	—	—	1	—	—	—	—	—	—	1
,, + salpingitis	—	1	—	—	—	—	—	—	—	1
Endometritis + dysgerminoma	1	—	—	—	—	—	—	—	—	1
Bicornuate uterus	—	1	—	—	—	—	—	—	—	1
N. A. D. (2 for prolopse)	1	4	—	2	—	—	—	—	2	9
Chorioadenoma destrueus	—	—	—	1	—	1	—	—	—	2
Uterus after abortion & optive trauma	—	—	—	—	—	—	1	—	—	1

	1	2	3	4	5	6	7	8	9	10	11
Haydatidiform mole		—	1	—	—	—	—	—	—	—	1
Postpartum with thrombosis & hge		—	—	—	—	—	—	1	—	—	1
Endometrial polyp		—	—	—	—	—	—	—	—	1	1
Follicular cyst ovary		1	—	—	—	—	—	—	—	—	1
Granulosa cell tumour ovary		—	—	—	—	—	—	—	—	1	1
Serous cystoma ovary		—	—	—	—	1	—	—	—	1	2
Endometrial hyperplasia + mucinous cystadenocarcinoma ovary		—	—	—	1	—	—	—	—	—	1
Epid. Ca Cx + adenomyosis		—	1	1	—	—	—	—	—	—	2
Cherion Carcinoma		—	—	—	—	—	1	1	—	—	2
Adenocarcinoma corpus		—	—	—	—	—	—	—	—	4	4
Adenocal Corpus+Adenomyosis		—	—	—	1	—	—	—	—	—	1
,, ,, + cervicitis + serous cystadenocarcinoma ovary		—	1	—	—	—	—	—	—	—	1
Total :—		89	46	28	58	13	4	3	1	21	263

Table XIV gives an analysis of major lesions responsible for abnormal uterine bleeding in 3847 cases of the present series, arranged by age groups. Uterine malignancy was responsible in 32/97 (33%) of postmenopausal and 8.5% of premenopausal group (table XIV).

Table XIV

Major lesions by age groups in 3847 cases with abnormal uterine bleeding (Peshawar, Multan and Philadelphia General Hospital USA

Age (year s)	Endometrial hyper plasia with or without polyp endometritis or cervicitis	Irregular maturation	Estin-phase progestin phase N. A. D.	Aberrations of pregnancy	Endometritis cervicitis or both with or without polyp	Tuberculous endometritis	Fibroid, Polyp with or without endometritis cervicitis
16—20	19	2	107	33	22	—	—
21—40	521	19	1353	449	428	19	107
41—45	84	2	147	14	39	—	28
46—50	36	1	74	5	28 (1 with Ca-in-situ)	—	21
51—60	16	1	15	—	15	—	5
Total :—	676	25	1696	501	532	19	161

Endometrial hyperplasia fibroid	End. hyperplasia + fibroid + Adenomyosis	Adenomyosis with or without cervicitis or polyp	Endometrial hyperplasia + Adenomyosis	Polyps-cervical or endometrial	Adenomyosis + fibroid	Uterine malignancy	Tota
—	Nil	1	—	—	—	—	184
5	—	31	—	28	1	41 (1 with adenomyosis)	3002
2	—	9	6	5	3	26	365
1	1	4	3	8	—	17 (1 with adenomyosia	199
3	—	2	1	5	2	32	97
11	1	47	10	46	6	116	3847

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DIETARY INTAKE OF SEVENTEEN PREGNANT WOMEN AT ISLAMABAD

By

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Introduction

No report of dietary intake of pregnant women in Pakistan could be found in the literature. Nutrition Survey report available to date (Nutrition Survey of East Pakistan 1966 and West Pakistan 1970) only gives per caput dietary intake for the family which includes the pregnant women and therefore does not help very much to ascertain individual dietary intake of pregnant women who are nutritionally the most vulnerable group among the adult population.

Individual dietary survey for pregnant women is very difficult to conduct in Pakistan because of the eating and culinary habits of the family. As a result no attempt has yet been made to ascertain their dietary intake. Recording and weighing of food by the individual women is also impracticable because of the fact that literacy rate specially among women is very low and as a result data collected may not be quite satisfactory. This report is based on a preliminary survey of dietary intake of pregnant women at Islamabad by the dietary staff of the Nutrition Division of the National Health Laboratories.

Material and Method

Sample comprises 17 pregnant women of different socioeconomic group. All heads of the family are Government servants of different categories with income ranging between Rs. 100 to Rs. 500. Only in one family the income

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was Rs, 1050. The average family size of the group is four with per head income of Rs. 78. Excepting one Primipara all women included in the study were multipara. All of them were having normal pregnancies with age ranging between 16—35 years. The average duration of pregnancy was 7 months.

Food weighing method was used for the study. The food for the whole family was first weighed raw. When the foodstuffs are cooked, they are weighed again. The different raw ingredients that goes in making a curry are weighed separately so as to enable calculations to be made later. A portion of the cooked food earmarked to be eaten by the pregnant women is weighed and on completion of eating the leftovers weighed again and subtracted from the original earmarked portion to get her actual consumption. The weighing starts with breakfast and ends with supper in a particular day. Consecutively 3 days foods were weighed.

Calculations

From the raw and cooked weight of food amount of water used in cooking was calculated and percentage of moisture worked out. Cooked weight of food is then converted back to raw weight and calculation of nutrients made from raw food using international food tables (FAO 1954). For calculation in case of curry eaten amount of water was subtracted and calculation made on the basis of proportion of individual item that went into the preparation of the curry. This procedure has obvious limitations but was nevertheless had to be used in the absence of any other satisfactory method. The averages for 3 days were calculated according to the recommendations of FAO/WHO.

Results

The results of the survey are set out in Fig. I and II which show that the diet of the pregnant women surveyed is deficient in almost all the nutrients with the lone exception of vitamin B_1. Vitamin A, riboflavin and calcium intake by the pregnant women are dangerously low. Intake of calorie and vitamin C is also quite unsatisfactory.

When the consumption of individual women was considered (Table I) it became evident that 100% of the women consumed less vitamin A and vitamin B_2, and 82—94% consumed less protein, calcium and iron than recommended. Seventy to 88% consumed less than 70% of the recommended allowance of calcium, vitamin A and B_2. Similarly 20—40% of them had intake less than

Table I

Mean Percentage Distribution of dietary intake compared with recommended allowance at arbitrarily selected levels

		Calo-ries	Pro-tein	Cal-cium	Iron	Vit. A	Vit. B_1	Vit. B2	Niacin	Vit. C
Upto & above Recommended Allowance.	%	11.76	17.66	5.88	17.64	Nil	76.47	Nil	29.41	35.30
	No.	(2)	(3)	(1)	(3)		(13)		(5)	(6)
Below 100% Recommended Allowance	%	88.24	82.34	94.12	82.36	100%	23.53	100%	70.49	64.70
	No.	(15)	(14)	(16)	(14)	(17)	(4)	(17)	(12)	(11)
Below 70% of the Recommended Allowance	%	41.20	35.29	88.24	23.53	70.60	Nil	88.23	29.43	41.17
	No.	(7)	(6)	(15)	(4)	(12)		(15)	(5)	(7)

70% of the recommended allowances for calories, protein, niacin and vitamin C. Thus the result showed clearly that the dietary intake of pregnant women is very unsatisfactory in the group studied.

Discussions

When the result of the present study is compared with the dietary intake of urban population of West Pakistan (Table II), no appreciable difference except in the case of calcium, vitamin A and vitamin C could be found. That is to say that pregnant women consume almost the same diet taken by other members of the family. Three reports from South India (Pasricha 1958, Leela Ayenger 1962 and Venkatachalam 1962) which presented the dietary intake of pregnant women gave figures much lower than reported in the present study. However, a study reported by Bagchi and Bose 1962 from Calcutta showed almost similar intake for protein and calories for the poor socio-economic group. Much higher intake was reported for well to do pregnant women of Calcutta. A summary of results of these studies is set out in Table II. The variations reported may be due partly to socio-economic and cultural factors prevalent in the different localities studied and partly due to the methods used in the survey. Unfortunately due to lack of details provided in the cited papers intake of all nutrients could not be compared.

Thus it can be seen that survey findings gave results comparable to similar studies done in Calcutta by Bagchi and Bose (1962). The number of subjects involved in the present study are small. However, even though number of subjects are small it is most likely to be more accurate because it is universally accepted that among the methods used for dietary survey weighing is more reliable and accurate than the questionnaire method used in other studies. The study will be further extended in near future.

SUMMARY

Dietary intake of 17 pregnant women is reported. The findings confirm that the diet of the pregnant women is deficient in almost all nutrients with the lone exception of vitamin B_1. It is hoped that the study will be further extended in near future.

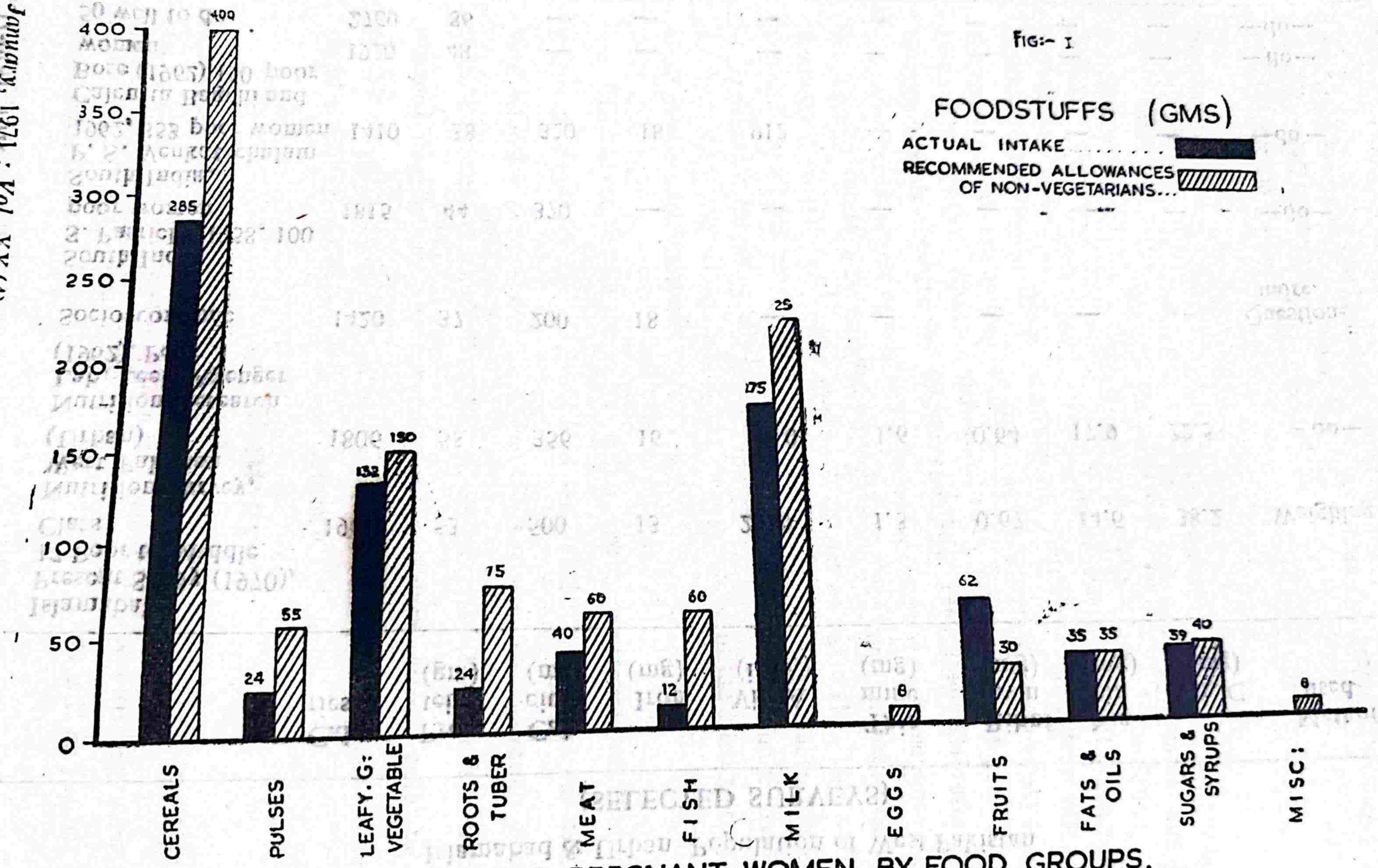
Table II

Mean daily intake of nutrients by pregnant women in South India, Calcutta, Islamabad & Urban Population of West Pakistan

(SELECTED SURVEYS)

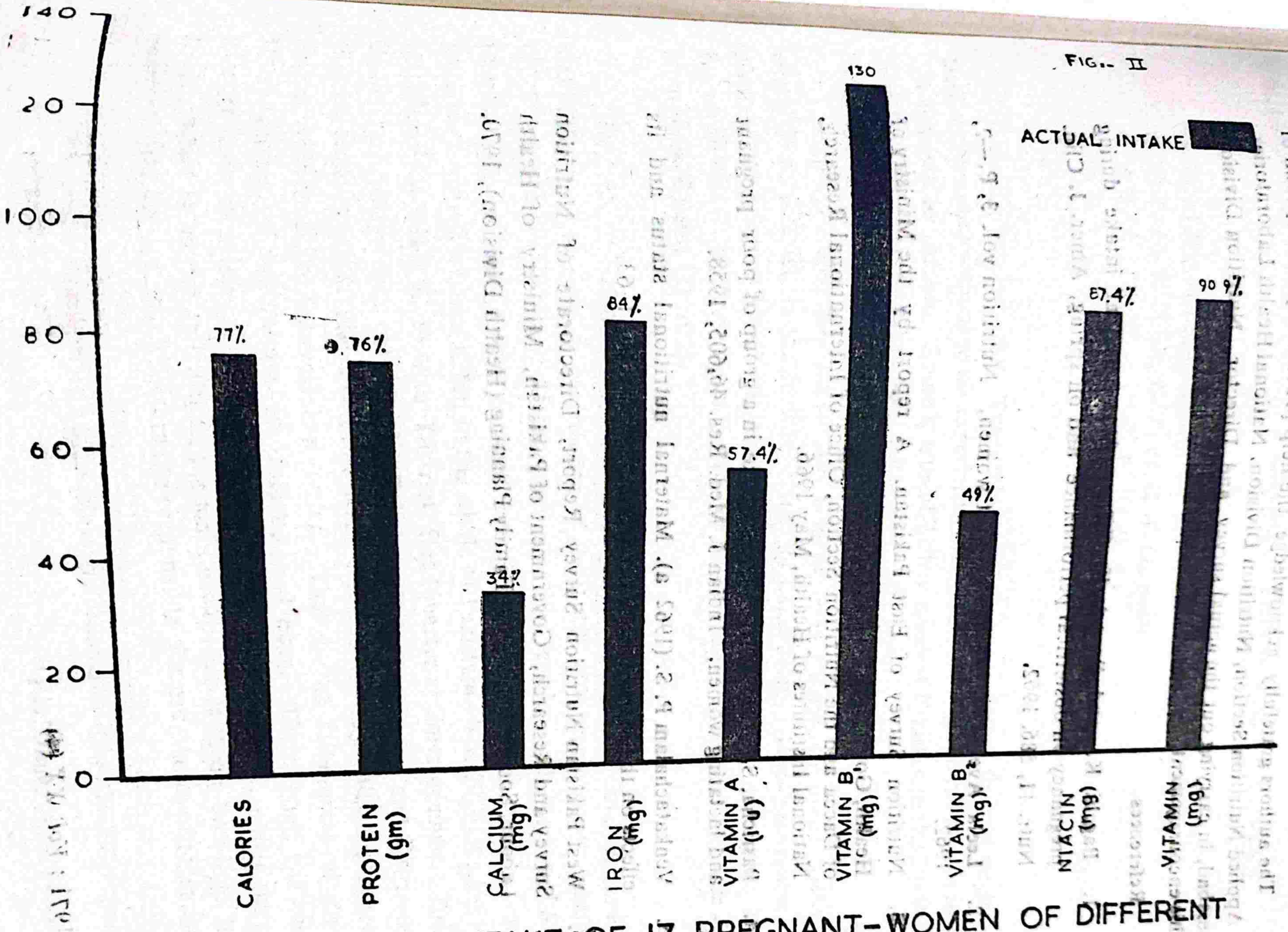
	Calo-ries	Pro-tein (gm)	Cal-cium (mg)	Iron (mg)	Vit. A (i. U.)	Thia-mine (mg)	Ribof-lavin (mg)	Nia-cin (mg)	Vit. C (mg)	Method used
Islamabad										
Present Study (1970), 17 Poor to Middle Class	1961	53	500	13	2210	1.3	0.67	14.6	38.2	Weighing
Nutrition Survey, West Pakistan (Urban)	1806	58	356	16	1610	1.6	0.64	17.9	22.5	—do—
Nutrition Research Lab. Leela Ayenger (1962), Poor										
Socioeconomic	1420	37	200	18	—	—	—	—	—	Question-naire.
South India										
S. Pasricha 1958, 100 poor women	1815	44	370	—	—	—	—	—	—	—do—
South India, P. S. Venkatachalam 1962, 353 poor women	1410	38	320	18	912	—	—	—	—	—do—
Calcutta Bagchi and Bose (1962) 150 poor women	1920	48	—	—	—	—	—	—	—	—do—
50 well to do	2760	86	—	—	—	—	—	—	—	—do—

FIG:- I



MEAN PER CAPITA INTAKE OF 17 PREGNANT WOMEN BY FOOD GROUPS.
★ RECOMMENDED ALLOWANGES TAKEN FROM DIET ATLAS OF INDIA 1969, 53.

FIG:- II



AVERAGE NUTRIENT INTAKE OF 17 PREGNANT-WOMEN OF DIFFERENT SOCIO-ECONOMIC GROUP STUDIED BY 3 DAYS FOOD-WEIGHING METHOD.

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PULMONARY TUBERCULOSIS RAVAGES TRIBAL AREA PAKISTAN

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It is a known fact that Tuberculosis takes an explosive form in virgin land, where the natives have no experience with white scourge in the past as in certain parts of Africa. Some is true of Tribal areas of Pakistan in the North West Frontier Region, adjoining Afghanistan and Persia.

Keeping in view the above fact, a survey was carried out in the Tribal area of Zhob District, in the remote corner of Baluchistan. Three years survey relates to the years 1964-65-66, and has been compared with the figures of Fatima Jinnah T. B. Sanitorium Quetta, of Baluchistan (Appendix : Table 1 & 2) Javed (1) & Khaliq (2) 1966.

Zhob District is one of the Backward parts of Quetta Division. It has an area of 10475 Sq Miles with population of about 100,000 persons, with density of 8-10 persons per sq mile. Males predominating females and literacy of 5.6 percent. The climate is dry, extreme cold in winter and hot in summer. Its position in respect of area is 6th in the whole of Pakistan. The District takes its name from the River Zhob with District Headquarter at Fortsandeman. Zhob District falls in the category of special area, with scanty rain and varies with altitude, from"4 to 10. Water of Zhob-River is difficult for agricultural purposes, because of its high banks and low water. Thus in the hilly areas of Zhob District. means of Agriculture are difficult and poor. There is only one main industry Chrome Mines at Hindubagh at the height of 7.200 feet, above the sea level. Apart from the agriculture, the main occupation is sheep breeding. It is observed that the following factors are responsible for prevalance of pulmonary

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and other forms of Tuberculosis in Tribal areas of Baluchistan, particularly in Zhob District.

1. **Heredity and Resistance**

Due to lack of past experience with the diseases the Natural Immunity is low and so the acquired immunity, atleast 60% of the papulation in still tuberculin-negative and hence at risk to catch infection.

2. **Age**

Most of the patients present symptoms of disease between the ages of 15-35 years that is during adult life.

3. **Sex and fertility rate**

Males are at more risk than females, the male members have to labour hard, firstly being the Head of the Family and secondly due to Custom of "WALWAR", in which the male has to struggle hard-through-out his life to pay the price of the woman, from Rupees 3,000 to Rs. 15,000. The marriages are thus late in life and the married couple has usually large family of 6-10 members.

4. **Race and Habits**

As a race the natives belong to Kakar [Mandokhel/Sherani/Bibr pathan tribes and are well built and sturdy persons, but are prone to catch infections, due to lack of immunity and less experience with the disease. They belong to the brave fighting class of Pathan, always armed with rifles and guns. However, they are simple in habits. Smoking is not common, but use of Tobacco "NASWAR" in the form of chewing is an integral part of their life.

5. **Climate**

The climate is extremely dry with scanty rainfall and extreme weather during summer and winter, snow fall is occasional. Due to dryness, the Haemoptysis and excessive menstruation etc. is more common in whole of Quetta Region.

6. **Contact**

Most of the patients give history of contact with open cases of Pulmonary T. B., although ignorant about the name of disease,

"PHTHISIS", they usually give history of having been in contact with a patient suffering from cough or low grade fever.

7. Housing

Although the density of population is 8-10 per sq mile, 5-15 members of the family live in katcha, low-lying houses (HUT TYPE), with small rooms. Gipsy (KHANA BADOSH) live in tents and keep on moving from place to place with their animals with whom they share accommodation. There is no proper ventilation except the main door of the house,

8. Sanitation and personal Hygiene

Due to lack of health education, illiteracy and poor economical conditions, the sanitation and personal Hygiene are of low standard. However men and women are clothed in heavy garments, though stink due to lack of washing clothes and not taking regular bath. There are no latrines, people squat outside in the adjoining fields.

9. Water supply

The water is obtained either from the streams running permanently or from "KAREZES" with channels of water connected under-ground and having opening at short distances. The water is neither chlorinated nor purified.

10. Economical Condition

The economical condition is poor. The main profession is sheep-breeding and agriculture. There is one Chrome Industry in Hindubagh a limited concern of M/s Amir Ali Fancy extending from Khanozai to Nasai, a mountainous range of fifty miles length. The income per capita is Rupees 200 However a few rich contractors and tribal chiefs share with the lot of the poor.

11. Industrial Lung Disease

About 500 labourers work in the Chrome mines at a time and hence a part of population is at risk, to catch respiratory infection. Most of the cases with bronchial catarrah ultimately leading to chronic bronchitis, are admitted every year in Civil Hospital Hindubagh. A

"*Pak. Jour. Hlth.* 1970-71"

survey has been started to estimate the incidence of tuberculosis in Pakistan Chrome Mines Hindubagh under arrangements with the Director of Chrome Mines and Medical Officer Civil Hospital Hindubagh. (Bilgrami) 1966-(3).

12. Food and Nutrition

The main diet consists of wheat-bread, potatoes, fresh and dry fruits. There is scarcity of vegetables. Protein intake in the form of Meat and eggs is low, although the area is noted for shooting of birds "Sisi and Chakours" serving delicious dishes ; and also "DEER and HURYAL" shooting. The running water contains certain minerals, good from Nutritive point of view.

13. Communicable and other Diseases

Smallpox and Cholera have been brought under control by the Public Health Department in Zhob District. Severe form of measles, whooping cough, influenza, bronchitis, tapeworm and round worm infestation, amoebic dysentry, chronic Malaria leading to severe anaemia, often lower resistance, making the patients more vulnerable to pulmonary Tuberculosis.

14. Road and Rail Communications

Although not industrialized except Chrome Mines in Hindubagh communication by road/rail, has immensely increased from the infected area of cities to virgin land of Zhob District, The area has been visited by British troops before partition, and now visited by Pakistani troops and Pawandas from Afghanistan in winter. All these movements certainly attribute to the spread of disease in the tribal area of Zhob District.

15. Discussion

Tuberculosis takes heavy toll in virgin land and Spreads like wildfire amongst the natives Meningeal, Miliary and Primary tuberculosis, Pleural effusions, Tuberculosis of glands, abdomen, bones and skin are common evils to start, in a virgin land. Too many deaths have not been noticed in Zhob District due to efficient Medical Organization in the District and lack of registration of cases in the distant

hills of Zhob District. Primary T. B. in the form of T. B. Glands, Pleural effusions, T. B. of skin and abdomen. and bone and joints including spinal T. B. have been observed in quite a number of cases, although cases of meningeal and Miliary T.B. (except only one,) have not been noticed due to lack of vigilance, lack of transportation facilities to the Hospitals/Clinics and inadequate X-ray and Laboratory facilities. Pulmonary T. B. had however spread like fire in both town and neighbouring rural areas during the past few years in the Zhob District ; but death rate was low in registered cases due to proper treatment with Chemotherapy and follow up treatment over a period of 1-2 years. This is due to the fact that such cases had not been treated before, and did not show drug resistance on clinical bases, except a few patients. Most of the patients with pulmonary T. B. presented with symptoms of cough with mucoid sputum, pain in the Chest, low grade fever, lassitude, loss of weight, anaemia, loss of appetite, and dyspepsia. Haemoptysis, Pleural effusion, secondary infection of the lung and asthmatic bronchitis, genitourinary Tuberculosis were common complications. Cases with Tuberculosis of glands, bone, abdomen, spinal T. B. and pleurisy responded well to Anti Tuberculosis chemotherapy, in addition to supportive therapy, Surgical interference was not necessary in most of cases.

Summary

305 patients of pulmonary T. B., were treated as indoor and 1330 Patients as outdoor in the tribal area of District Zhob during three years survey of 1964-65-66. Apart from this 90 patients indoor and 553 patients as outdoor, i e. a total of 643 patients, of other forms of T. B. were also treated during the above period. Both pulmonary and non-pulmonary cases responded well to Antituber-cular Chemotherapy, as majority of these cases had not been treated before and did not show drug resistance over a period of 1-2 years.

In contrast to this 1201 indoor patients and 41449 outdoor patients were treated in Fatima Jinnah T. B. Sanatorium Quetta from tribal areas of Baluchistan, most of the cases showed complications and quite a few showed drug resistance to chemotherapy and needed prolonged treatment both chemotherapy and surgical. Such cases had been treated before in their districts, prior to admission. The

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incidence of pulmanory T. B. in children was low, while the incidence in males was 1½ times that of females. The disease has spread like fire in the recent years, due to moderenization of Road-Rail communications in Zhob District. Military troops have also added to the increase of the number of T. B. cases and infection of virgin soil of Zhob District, from the infected area of the cities and towns of Pakistan. Pakistan Chrome mines is also blamed for the spread of the disease in Hindubagh area. Poor economical condition, old customs, superstitions, lack of health education, close contact, poor sanitation and housing, as well as infectious diseases during childhood and chronic Malaria during adult life are contributary factors. Remember the Motto "Save the Virgen land from white scourge"

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Appendix Table II

Abstract chart of tuberculosis from 1964 to 1966
Fatima Jinnah T. B. Sanatorium Quetta

For the year	Indoor patients			Outdoor patients		
	Male	Female		Male	Female	Child
1964	180	62	3	7299	4380	607
1965	240	178	6	8375	5420	708
1966	313	208	11	8290	5580	790
Total :—	733	448	20	23964	15380	2105

Table I

PROFORMA

Abstract Chart of Tuberculosis from 1964 to 1966 (District Zhob)

For the year	T. B. Lungs			T. B. Glands			T B. Spinal			T. B. of Skin			T. B. Abdominal		
	In	Out	Death	In	Out	Death	In	Out	Death	In	Out	Death	In	Out	Death
1964	98	542	3	4	104	—	3	2	—	8	12	—	4	—	—
1965	89	432	2	2	73	—	2	3	—	—	15	—	10	21	—
1966	118	356	2	1	51	—	5	7	—	—	4	—	—	15	—
1964,65,66	305	1330	7	7	228	—	10	12	—	8	31	—	14	36	—

For the year	Meningeal Miliary T. B.			Genitourinary T. B.			Pleurisy T. B.			Osteoarticulr T. B.			T.B. all other form including T B. Bone		
	In	Out	Death	In	Out	Death	In	Out	Death	In	Out	Death	In	Out	Death
1964	1	—	—	—	40	—	6	—	—	2	—	—	13	72	—
1965	—	—	—	—	18	—	2	8	—	3	4	—	11	41	—
1966	—	—	—	—	13	—	3	4	—	2	3	—	8	43	—
1964,65,66	1	—	—	—	71	—	11	12	—	7	7	—	32	156	—

2. Textbook of operative dentistry

By Hampson, E.L., M.D.S., F.D.S., H.D.D.. R.C.S. 2nd Ed. 1964. (Pp. 329, & 2.50) London ; William Heinmann Medical Books Ltd.

This book is based on lectures given at the School of Dentistry, University of Sheffield, England. The aetiology, pathology of dental diseases with pharmacology of drugs and chemistry of dental materials are described. The operative techniques dealt with are those which had proved of value by sufficient experience. Improved methods of making gold inlays and porcelain inlays are descibed in an excellent manner. It is well illustrated and would be of great value to the Dentist in practising conservative.

2. Aids to tropical hygiene and nursing

By Fream, W.C. S.R.N., B.T.A., S.E.R.T., S.T.D. 5th Ed. 1970 (Pp. 220, 16sh). London : Bailliere, Tindall and Cassell.

The present edition has been reprinted twice during the last two years and this reflects on the popularity this book has enjoyed. It is divided into eight chapters. The first chapter deals with the nutritional disorders in the tropics and includes some excellent diet schedules for different diseases. The other chapters describe the ailments associated with poor hygiene, parasites, insect vectors etc. It is a well written book and presents the material in a clear concise manner. It is recommended to the Nursesand those who are responsible for their traing.

ZAFFAR-UL-AZIZ
M. B., B. S. (Pb,), D. Bact. (London),
M.R.C. Path. (London). F. I. C. S.

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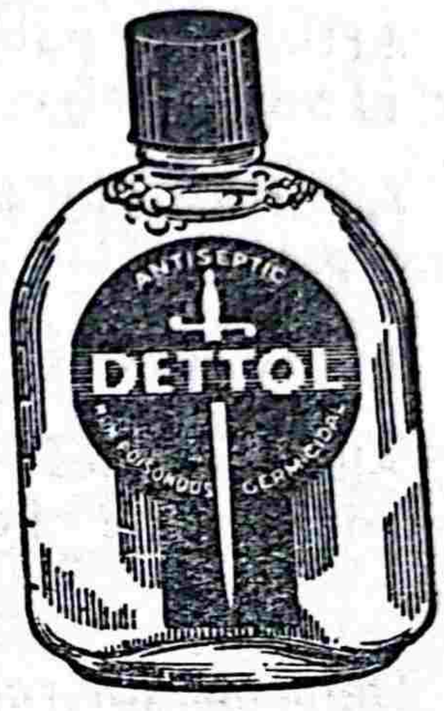

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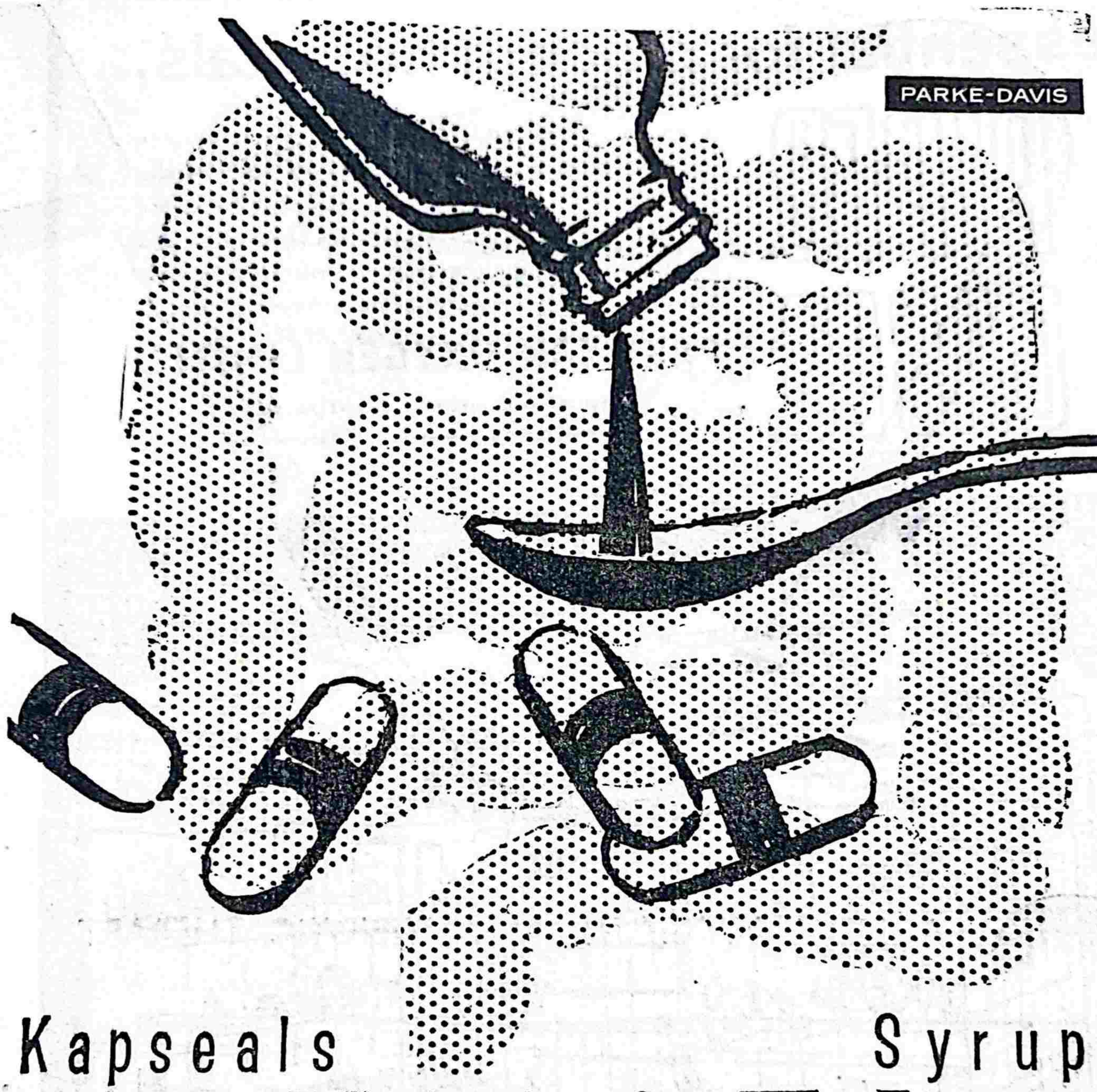

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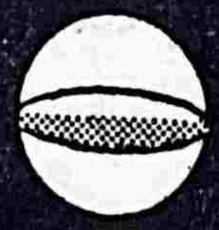

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A SURVEY OF HUMAN INTESTINAL PARASITES IN THE CONSERVANCY STAFF OF THE CITY CORPORATION OF LAHORE

BY

PROFESSOR DR. M. ATIQUR RAHMAN ANSARI, PH. D. (PB.)., SC. D. (PARIS), D. SC. (PB.), F. P. A. S., F.E.S.I., F.R.E.S., *Professor of Medical Entomology and Parasitology* ; *and*

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*Continued from the *Pakistan Journal of Health, Lahore;* 13(3) : 196—232 (1963)

SINGS AND SYMPTOMS

Our wills and fates do so contrary run
That our devices still are overthrown,
Our thoughts are ours, their ends none of our own.
(*Shakespeare: Hamlet*)

Human parasites represent a very small portion of much wider world of closely related parasites occurring in other animals. Formerly, the parasitologists claimed to determine the culprits involved with precision and accuracy. The discovery of closely allied forms and strains and races of same parasites has made the job rather complicated. Thus at times it becomes a problem to determine what clinical laboratory procedure will be of value in a particular survey to differentiate the race involved.

The parasites, residing in the human intestine, are now known to involve so many organs and tissues of the body, that the modern parasitologist cannot help thinking of the symptoms and syndromes involved.

Entamoeba histolytica, for instance, is both a parasite and a commensal. This parasite is notorious for a multi-symptom disease *viz.*, amoebic colities and dysentery, septic peritonitis, hepatitis, lung infection and skin ulcers. This state of affairs warrants the laboratory worker to be familiar with various classical syndromes evoked by the presence of different parasites.

From a perusal of the symptoms manifested by different parasites, we are forced to conclude that parasitologists should not only be familiar with parasites alone but should also be well versed in other different fields viz., dentistry, dermatology, gastro-enterology, haematology, neurology, ophthalmology, urology, etc., etc. The other alternative, rather sober and serene solution, is that there should be a team work between clinicians, pathologists and parasitologists.

Practically all the text books, both on clinical parasitology and medicine, give a long list of symptoms under each parasite and, we the teachers, probably without an exception, pass this information from year to year, to our pupils without comments. Although we adopt lying down position yet we always feel that there is a necessity to

assess the symptomatology and dispel the confusion from the mind of the practitioners, laboratory workers and parasitologists.

We agree that each animal, which gets an opportunity to enter our body, tries to live in a state of equilibrium. Accidentally some, in a zeal to exist, invade and live within tissues and begin to play a role of a parasite. Those which choose to live in the intestinal lumen, for instance, continue undergoing tremendous evolutionary processes. Side by side with a pathogenic race, an innocent race continues evolving. Experimental evidence exists, practically for all the animal parasites of man, to show that the host parasite relationship oscillates fairly regularly between virulence and avirulence. In some the phenomenon is well marked, while in others it does not modify to a perceptible level. A simple diagramatic representation of the affairs may be of the pattern given in the figure.

The avirulent form is not associated with any symptoms. For a practicising physician, a parasite may provoke symptoms at one time but it has also a latent phase of chronic infection and the carriers are all symptomless.

Signs and symptoms, as given in books, do not seem to have any definite diagnostic value. Real symptoms, if well defined for any one parasite, may probably be suggestive of a tentative possibility. Experience in clinical method of approach is by no means an accurate presumptive diagnosis but always precedes a laboratory work. Is this procedure fine enough or mutable, is the question and since long awaiting an answer. At present there appears to be an elaborate exaggeration and erroneous correlation of unrelated symptoms to simple parasitism. Standardlisation of symptoms and signs attributable to each parasite is now over due.

The list of symptoms and signs attributed to different parasites, commonly encountered in this country, are given below. A perusal of this list will show that almost all the symptoms are attributable to all the intestinal parasites. At the very sight, it appears to be an extensive stretch of imagination. If one worker speaks of headache and visional disturbances and acute conjunctivitis due to common round-worm infection, the other does not hesitate to add sexual disturbances, rhematoid arthritis, meningititis and hematuria due to it. We are afraid, that if this competition continues, some original thinker may claim one day that this parasite is very helpful in spacing pregnancies and inducing safe abortions. Gods of Family Planning will certinly owe a sense of gratitude to such a contributor. This fine addition to our knowledge will not only help them in Family Planning approach but will also provide an answer to taboos viz., social, ethical and religious. Parasitologist are thankful to their colleagues in the clinical field for the valuable guidance they rendered in an indirect identification of suspects, but not for silly brain waves. All efforts, therefore, should be made to refute what the poet C. Churchill once said : "*Most of those evils we poor mortals know, from doctors and imagination flow.*"

Professor M. Afzal Husain, H.Q.A., M.A. (Cantab), Hon. D.Sc. (Pb.), Hon. LL.D. (Toronto),F.N.I.,F.P.A.S., F.E.S.I. (formerly Chairman, Pakistan Public Service Commission, Karachi and Vice-Chancellor Panjab University, Lahore) once said to his post-gra-

duate classes that it took a decade or two to pull a discovery into a text-book but alm st a century to push a blunder out of it. *We know that good discoveries are monuments without a tomb.* One who dedicates his life to extend the boundaries of science must combine the qualities of a saint/scholar/soldier. It is always profitable to have advice and councel. As one of his desciples, we advocate that lies are preventable in science and must be prevented. Research is to remove confusion. We should do the right, when we would, for the would change, who knows what.

Most of these observations are probably based on the response of the host to the specific treatments. Does the elimination of a symptom indicate that it was positively due to a particular parasites? Serious attempts should always be made to correlate this basic sciences with clinical interpretations. Clinical symptomatology, pathology and parasitology must go together. Work in a directly opposite direction has not served as useful purpose in the past and is not going to serve any useful purpose in future either.

False association of symptoms to a parasite has done greater harm than good. *Parasite-phobea* is a worst disease caused by a knowledge of these symptoms. We know of cases, who just after knowing of the presence of an endo-parasite showed serious clinical manifestations and sensitization phenomenon but before such a knowledge the parasite was tolerated without even mild symptoms. We, therefore, must resolve to sift chaff from the grain and establish true relationship between parasites and their manifestations. The world-wide tendency to widen symptomatology to include syndrome remote from the seat of predilection of the parasite are due to various reasons. One is zeal on the part of the worker to give something new. This latter trend must be stopped. It reminds us of Shakespeare who said:

"I come to bury Caesar, not to praise him,
The evil that man do lives after them,
The good is oft interned with their bones".
(Julius Caesar : 3 (2)

Signs and symptoms associated with infection with intestinal parasites may be conveniently mentioned under various systems as follows:—

A : SYMPTOMS AND SIGNS REFERRABLE TO DISGESTIVE SYSTEM (INCLUDING GASTRO-INTESTINAL TRACT, LEVER, PANEREAS, GALL BLADDER)

1. *Anorexia* (*Loss of appetite*)—

(*i*) There may be marked loss of appetite in *balantidiasis.*

(*ii*) Appetite may also be killed in early stages of *amoebiasis.*

(*iii*) There may be poor appetite in *giardiasis* if accompanied with chronic diarrhoea.

(*iv*) Poor appetite is also one of the manifestation in *trichuriasis* of children.

(*v*) Children, who remain disturbed during night due to nefarious activities of seat-worm (*enterobiasis*) also have poor appetite.

(*vi*) Anorexia is one of the symptoms in early stages of hook-worm (*ancylostomiasis*) infection.

(*vii*) Loss of appetite may be the only symptom in children suffering from dog. tapes worm (*Dipylidium caninum*).

(*viii*) Round-worm infection, though notorious for gluttony, yet in sensitive cases it may also kill the appetite.

2. *Capricious appetite* (Hunger).—

(*i*) *Taenia saginata*—Infection make one glutton.

(*ii*) Some children, carrying a heavy load of *Ascaris* worms, remain hungry and have an unsatisfiable desire to eat. In endemic areas, the children are usually greedy.

3. *Vague Abdominal Discomfort*—(*i*) Large proportion of infected persons in *amoebiasis*, complain of abdominal discomfort.

(*ii*) About 30—40 per cent whip-worm infections have periodic abdominal distress. A vague burning sensation is reported by 15—20 per cent patients.

(*iii*) Vague abdominal discomfort and acute coliky pain in epigestric region is common symptom of *ascariasis*.

(*iv*) Heaviness and sensation of weight is usually felt in epigastric region in *Taenia saginata* infection.

(*v*) Slight intestinal disturbances may be complained of by children carrying dog-tape-worm. These symptoms are due to the metabolic wastes of the worm absorbed into the system.

4. *Nausea*—(*i*) Nausea may suggest *balantidiasts*, *ameobiasis* of caecum, *ancylostomiasis/necatoriasis* and beef-tape-worm infections.

(*ii*) Nausea is often a symptom in *D. feragilis* infection.

(*iii*) Nausea may also be one of the symptoms in *ascriasis*.

5. *Vomiting*—(*i*) Frequent vomiting may occure in *balantidiasis* and *amobiasis* of caceal area alone.

(*ii*) A patient harbouring *D. faragilis* occasionally suffers from this symptom.

(*iii*) Vomiting may occasionally occur in *ancylostomiasis/necatoriasis*.

(*iv*) *Post-prandial* vomiting has been recorded in beef-tape-worm infection.

(*v*) Vomiting may also precede, coincide or follow the reverse passage of rounds worms through the mouth opening. In more sensitive persons, this may, however, also occur independently of the passage of the worm.

6. *Abnormal Eructations* (belching)—(*i*) Eructations may occur in 25—30 per cent whip-worm infections.

(*ii*) The act of bringing up gas is also frequent in *ascariasis*.

7. *Flatulence*—(*i*) Distended abdomen and severe flatulence may occure in heavy hook-worm infection.

(*ii*) Abdominal distention with flatulence also occur in some cases of whip-worm infection.

(*iii*) Flatulence is also an evidence of duodenal disturbance in *giardiasis*.

8. *Indigestion*—(*i*) About 30—40 per cent cases of *trichiuriasis* suffer from indigestion.

(*ii*) Severe hook-worm infection also interferes with proper digestion of the ingested food.

(*iii*) Severe *ascariasis* may also upset digestion. Poor digestion in this case is mainly due to the inflammed bowels.

(*iv*) Indigestion may also be caused by dog-tape-worm infection in children.

9. *Geophagea*—Craving of ingesting clay is a characteristic feature in hook-worm infections. Further it has been observed to be of more severe nature than noticed in women during pregnancies.

10. *Heart burning*—Heart-burning is a queer sensation and has been encountered in some cases suffering from *ancylostomiasis/necatoriasis*.

11. *Pot-belly*—(*i*) The bulbous belly is a typical physical sign in children carrying *ancylostomiasis/necatoriasis*.

(*ii*) Heavy round-worm infection in children is well known to cause protuberant abdomen.

12. *Abdominal tenderness*—(*i*) There may be exquisite tenderness of the entire abdominal region if the peritonitis is involved in *amoebiasis*.

(*ii*) Tenderness of abdominal wall may be complained of in *D. fragilis* infection.

(*iii*) Tenderness of duodenum, jejunum and/ilium may occur in severe hook-worm infection. In some cases there may be pain in this region.

13. *Pain*—(*a*) *Epigastric region*—(*i*) Dull epigastric pain is common in severe *giardiasis*.

(*ii*) Marked epigastric pain is also experienced in children suffering from *trichiuriasis*.

(*iii*) Epigastric pain is accompanied with fullness of abdomen in hook-worm infections and is usually received when the patient eats bulky food or ingests clay.

(*iv*) Acute epigastric pain may occur in *ascariasis*. In some cases, however, the pain experienced is vague and dull.

(*v*) Hunger-pain frequently develops towards the end of incubation period in beef-tape-worm infection.

(*b*) *Lower abdominal pain*—(*i*) When caecal area is exclusively involved in *amoebiasis*, the patient usually complains of pain over Mac Burney's point.

(*ii*) Vague to moderate pain may be one of the complaints in *D. fragilis* infection due to superficial irritation. This amoeba has been found in the lumen and crypts of intestinal glands with ingested R. B. Cs.

(*iii*) More or less 35—40 per cent cases of *trichiuriasis* may complain of pain over MacBurney's point.

(*iv*) Abdominal pain is common feature of whip-worm infection and is more felt in the lower quadrant.

(*v*) Children suffering from *hymenolepiasis* also commonly complain of abdominal pain, which is said to be of nervous origin especially in case of multiple infection.

(*v*) Hook-worm infection is sometimes the root cause of the painful abdomen. It occurs when the duodenum, jejunum and ilium are involved in the infection. Due to disorder of the motor function in the mesenteric and submucosal nerve plexus the patient becomes irritant and short tempered.

(*vi*) Acute intermittent intestinal colic, sometimes mimicking appendicities, is initiated by the presence of *Ascaris* worms.

14. *Tenesmus*—(*i*) Tenesmus is frequently reported in *balantidiasis.*

(*ii*) It is usually absent in *amoebiasis* but in some cases, however, moderate tenesmus occurs.

(*iii*) Tenesmus may also occur in heavy *trichiuriasis.*

15. *Colic*—(*i*) The presence of many seat-worms in the rectum may cause violent rectal colic in some cases.

(*ii*) Abdominal colic, accompanied by severe diarrhoea and dysentery, are usually characteristic of chronic *balantidiasis.*

(*iii*) Beef-tape-worm infection may give rise to severe or dull intestinal colic.

16. *Intestinal obstruction*—(*i*) Tangled mass of writhing *Ascaris* worms are notorious for mechanical obstruction in the intestines and provoking *ileus* or causing acute intestinal obstruction.

The vagrant *Ascaris* worms also sometimes are responsible for blockage of ampula of Vater (hepato-pancreaticampula) and the common bile duct.

(*ii*) Beef-tape-worm infection has also at times been responsible for acute intestinal obstruction.

(*iii*) Chronic intestinal *amoebiasis* may cause stricture of the lumen of large intestine and lead to chronic intestinal obstruction.

(*iv*) Amoebic granuloma may sooner or later cause intestinal obstruction.

17. *Intestinal interssusception*—(*i*) Intestinal interssusception may sometimes be the result of intestinal *amoebiasis* in adults usually with the ileo-caecal part of the intestine, forming the apex of the tumour mass.

(*ii*) Heavy round-worm infection may, when the parasites form a mass in the intestine, induce a state of volvulus.

18. *Constipation*—(*i*) About 30—40 per cent of the chronic cases, suffering from whip-worm infection, usually complain of periodical constipation.

(*ii*) Severe constipation may also alternate with acute dysentery and diarrhoea in hook-worm infection.

19. *Diarrhoea*—(*i*) Almost every patient suffering from *balantidiasis* passes diarrhoeal stools.

(*ii*) Chronic diarrhoea, with gruelly and steatorrhoic stools containing large amount of mucous and undigested free fat, but without blood may occur in acute *giardiasis*, generally in children but rarely in adults.

(*iii*) Usually moderate and sometimes profuse diarrhoea is a prodromal episode in *amoebiasis*. There is generally an urgent desire to deaecate. The stool may occasionally be formed or semiformed but always suspended in copious amount of mucous. Sometimes the stool may contain mucous alone.

(*iv*) Frequency of stools with mucous may be experienced in *D. fragilis* infection.

(*v*) Diarrhoea, containing blood-streaked stools, may occur in a heavy infection of whip-worm. Such case may pass sufficient amount of mucous, if the infection is confined in the recto-sigmoidal region.

(*v*) Diarrhoea may also occur in severe *ascariasis*.

(*vi*) Diarrhoea usually occurs towards the end of incubation period in beef-ta pe-worm infection. In some cases, prolonged watery diarrhoea may even prove fatal.

(*vii*) Diarrhoea, of imperfectly digested food, occurs in *ancylostomiasis* where the victim eats ravenously due to insatiable appetite.

20. *Melaena*—Black, tarry stools may be only prominent symptoms in some cases of intestinal *amoebiasis*.

21. *Steatorrhoea*—According to Veghelyi (1941), *giardiasis* establishes a (partial or complete) barrier to fat absorption in the upper small bowels. When the infection is very heavy, the stool may have overtly characteristic appearance—bulky, acidic, pale, frothy and fatty. In case of the barrier being partial, the steatorrhic condition may be overlooked.

22. *Dysentery*—(*i*) Severe type of dysentery may occur in some cases of *balantidiasis*—Such cases are usually with ulceration of the intestines.

(*ii*) In amoebic dysentery, considerable amount of blood and mucous is evacuated with formed or semi-formed stools. There is urgent desire, after every short interval, to defaecate and after evacuation there is slight relief.

(*iii*) The presence of *G. intestinalis* in dysenteric stools is more than mere coincidence. The presence of these flagellates in the intestines produces lot of mucous at the site of infection. The incidence of flagellate dysentery is however, high in children and of low frequency in adults.

(*iv*) In some cases of *ancylostomiasis*, usually in terminal stages, the patient may pass frequent dysenteric motions, which may be difficult to distinguish from other dysenteries.

23. *Prolapsus recti*—(*i*) *Prolapsus recti* is characteristic of heavy chronic whip-worm infection of the rectum in children.

(*ii*) The condition is also common in long standing cases of *enterobiasis*.

24. *Mega colon*—Colon may get enormously distended in chronic cases of *amoebiasis.*

25. *Duodenitis*—Chronic and haemorrhagic duodenitis is most common manifestation in severe *giardiasis.*

26. *Diverticulitis*—(*i*) Inflammation of diverticulum usually occurs in severe *ascariasis.*

(*ii*) Diverticulitis may also occur in intestinal *amoebiasis.*

27. *Gastric and duodenal trauma*—Wandering *Ascaris* worms are sometimes responsible for gastro-duodenal trauma in endemic areas. This injury in some cases leads to serious consequences.

28. *Appendicitis*—(*i*) Acute and sub-acute appendicitis may arise as a complication of *amoebiasis* of caecal area.

(*ii*) *D. fragilis* may also induce appendicitis.

(*iii*) Heavy infection of appendicular mucosa with *Trichocephalus trichiurus* may allow pathogenic organisms to enter the tissue and set in an acute or sub-acute inflammatory process.

(*iv*) Brisk movements of female seat-worms may cause appendicitis.

(*v*) At times, the individual segment (proglottids) of beef-tape-worm become lodged in appendicular lumen and cause acute inflammation in the appendix.

(*vi*) Vagarant round-worms may pass into an intestinal diverticulum and initiate *diverticulitis.*

(*vii*) Appendicitis may, in rare occasions, be caused by a female round-worm getting into the lumen of appendix and laying eggs therein. In such cases, the appendicular lumen has generally been found to be packed with *Ascaris* ova.

(*viii*) Vagarant seat-worms may get into the lumen of appendix and cause appendicitis.

29. *Amoebic typhlitis*—In intestinal amoebiasis, caecum may be the only portion involved, giving rise to symptoms which may be confused with appendicitis.

30. *Tumours, ulcers and abscesses*—(*i*) Chronic *amoebiasis* may cause the formation of a granulomatous mass (amoeboma) along the length of the large intestine. This tumour is most frequently found in caecum, transverse colon and sigmoid colon and may be mistaken for a carcinomatous growth.

Ulceration of the skin, generally in the peri-anal regions, may result due to invasion with *E. histolytica* which, in extreme cases, may lead to extensive necrosis and gangrena. Colonic appendicular and hepatic drainage in surgical conditions of *amoebiasis* may also be a cause of dermal *amoebiasis* leading to ulceration, necrosis and gangrene.

(*ii*) Ulceration of stomach, small intestine and colon may also be caused by *I. butschlii.*

(*iii*) Rarely a nasal polypus may be caused by metastatic invasion by *E. histolytica.*

(*iv*) *Tangled masses of Ascaris* worms are sometimes suggestive of abdominal tumours, gastric and duodenal ulcers.

Volvulus of the intestines may be caused by heavy *Ascaris* infection. In such cases, round-worm infection has to be seriously considered in differential diagnosis.

(*v*) Syndrome of peptic ulcer may also be met with in some case of beef-tape-worm infection.

31. *Perforation*—(*i*) Perforation of the descending and ascending colon may result when the patient's resistence is very low and the motile *E. histolylica* (trophozoite) is highly virulent. When such perforation is slow and insidious, it may lead to pericolic or paracolic abscess (intra-peritoneal or retro-peritoneal). In fulminating cases the intra-peritoneal perforation may occur with dramatic suddenness and cause generalised peritonitis. The retro-peritoneal abscess may ultimately make its way down through the psoas muscle and appear in the inner aspect of the upper part of the corresponding thigh. In some rare cases however, the supprative invasion of the ischiorectal mass of tissues may occur and form *Ischiorectal abscess*.

(*ii*) *Ascaris* worms may perforate the bowels, most commonly in the region of ileo-caecal valve and burrow into the abdominal wall, giving rise to the formation of an abscess—an unusual attempt on the part of human organism to get rid of the parasite.

Sometimes the tiny, pearly, nodules in the mesentery, formed by the encysted ova of round worm, may perplex the surgeon as to their exact nature.

Wandering worms may also perforate oesophagus and vomited out or perforate the diaphragm and enter the pleural cavity causing pleurisy and empyema.

32. *Peritonitis and Peritoneal abscess*—(*i*) *Amoebiasis* may be accompanied by the perforation of bowels and peritonitis. This condition may occur when the colonies of the parasite become established in submucosa and continuously erode mesenteric venules.

(*ii*) Whip-worm may reflexly cause appendicitis and peritonitis (Savil).

(*iii*) The thread-worms may directly be responsible for catarrhal *appendicitis*.

(*iv*) Penetration of the round-worms into peritonial cavity leads to *peritonitis* either of generalised order or a localised one resulting in the formation of an abscess, which may find its way to the exterior helping the worms to escape.

33. *Enlargement and tenderness of Liver with or without suppuration*—(*i*) Amoebic-colitis may sometimes be associated with the enlargement and tenderness of liver (hepatic amoebiasis) without fever, and without subphernic bulging or diaphragmatic impairment.

Single or sometimes multiple abscesses in the liver are commonly known to occur with or without manifest involvement of intestines with amoebic infection.

Enlarged, tender liver may occur in accute amoebic hepatitis, if one or more foci, are established. Fever with chill (mimicking malaria) together with moderate neutrophilic leukocytosis, are also some of the signs and symptoms of acute amoebic hepatitis.

(*ii*) Entery of vagarant *Ascaris* worms into the parenchyma of the liver may cause necrosis of liver cells and liver abscess.

(*iii*) Segments of *T. saginata* have been recovered from liver abscess.

34. *Jaundice*—(*i*) Obstructive jaundice may result from pressure exerted on biliary

passages by wide and extensive amoobic abscesses of the liver.

(*ii*) Round-worm may enter bile duct and lead to obstructive jaundice.

35. *Cholecystitis*—(*i*) Either the extension of necrotic process from liver abscess or the infection ascending from the gut through the biliary tract may involve gall bladder and cause *amoebic cholecystitis*. It is a rare manifestation and has been recorded chiefly by French workers.

(*ii*) Inflammation of gall bladder has been usually traced to *giardiasis*. Whenever the system is involved in the infection, this acute and failrly long ailment may occur in 20 per cent cases.

(*iii*) *Ascaris* worms may also be cause of this condition by obstructing common bile duct .

(*iv*) Syndrome of gall-bladder disease may be met with in beef-tape-worm infection.

36. *Haemorrhagic pancreatitis*—The wandering *Ascaris* worms may initiate hemorrhagic pancreatitis by blocking the pancreatic duct due to occlusion of ampula of Vater by inflammatory reaction induced by the infection.

37. *Hernia*—Cases of inquinal or umbilical hernia due to heavy infection with *Ascaris* worms have also been reported.

B. SIGNS AND SYMTOMS SUGGESTIVE OF CARDIO-VASCULAR DISORDERS.

1. *Vasomotor disturbances*—It is of occasional occurrence in hook-worm infection. It may be accompanied with lassitude.

2. *Blood Pressure*—Almost in all hook-worm infections, the blood pressure is of the following pattern:—

(*i*) Diastolic blood pressure may remain normal.

(*ii*) Systolic blood pressure may be considerably diminished.

3. *Weak pulse*—Large percentages of persons carrying severe hook-worm infection usually have weak pulse.

4. *Palpitation of Heart*—Palpitation of heart and haemic murmurs in chronic hook worm infection is one of the common findings.

5. *Cardiac Hypertrophy*—Tremendous cardiac hypertrophy has been found a constant sequal in prolonged hook-worm anaemia.

6. *Cardiac Dilatation*—Cardiac dilatation is one of the complications encountered in severe cases of whip-worm infection.

7. *Amoebic pericarditis*—Chronic amoebic abscess of liver, especially of the left lobe, may involve the pericardium through the diaphragm and cause purulent pericarditis, which, if the patient survives at all, may result in constrictive pericarditis. The infection may also be carried through blood.

8. *Syncope*—Attacks of syncope are liable to occur in severe hook-worm infections of fairly long duration.

9. *Cardiac murmur*—Haemic murmur is a common auscultatory finding in advanced cases of hook-worm infections.

10. *Cardiac Penetration*—The wandering *Ascaris* worms may some times penetrate the heart often with fatal consequnces.

11. *Haemorrhage*—(*i*) Bleeding is one of the complications encountred in some cases of amoebic colitis.

(*ii*) The ulceration caused by ciliates, sometimes becomes haemorrhagic.

(*iii*) Removal of whip-worm with anthelminthics usually results in petechial haemorrhages at the site of attachement. It mimicks hook-worm infection and one of the common causes of confusion in clinical practice.

(*iv*) Petechial hamorrhages is also produced by the filiform larvae of *S. sterocoralis* during skin invasion.

(*v*) The migrating larvae of the giant-round-worm, when escape from the blood vessels into the lung alveoli do not only cause trauma but also petechial haemorrhages.

12. *Blood Picture*— (*a*) (*i*) *RBCs* may decrease in hepatic and intestinal *amoebiasis, balantidiasis, strongyloidiasis, trichiuriasis, ascariasis and* (definitely) *encylostomiasis.* The protozoal parasites may cause fall in R.B.C. count because of the haemorrhage caused by their activity. The helminthic parasites, however, may lower the count either by sucking blood of their host or through the action of their toxins or both.

(*ii*) *Hoemoglobin percentages* may fall in hepatic and intestinal *amoebiasis, balantidiasis, strongyloidiasis, trichiuriasis, ascariasis* and (definitely) *ancylostomiasis.*

(*b*) *Leukocytosis*—(*i*) It is one of the syndromes of hepatic amoebiasis.

(*ii*) During early stages of active infection, polymorphonuclear-leukocytosis (upto 25000) with eosinophilia (as high as 40 p. c.[m. m.) is characteristic in *strongyloidiasis.* In chronic infections, leuckocytosis changes to neutropenia with relative monocytosis and moderate eosinophilia. It persists for years.

(*iii*) Typical leukocytosis (upto 17,000 per cu mm. of blood) with predominant eosinophilia occurs in hook-worm infection. Later leukocytosis count drops to normal or below but eosinophilia may remain moderately high.

(*iv*) Leukocytosis (with 6-34 per cent eosinophilia) is characteristic in *Taenia sagigata* infection. Later in the course of the disease there may be leucopenia.

(*v*) *Large mononuclears* increase in *trichiuriasis, strongyloidiasis* and *ancylostomiasis.*

(*c*) *Leukopenia*—Slight and perceptable leukopenia may occur in some cases having severe infection of beef-tape-worm.

13. *Anaemias*—(*i*) Prolonged watery diarrhoea in *balantidiasis* may cause anaemic skin ,

(*ii*) Yellow, palor skin usually occurs in *ancylostomiasis/necatoriasis.*

(*iii*) In *trichiuriasis*, there may be systemic intoxication and frequently secondary anaemia.

(*iv*) There is mechanical loss of blood at the site of attachement of hook-worms, together with copious amount of seapage from abandoned holds. Anaemia usually begins to appear 10—20 weeks after exposure to infection. In mild infection, however the anaemia is negligible and therefore, not appreciated.

14. *Oedema*—(*i*) Oedema of the face and hands commonly occurs in whip-worm infections.

(*ii*) Congestion and oedema is a usual feature at each site of infection by the filiform larvae of *S. stercoralis.* Inflammation however, does not occur if the infection is not accompanied by pyogenic organisms.

(*iii*) Severe hook-worm infection also causes oedema of the face or legs and in some cases of the whole body. It is one of the characteristics of this disease. When oedema is confined to face or legs alone, the other parts of the body however, show signs of emaciation.

(*iv*) *Palpebral oedema* generally occurs when the vagrant larvae of *Ascaris* pass through the tissues of eyelids.

C. Symptoms and Signs relating to Respiratory system

1. *Halitosis*—Considerable number of childern carrying *Ascaris* worms have bad and offensive breadth.

2. *Cough and Coryza*—(*i*) Some cases with hook-worm infection, one or two weeks after the skin infection, complain of cough with coryza and sore-throat. These symptoms may last for 2-3 weeks.

(*ii*) *Ascaris* infection may also cause symptoms of bronchitis, when the larvae are passing through bronchial tubes.

(*iii*) Cough is a common symptom in children infected with intestinal worms.

3. *Bronchial asthma*—The metabolites of the mature *Ascaris* worms are notorious for initiating one or the other serious sensitization phenomenon in their victims. Bronchial asthma has been reported to have occurred only in sensitive patients.

4. *Empyaema*—(*i*) Injury to pleural cavity in *ascariasis* is frequently associated with empyema.

(*ii*) *Empyaema* may also be due to extension of amoebic liver abscess into pleural cavity.

The pus formed and collected in the cavity is not only irritating but a serious sign.

5. *Pneumonitis*—The migrating filiform larvae of *Strongyloides*, *Ascaris* and hook-worm produce petichial haemorrhages at site, where pulmonary blood vessles are broken into capillaries in the inter-alveolar septa. In severe infections respiration is usually embarrassed considerably because small pools of blood accumulate in the alveoli and smaller branchioles. There is general oedema of the lungs also.

This condition is comparatively well marked in *strongyloidiosis* and *ascariasis* than in hook-worm infections. In children, *Ascaris* pneumonitis may occur usually 1—5 days after exposure. Pneumonitis of *Strongyloides* origin is said to be the basis for activating quiescent pulmonary tuberculosis.

6. *Dyspnoea*—(*i*) According to Getz (1945), dyspnoea is one of the symptoms in *trichiuriasis*.

(*ii*) *Dyspnoea* may also occur in severe hook-worm infection as also when the larvae of *Ascaris* worms are present in the alveolar spaces.

Dyspnoea may also be accompanied by peritonitis due to *ascariasis*, *amoebiasis*, *trichiuriasis*, etc.

Some degree of oedema of glottis and larynx may occur when round-worms are vomitted out and this leads to dyspnoea. Larvae of round-worms during their passage may also initiate dyspnoea.

(*iii*) *Empyaema* caused by perforating *Ascaris* worms, as well as amoebic abscess may cause severe dyspnoea.

(*iv*) Marked dyspnoea may result from invasion of right lung by amoebic abscess.

7. *Haemoptesis*—(*i*) During migration, the *Ascaris* larvae may initiate mild haemoptesis, generally with several larval worms in the sputum.

(*ii*) Blood-tinged, rusty expectoration may also be encountered in cases infected with *Strongyloides*.

(*iii*) Feeble haemoptesis has been observed in *ancylostomiasis*.

8. *Suffocation*—(*i*) There are cases on record, where the vomited *Ascaris* worms are passed into larynx and produced suffocation. Obstruction of larynx may initiate serious crisis or even cause death in children.

(*ii*) The migrating larvae of hook-worm or *Ascaris* may cause odaema of glottis and induce symptoms of suffocation.

9. *Pulmonary abscess and gangrene*—(*i*) *Amoebiais* may also cause directly or indirectly abscess of lung and in rare cases gangrene.

(*ii*) A solitary case of lung abscess is also on record due to *I. butschlii*.

(*iii*) The wandering *Ascaris* worms, some times penetrate the lung of the patient, and produce pulmonary abscess or gangrene.

(*iv*) Segments of beef-tape-worms have been, in rare instances, encountered in lung abscess.

10. *Broncho pneumonia*—(*i*) *E. histolytica* may be carried through the circulation to the lungs and may give rise to a condition resembling broncho-pneumonic consolidation especially on the right side.

(*ii*) Young *Strongyloides*, when passing through the lung, produce symptoms simulating broncho-pneumonia.

(*iii*) Chronic bronchitis may also be due to heavy whip-worm infection.

(*iv*) In the course of their journey through the lung alveoli, the larvae of *Ascaris* worms may, especially when pathogenic organisms are also present, cause broncho pneumonic patches of consolidation and in extreme cases of complete lobes thus givingrise to serious symptoms characteristic of the lesion.

11. *Pain in chest*—(*i*) Intense pain in lower right region of chest occurs due to inflammation of diaphragmatic pleura in *amoebiasis*. There is also persistent unproductive cough associated with this condition.

(*ii*) Pain of chest may also be referred to peritonitis due to *ascariasis, trichiuriasis.*

12. *Intra-thoracic burning*—In *strongyloidiasis*, generalised intra-thoracic burning is experienced between 3rd-4th day of infection.

D.—Dermatological Reactions

1. *Macular erythematous spots*—(*i*) Minute macular erythematous spots are characteristic of *strongyloidiasis*. These are found ar each point, a filiform larva of the worm penetrates the skin of the victim.

(*ii*) Similar spots may appear at the point of penetration of hook-worm larvae on the skin.

2. *Pruritis*—(*i*) Violent pruritis occurs in 15—20 per cent whip-worm infections.

(*ii*) Intense pruritis, usually accompanied by petecheal haemorrhages, i s produced by the invading filiform larvae of *S. stercoralis*.

(*iii*) Almost intolerable *pruritis ani* is set up in *enterobiasis*. It is usually more severe after sunset and during night in most children.

(*iv*) Intense itching is usually experienced when hook-worm larvae penetrate the skin.

(*v*) Troublesome itching at the anus may be experienced when the round-worms are making their way out.

3. *Dry-ness of skin*—(*i*) It occurs in whip-worm infection.

(*ii*) Skin may become dry and harsh in hook-worm infection. In advanced cases of the trouble the hair also be come dry and life-less.

4. *Pallor of the skin*—There is always increased demand for a greater circulating blood volume in the vital areas in hook-worm infection. This urgent demand is met at the cost of the peripheral blood-flow which is considerably reduced rendering the skin markedly pale.

5. *Perspiration*—Severe hook-worm infection usually reduces rate of perspiration.

6. *Creeping eruption*—Fillform larvae of various hook-worms cause cutaneous lesions on unprotected parts of the body.

7. *Ground itch*—Ground itch, incessant itching followed by burning, oedema and erythema of the site usually occurs when III stage filiform larvae of hook-worm bore the skin. These sites of entry, may ultimately develop into papular eruptions and ending in painful vesicles. The usual sites are between toes, lateral and dorsal surfaces of feet, but may also develop at other places.

8. *Allergy*—The *Ascaris* and other worms are known to secrete/excrete several toxins in their body.

The round worms are known to produce *anaphylaxins, neurotoxins hemolysins* and *endocrinotoxins*, etc. It has been observed that laboratory workers, who handle material contaminated with the tissue of these animals, often develop serious allergic manifestations. These effects may also be due to emanations. The following allergic manifestations have been observed :—

(*a*) *Status asthmaticus*—This catastrophe may manifest itself as a complecation in cases who develop asthma due to *ascariasis.*

(*b*) *Urticaria*—(*i*) Intense periodic or regular urticaria has been found occurring in some cases having whip-worm infection.

(*ii*) Individuals, carrying *S. steroralis* infection previously, if subjected to another exposure to this worm, frequently develop incessant urticaria together with other sensitization reactions.

Men and women, who have genito-urinary *strongtyloidisis*, usually have severe urticaria of the skin around the external orifice.

(*iii*) *Ascaris* worms, after attaining maturity in the bowels, are reported to add metabolites and cause severe and pronounced urticarial rash and other sensitisation phenomenon in their host.

(*c*) *Dermatitis*—(*i*) Migrating females of seat-worm are notorious for causing *pruritus ani.* Severe scraching of the irritated region may some times result in dermatitis of the perianal and perineal regions. In some books this condition has been described as one of eczema which appears to be an exaggeration of the exact nature of the lesion.

(*ii*) Localised dermatitis is generally the rule with the penetrating larvae of hook-worm.

E. Symptoms and Signs Involving Genito-Urinary System.

1. *Haematuria*—In the presence of *Ascaris* adults in the bowl and also during the bilogical incubation period, signs and symptoms of haemuturia may often occur.

2. *Albuminuria*—In advanced and chronic stages of hook-worm infection, the patient may show symptoms of albuminuria.

3. *Renal Colic*—(*i*) If the *Ascaris* larvae reach the general circulation and filter out into the kidneys, they may cause most dramatic attack of renal colic.

(*ii*) Hook-worm larvae may also initiate an attack of renal colic.

4. *Nephrosis*—Serious nephrosis may occur in advanced stages of *ancylostomiasis/necatoriasis.*

5. *Nocturia*—(*i*) Genito-urinary *strongyloidiosis* does not only cause diurnal urgency to urinate but also urinary incontinence.

(*ii*) Severe *enterobiasis* in children, may be a common cause of "wet-beds". Involuntary discharge of urine (enuresis) has some times very bad effect on the per-

sonality of children. The badly infected children have a feeling of inferiority complex, become timid and are guilty conscious.

(iii) Whip-worm infection in children and some times in adolescents may also, cause "wet-beds".

6. *Mucoid vaginal discharge*—*(i)* Young girls, having heavy infection of *Enterobius vermicularis*, may often pass mucoid vaginal discharge.

(ii) Vagrant *Ascaris* worms may some time get into the female urinary—genitalia and cause serious disturbances.

(iii) Infection with *Trichomonas vaginalis* is frequently the cause of leucorrhoea with creamy white, viscid frothy discharge.

7. *Salpingitis*—*(i)* Invasion of the fallopian tubes with *E. histolytica* may be one of the causes of salpingitis, which in severe cases may proceed to ulceration, necrosis and gangrene of the tissues invaded.

(ii) Round-wrom may insinuate its way into the fallopian tubules and initiate salpingitis. The worm has been found sometimes encysted in the fallopian tubes.

(iii) The vagrant gravid females of seat-worms sometimes migrate up the vagina, uterus and fallopian tubules, where they either become encysted or penetrate into the peritoneal cavity and encyst on the peritoneum. While in tubules, the worms may some times produce symptoms of salpingitis.

8. *Vaginal and cervical amoebiasis*—Cases are on record where ulceration of vagina and uterine cervix was found to be the result of invasion with *E. histolytica*. It may be a pre-disposing factor in abortions and miscarriages.

9. *Penile amoebiasis*—Amoebic lesions of the penis have been observed in those husbands whose wives had amoebic infection of vagina and cervix.

Penile *amoebiasis* has almost exclusively been noticed amongst people who had not been cercumcised. Such patients exhibit lesions, characteristic of phagedena.

10. *Puberty*—Hook-worm infection retards, retracts or retrenches endocrine secretions. The diminished and damaged secretions delay puberty level in the community.

11. *Nosurnal emissions and Spermatorrhoea*—Long continued irritation due to *Enterobius vermicularis* and *trichiuriasis* may be a predisposing factor in the causation of abnormal night discharges in adults. Severe enterobiasis, of long duration may also similary induce spermatorrhoea.

12. *Sexual perversions*—Tickling sensation caused in some individuals by the activity of seat-worms and whip-worms may arouse sexual feeling and ultimately lead to indulgence in *masturbation* in both sexes. History of *sodomy cases*, in case of passive agents, has in some cases, been traced to infection with seat-worms and whip-worms.

Uncontrolable sex urge may arise in some case of acute *enterobiasis* and *trichiuriasis* and be responsible for serious sex crimes.

13. *Impotency*—*(i)* Damage to sexual urge and power has been attribtuted to severe hook-worm infection in some communities

(*ii*) The vagrant *Ascaris* worms sometimes penetrate the male genito-urinary tract and cause severe damage.

14. *Abortions and Miscarriages*—(*i*) *Amoebiasis* of vagina and uterine cervix may play an important role in abortions and miscarriages.

(*ii*) Hook-worm infections may in severe cases cause abortions and miscarriages.

F. Signs and Symptoms relating to Special Sense Organs.

1. *Retinitis*—(*i*) *Ascaris* larvae, if per chance get into general circulation and pass through eyes, may cause retinitis.

(*ii*) Haemorrhagic retinitis may also be caused by hook-worm infection.

2. *Dark shades below eyes*—Tired eyes in children usually indicate several sleepless nights due to noxious *enterobiasis*. Highly disturbed children ultimately develop dark shades under their eyes.

3. *Acute conjunctivicis*—Presence of giant round-worms in the bowel may cause acute *conjunctivitis*.

4. *Photophobia* (morbid dread or fear of light)—

Presence of the giant round-worms in the bowels, intereferes with the adjustment mechanism of the eyes to light.

5. *Dimness of vision*—Well marked infection of hook-worms may give rise to dimness of eyes.

(*i*) *Strabismus*—State of having cross-eyes is common in beef-tape-worm infection

(*ii*) *Optical illusions* is of common occurrence in beef tape-worm infection.

(*iii*) *Diplopia*—Beef tape-worm infection may be responsible for double immage of the same object.

(*iv*) *Pupillary* inequality may also occur in beef-tape-worm infection.

6. *Otitis media*—If, by accident, the *Ascaris* worms pass into the eustachian tubes, they provoke *otitis media*.

G. Signs and Symptoms Suggestive of lesions of Central Nervous System and Psychotic conditions.

1. *Headache*—(*i*) Patients suffering from *balantidiasis* may have severe headache.

(*ii*) Severe *hymenolepiasis* is also cause of severe headache in young girl patients.

(*iii*) Chroni *trichiuriasis*, *strongyloidiasis* and hook-worm infections also cause dull headache.

2. *Lassitude and general malaise*—(*i*) For several weeks, in early stages of *amoebiasis*, the patient has a vague feeling of uneasiness.

(*ii*) Round-worm infections, hook-worm infections and *trichiuriasis* are also cause of uneasiness.

(*iii*) In hook-worm infection, lassitude is almost always complained of during advanced stages of the disease.

3. *Depression*—There is usually profound depression in chronic *strongyloidiasis*, round worm infections and severe hook-worm disease.

4. *Neurasthemia*—Chronic intractable intestinal *amoebiasis* may establish a state of neurasthenia in some patients.

5. *Restlessness*—(*i*) Twitching and restlessness occurs in *ascariasis*. It is said to be due to the absorption of metabolites and foreign proteins from the worm by sensitive persons.

(*ii*) In chronic *strongyloidiasis*, a patient may become restless if auto-infection occurs uninterruptedly.

(*iii*) Restlessness is a common feature in *enterobiasis*. 99·9 per cent experience it and 0·1 per cent who do not, are liars because of a feeling of shame and inferiority complex.

6. *Insomnia*—(*i*) Severe *balantidiasis* may predispose victim to insomnia.

(*ii*) Severe and constant pruritis due to *enterobiasis* may also cause insomnia in children.

(*iii*) Abnormal wakefulness also occurs in *trichiuriasis*.

(*iv*) A patient suffering from chronic *strongyloidiasis* may also complain of sleeplessness.

(*v*) Foreign proteins derived from *Ascaris* worms, when absorbed in the system, also cause insomnia in senstive persons.

(*vi*) Severe *hymenolepiasis* also is one of the causes of sleeplessness in children.

7. *Convulsions*—(*i*) Very sensitive children having severe *enterobiasis* may have attacks of convulsions.

(*ii*) Heavy *hymenolepiasis* may also be responsible of convulsions in children.

(*iii*) Heavy *ascariasis* in children is some—times cause of severe attacks of convulsions and death.

8. *Neurotoxic symptoms*—(*a*) *Melancholia* may be induced in repeated and heavy hook-worm infections.

(*b*) *Apathy* is somtetimes attributed to severe hook-worm infection.

(*ii*) Mental apathy may also occur in *amoebiasis*.

(*c*) *Sympathetic neurosis*—(*i*) Getz (1945) has attributed sympathetic neurosis to whip-worm infection.

(*ii*) In occasional infantile *trichiuriasis* there is marked nervousness.

(*iii*) Reflexes are usually exaggerated in whip-worm infections.

(*d*) *Mental dullness*—(*i*) Children having severe hook-worm infection are comparatively dull than those who are not carrying the worms. Intelligence quotient was found to vary remarkably with the intensity of infection.

(*ii*) Nervous manifestations in children due to heavy *hymenolepiasis* are also well marked.

(*e*) *Meninges and choroid plexus*—Filiform larvae of thread-worms (*Strongyloides stercoralis*), when fail to penetrate through the pulmonary capillaries to reach alveoli, may migrate into pulmonary venules and gain access to the general circulation and may reach anywhere in the body. Their presence in cerebral blood vessels, and, perivascularly, in pools of blood, in the cerebellar mininges, and choroid plexus has been demonstrated by Yamaguchi (1925) and Faust (1935).

(*f*) *Angio-neurotic oedema* has been frequently reported in late stages of *Ascaris* infection. It is attributed to strong sensitization to the worm.

(*g*) *Meningismus* may be caused by the absorption of toxic secretions/excretions (*ascarions*) in case of round-worm infections. Symptoms simulating encephalitis may or may not coexist in such cases.

(*h*) The seat-worms erode caecal mucosa, expose sympathetic nerve endings, and ultimately cause nervous reactions in children.

9. *Neuropathy*—A variable types of neuropathies may occur in beef-tape-worm infection.

(*i*) *Chorea*—Characteristic twitching tremors and odd purposeless movements, simulating chorea, may occur in certain cases of beef-tape-worm infections.

10. *Epilepsy*—(*i*) Occasional epilaptic fits may occur in children carrying a heavy load of whip-worm infection.

(*ii*) Epileptic fits may also be due to *Ascaris* larvae, when accidently got into general circulation and migrate through the capillaries of the brain.

(*iii*) Heavy laod of *Hymenolepis nana* is also frequent cause of epileptiform—fits in delicate children.

(*iv*) Epileptic fits may also occur in beef-tape-worm infections.

11. *Brain abscess*—Single or multiple brain abcesses may occur in *amoebiasis* and very rarely in case of infection with *Iodamoeba butschilii*.

It is commonly haematogenous in origin but may be concomitant with amoebic abscess of liver or lungs.

12. *Idiocy*—(*i*) Children suffering from chronic *ascariasis* have been shown to be mentally less alert. After removal of worms, the scholastic ability usually improves considerably.

(*ii*) The proportion of idiots is more in hook-worm stricken communities than in those where the disease is not endemic.

13. *Night mares*—(*i*) Heavy infection with seat-worms may initiate nervous disorder resulting in the manifestation of night-mares in children.

(*ii*) Children having round-worms have also severe night-mares.

14. *Tinnitis*—Tinnitis is complained of in severe *ancylostomiasis*.

15. *Vertigo* (*Dizziness*)—(*i*) 20—30 per cent of the children carrying a heavy load of whip-worm infection suffer form vertigo.

(*ii*) Heavy infection of *Hymenolepis nana* in children induces a sensation of whirling or unsteadiness. The dizziness is frequently attended by nausea, vomiting and weakness.

(*iii*) Vertigo is frequently complained of in advanced cases of hook-worm infection.

16. *Meningitis*—Meningitis is one of the common manifestations in *ascariasis*.

17. *Invasion of brain*—Some workers have encountered round-worm astrayed into brain substance.

18. *Neurotoxic Symptoms*—(*i*) At least 25—30 per cent patients suffering from whip-worm infection show one or the other symptoms which may be referrable to neurotoxic manifestations.

(*ii*) Absorption of metabolic waste in dog-tape-worm infection generally causes toxic nervous manifestations in children.

19. *Systemic intoxication*—(*i*) The absorbed products of *Taenia saginata* create systemic intoxication in the patients.

(*ii*) Waste metabolites in dog-tape-worm infection may also evoke systemic intoxication.

(*iii*) Systemic intoxication has also been encountered in patients carrying dwarf-tape-worms (*Hymenolepis nana*).

H. Biochemical Changes in Body Fluids.

1. *Hypercholesterolaemia*—Some workers have noticed hypercholesterolaemia in advanced hook-worm infections.

2. *Hypoproteinaemia*—Hypoproteinaemia is also one of the serious signs in advanced cases of *ancylostomiasis/necatoriasis*.

3. *Pre-eclamptic/toxaemia and eclampsia*—Advanced cases of hook-worm infection exert deleterious effect on the kidneys, which may ultimately lead to toxaemias of pregnancy such as pre-eclamptic toxaemia and eclampsia.

I. Behaviouristic Symptoms

(*a*) *Nail-biting* occurs commonly in *enterobiasis* and latter may develops into a habit.

(*b*) *Nose-picking* habit commonly occurs in *enterobiasis*.

(*c*) *Grinding of teeth* at night occurs in *enterobiasis*, *ascariasis* and hook-worm infections.

(*d*) *Inattention*; loss of concentration is a common feature of severe infection of *enterobiasis* in children.

(*e*) *Masturbation* : considerable proportion of children get into this habit as a result of noticeable scratching of the anal region in *enterobiasis*.

J. Granulomatous Reactions

(*i*) *Ascaris* larvae, which are normally not used to invade human body and are ingested accidently, in their zeal to adapt to new environments, may wander about

into unnatural foci and usually get trapped there and elicit chronic inflammatory reactions, frequently providing granulomatous masses and simulating neopsaltic growths. Some times a clinician is tempted to diagnose a mass of round-worms as a new growth.

(*ii*) Similarly in chronic *amoebiasis*, a granulomatous tumour appears in the rectum which unless subjected to biopsy is confused with cancer of the rectum.

(*iii*) *Nasal polypi*—Invasion of nasal mucosa with *E. histolytica* may provoke chronic inflammatory granulomatous hyperplastic reaction, leading to formation of nasal polypi.

K. Febrile Reactions

(*i*) *Amoebiasis* does not only give local pain, if viscera are involved, but also accompanied with fever. Low grade fever however, usually occurs with hepatic *amoebiasis*.

(*ii*) Low grade fever may be found in *D. fragillis* infection.

(*iii*) Moderate febrile reaction (99—101°F) may be found in whip-worm infection.

(*iv*) Low grade, intermittent or subcontinued type of fever usually occurs in hook-worm infections.

(*v*) There is always elevation of temperature to 102·9—104°F (39·4—40°C) with rapid, shallow, irregular respiration of asthmatic type, accompanied with frequent spasms of coughing, bronchiolar rales and physical signs of lobular involvement, usually lasting for 1-2 weeks in *ascariasis*.

The presence and activity of *ascariasis* in young children may be characteristically associated with low grade fever.

L. Sub-normal Temperature

The patient suffering from hook-worm infections, may have sub-normal temperature and their body may feel cold even in hot weather.

M—Spontaneous Expulsion

(*i*) The *Ascaris* adults may some times pass out spontaneously per anus in some patients. It may occur either due to slight dietary indiscretions or due to disturbed digestion. The wandering worms may also be vomited. At times they also manage to pass through the nares and cause alarming sequelae in children.

(*ii*) Patients carrying *T. saginata*, from time to time, sometimes frequently and even daily, pass gravid segments in their stools but may also pass out spontaneously while the patient is sitting or standing. If such thing happens, while one is sitting with friends, one becomes nervous and feels small.

N—Miscellaneous

1. *Splenomegaly*—Spleen may be enlarged due to amoebic abscess of the organ or due to thrombosis of portal vein in cases of chronic hepatic *amoebiasis*.

2. *Spleen abscess*—In rare cases, spleen may be the site of infection with *E. histolytica*, resulting in the formation of an abscess the parasites being carried through blood circulation from primary amoebic foci in the intestinal tract.

3. *Adrenal, nephritic, ureteric, vesicular and urethral abscesses* may also be formed due to invasion with *E. histolytica*, haemmatogenously.

4. *Ascites*—Chronic amoebic liver abscess, in the neighbourhood of portal fissure, may induce thrombosis in the portal vein and lead to ascites.

5. *Sigmoido-vesical fistula*—This has been observed in very rare cases of chronic intestinal *amoebiasis*. This condition is invariably accompanied by the formation of adhesions with neighbouring structures.

6. *Paraplegia* may be due to invasion of motor area of brain with the round-worm larvae.

O—Signs and Symptoms due to Nutritional Deficiencies and Disorders

1. *Avitaminosis*—Marked Signs of avitaminosis may occur in prolonged *hymenolepiasis*. It is the result of continued poor intake of food.

2. *Muscular Weakness*—(*i*) *Balantidiasis* of long duration, lowers physical conditions of the patient and muscular weakness is one of the common complaints.

(*ii*) Children suffering from *ascariasis* are usually physically less developed. With the removal of the parasite, the general condition speedily improves, the metabolism is corrected, the nutrition level rises and the children becomes physically fit and mentally alert.

3. *Emaciation*—(*i*) There is usually considerable loss of weight in *balantidiasis*.

(*ii*) in *giardiasis*, if accompanied with chronic diarrhoea, there is always constant loss of weight in children. It is attributed to dehydration.

(*iii*) Rapid loss of weight may occur as prodromal episode of diarrhoea in *amoebiasis*, hepatic involvement and perforation of the liver abscess usually aggravates the condition. Emaciations may be well marked in these patients who worry and resort to unbalanced diet.

(*iv*) Twenty-five per cent cases of *trichiuriasis* are under weight. In some cases, there is marked emaciation and dry-ness of skin.

(*v*) General emaciation of the body, combined with oedema of the face, is one of the characteristic features of *ancylostomiasis/necatoriasis* and there is notable loss of weight in children.

(*vi*) Constant annoyance caused by the wandering females of *E. vermicularis* is also responsible for the constant loss of body weight in children.

(*vii*) Loss of body weight has also been observed in heavy infection of *Taenia saginata*.

4. *Prostration*—Great depression (and exhaustion) of the vital functions is one of the symptoms in acute *amoebiasis.*

5. *Lumbago*—Presence of whip-worms may some times cause continuous pain in the *lumbar region* of the child patients.

From the foregoing narration of signs and symptoms, that may be associated with or consequent upon parasitic infection of gastro-intestinal tract, it seems apparent that practically few lesions and abnormalities of any system of human body have been spared to which one or more combination of the signs and symptoms of intestinal parasitic infection do not coincide. The signs and symptoms of an infection of the digestive tract may sometimes be so misleading that an average clinician is apt to concentrate his attention to the investigation of the system to which they usually pertain. It is quite possible that the parasitic infection may co-exist with any disease complicating the clinical picture and thus confusing the mind of the clinician or, that signs and symptoms of intestinal parasitic invasion may aggravate those of another disease to which they simulate and in this way the clinician may be misled to misjudge the prognosis. But the greatest misfortune comes when, due to utter disregard of the possibility of a particular medical or surgical emergency being the result of intestinal parasitic invasion, the clinician is tempted to go ahead with his usual line of treatment often with disasterous results. Perforation of intestines, causing peritonitis or peritoneal abscess by round worms, appendicities by *E. histolytica*, *Enterobius vermicularis*, lung abscess and empyema by *E. histolytica* or round worms or oedema glottis by *Ascaris* worms in the course of their migration towards the mouth are some of the classical examples which may be cited in this connection. It would, thus be clear, that if the clinician wants to avoid the chances of a dangerous situation, a careful stool examination by a highly experienced clinical pathologist of all patients is an absolute necessity. A single stool examination, if found negative, must be repeated if the circumstances of the case so require.

It warms the very sickness in my heart.
that I shall live and tell him to his teeth,
"Thus diddest thou". (Shakespear).

Method and Material

An ideal method of stool collection is that the individuals, who are to be examined should come to the laboratory to daefecate. A dose of saline purgative (we generally recommend Mag. Sulph.) if administered a couple of hours before, will help in the recovery of the stool sample from the right person. We have recorded more frauds in stool supplies than we have heard of for other commodities. In another survey, not so far published, we experienced that "substitution of other human or animal stools" is not an uncommon practice. Most of the mothers, in this country, due to certain fags and superstitions, usually refuse point blank to give the stool samples of their children. Not infrequently, we were supplied the stools of a servant or an old great grand granny, divided in several faecal containers and labelled as (1) *Ghazala* (12 yrs); (2) *Ghayur* (10 yrs); (3) *Ghias* (7 yrs); (4) *Ghosia* (3 yrs) and etc., etc. This experience unfortunately came from one of the very highly cultured family, living in elite quarters of Lahore. We are now amply used to the rhythms of sarcastic remarks and dare not grumble at these here. We know

"a double-blessing is a double-grace". It will not be out of place to mention that on the other hand we had a tame success with a warry mother in obtaining a stool sample of the child complaining of tooth-ache. For this success we owe a sense of gratitude to one of our colleagues in "*practicing field.*" This instance gives us a glimpse of attitudes. Our intelligentia even, does not understand that a stitch in time saves nine. They do not mind a pound for cure but grudge a penny for prevention. Probably they do not want to be conscious of the coming events, as it makes cowards of us all. But we will also remind them the poet of young who said "Pygmies are pygmies still, though perched on alps."

Alas, regardless of their doom,
The little victims play!
No sense have they of ills to come,
Nor care beyond today. (Gray).

The conservancy staff of the City Corporation of Lahore collects daily at a centre. From this place, their chief deploys them for duty. We made use of this situation for these studies with his active co-operation.

The conservancy staff was fully briefed of the devastating activities of the intestinal parasites and damage feared to the community. The aims and objects of the fact-finding programme were fully made known to them. Special instructions were also given regarding the collection of the stools samples. This briefing in our experience worked well in creating an "acceptance attitude." All out co-operation was not only promised but also came forth as a spontaneous action and our procedure was simplified. A group of persons (as many as could be conveniently examined every day) was given labelled collection jars a day before examination, with the instructions that the jar (with the stools in it) will be brought next day to the depot and delivered to the laboratory man on duty. At the time of collection, a brief history and necessary details of each donor was also entered on the correspondingly numbered *pro forma* on the spot. The distribution of bottles to the fresh batch were also made at this time. The whole work was carried out under the strict supervision of the Chief Sanitary Inspector-in-Charge of the conservancy staff.

Gross inspection of each stool sample was made to know the consistency and component elements before subjecting it to miscroscopic examination.

For direct examination, small lump of stool was mixed in concentrated solution of NaCl (Sp. gr. 1,200) in a 15 ml capacity specimen tube. A clean 22 × 22 mm. square cover slip was super imposed on the top of the film. The cover slip, after 20—30 minutes was removed and placed on a clean microscope slide for examination. Each examination was made for 10—15 minutes under high power before it was stained with a drop of lugol's iodine to study the diagnostic features of parasites present. This method usually gives information about these parasites which are far the largest in number in the case under study. Slight and neglegible infections are, however, invariably missed. In surveys, we are interested in knowing what ordinarily we do not bother to find, i.e. over-all quality of parasite load in the community.

In some cases, a small lump of the stool was emulsified in a drop of physiologic saline (normal salt solution) on a slide. A portion was stained with Lugol's iodine and a portion

was left unstained. Each portion was then covered with a separate cover slip. Precautions were taken that a film of *reading opacity* was obtained in each case.

A garden-pea size of the stool sample was well macerated with a glass rod - in tap water or physiological solution (with 10 parts of water or physiological salt to each part of sample in a centrifuge tube and spinned at a moderate speed (1500—2000 r. p. m.) for 1-2 mts. The supernatant liquid was poured off. Two to three spinings were done before final examination.

Zinc Sulphate centrifugal-floatation technique although considered by some to give better results did not give any substantial advantage over the direct method was not therefore, used extensively.

Polyvinyle Alcohol fixative (PVA) and MIF (Merthiolate-iodine-Formaldehyde) fixative-stain were also not used.

We must admit, that the methods we made use of do not pretend to give the exact number of parasites harboured by the case but these techniques do furnish an approximation, which makes it possible to classify the cases. The relative severity of the parasites was conveniently ascertained from the number of cysts/ova per field. The presence of each parasite was categorically graded into light, mild, moderate, heavy and severe infection in the light of their presence *viz.*, one in several fields, 1-2 in each field, 2-3 in each field, 3-4 in each field, or 4 and above in each field on an average.

All these examinations were made for 41 days, commencing from February 16 through March 29, 1963. A perusal of table V will show that Lahore has minimum number of clinical cases of diarrhoea and dysentery during this part of the year. During this period the climatic conditions and other factors conducive to higher incidence of gastro-intestinal disorders are least favourable. It is to be pr`sumed that the incidence of clinical manifestations of the pre-existing parasitic infections as well as factors favourable for transmission of parasites are comparitively few during this period.

Original Observations

"I cannot confirm the public charge against him, to help. Avenge the dark murder."
(Sophocles: Oedipus Rex).

A. Incidence of all species of intestinal parasites—

The incidence of various intestinal parasites in the conservancy staff of the City Corporation, Lahore (based on one, normally passed stool examination, both by direct and concentration methods) is presented in table VII.

It may be considered as a most conservative estimation, because consensus of opinion warrants that at least six repeated examinations should be made before to be certain that a possible infection has not been missed. To obtain a satisfactory diagnosis, a microscopist needs to be slow and steady because in survey work, the examination is something more than mere routine. In our experience, repeated examinations do not ordinarily add significantly to what may be discovered in two-direct and one concentration methods. For all practical purposes, therefore, we may take these figures as true approximations.

During this survey, we found that 89·75 per cent of the total number of conservancy staff examined, were infected with either protozoan or helminthic parasites. The majority

were, however, found carrying helminthic parasites.

The table illustrates most graphically that we could recover only five species of protozoas and six species of helminths.

1. *Protozoan parasites*—55·13 per cent of the persons examined were found carrying protozoal parasites—

(*i*) The outstanding feature of this survey for intestinal parasites is that although *Entamoeba histolytica* is one of the much more talked about protozoan parasites in this country, yet the figures obtained in this survey do not seem to present any serious situation. Out of 86 persons with protozoan infection, only 8 or 5·13 per cent had *E. histolytica*. In all of these positive cases, the infection level was of very low degree and the form of the parasite involved was small. We did not prepare any permanent stained preparation and therefore, could not ascertain the race involved. None of the positive cases complained of any abdominal distress, diarrhoea or dysentery at the time of examination nor gave a history of dysentery, having occurred on any previous, or very recent past occasion. These were not dysenteric convalescents. Shall we look upon these cases as potential sources of danger to the community or consider them carriers of a closely allied commensal amoeba (*E. hartmanni* or non-pathogenic small strain of *E. histolytica*. We wonder, if small strain of pathogenic *E. histolytica* is of common occurrence and is decidedly a separate and stable entity. Morphological differences—position of karyosome, character of encrusted chromatin on the periphery of the nuclear membrane, character of chromatoidal bodies, presence of glycogen mass and structure of vacuolation in cysts are better appreciated in haematoxylin stained slides. To differentiate variants in routine surveys is something which appears more of cheating than real.

(*ii*) *Entamoeba coli* was found only in 20·52 per cent cases. The infection rate in all cases remained light. In few odd cases the infection level was found of mild grade.

(*iii*) *Iodamoeba butschlii* occurred in 15·29 per cent cases. The quality of infection was again, generally of light or mild nature. In two persons, however, the infection was found to be of moderate or heavy degree. This amoeba is of doubtful importance. It was once suggested, that the motile forms may sometimes invade intestinal wall and taken to extra-intestinal viscera and initiate lesions. We failed to encounter any case with symptoms.

(*iv*) *Endolimax nana* was found to be most uncommon. This amoeba is most delicate and does not survive long under unfavourable extrinsic conditions. These studies were made during early spring. If the dry and moderately cold climat accounts for the low incidence; it will be answered in surveys to be conducted during an other season. Light infection was discovered in one

solitary case.

(v) *Giardia intestinalis* was the only one species of flagellates, which was found during these studies. Out of 86 persons carrying protozoan parasites, 45 were passing cysts of the duodenal flagellate. 29 per cent of the infections, appears to be an unusually high incidence in an adult group. These observations lead us to suspect this infection to be strongly endemic in Lahore. As far as we remember the infection rate in adults elsewhere usually varies from 1—10 per cent. In very few surveys however, the incidence was found to be 20—30 per cent. We should expect much higher incidence in children.

These flagellates are reported to feed on the mucous secretions. Their presence and constant irritation to the glandular crypts is not considered to give an evidence of duodenal disturbances. In our experience it does manifest dull abdominal distress and periodic loose bowels. The trouble has been periodically well marked in children than in adults. This survey, however, did not lend us any support to understand the symptoms initiated by this parasite alone.

2. *Helminthic parasites*—The conservancy staff of the city Corporation of Lahore was found infected with six different types of helminths. Out of 156 persons examined, 112 (71·8 per cent) were found carrying one or the other of these parasitic worms.

(*i*) Highest number of cases suffering from helminthiosis were infected with hook worms. Since this report is exclusively based upon the recovery of eggs in the stools, an attempt to identify the species involved was not made. However, on our previous experience we may mention here that in Lahore both *Ancylostoma duodenale* and *Necator americanus* are present. The old world form predominates, fairly widely, over the American murderer which may occur in negligible few numbers.

Ahmadet el (1959) found 454 males and 924 females out of 1378 adults isolated, two of which, however (one male and a female) were *N. americanus*. In another communi- cation, it is reported that for every 500 *Ancyclostomas* there occur 1000 *Necators* in Indo-Pakistan sub-continent. From a study of the socio-economic level of this community, its cultural pattern, appropriate conditions of the surrounding fields and suitability of meteorological conditions, we may probably get an answer to the high ncidence.

We did not try to find out the intentity of hook worm infection in any of the 69 positive cases found. We always wished to know the worm burden in these cases by one of the egg count techniques but could not manage easily in the midst of great, mural and extral mural stress. Light, mild, moderate, heavy and severe cases of infection shown in the table I are based on the number of eggs found per field and the clinical symptoms presented by the positive cases. We hope to fulfil this desideratum in some of our next attempts.

(*ii*) *Ascaris lumbricoides* is one of the most familiar parasite of the Lahorites. We have yet to come across a man who would say that he did never pass "earth worm" like nematod in his life time. According to Stoll 488 million cases, constituting approximately 75·78 per cent of the total world cases, occur in Asia alone.

More or less one third of the total persons examined had round worms. The infection, however, was found to be of mild or moderate grade.

(*iii*) *Enterobius vermicularis* is also one of the most common parasites. About seat worm we always feel that on interogation 99·9 per cent will claim of having *enterobiasis* and 0·1 per cent who will deny will be either liars or shy. The eggs of these worms are seldom present in more than 5·10 per cent stools of infected individuals. For significant results, perianal scrapings or Grahams swab may be examined soon after the subject awakens in the morning. It is amazing to us that in this survey, the coprological examination gave us 14·74 per cent infection. According to Stoll the incidence of *enterobiasis* in Asia is 32·5 millions, and constitutes 15·5 per cent of the world figure.

(*iv*) *Trichocephalus trichiurus* is usually common parasite in Lahore but it was most unexpected experience for us to find it only in one person in this group.

Stoll has stated that 355·1 million persons in the world suffer from whip-worm annually, out of which 63·93 per cent inhabit Asia alone. Ordinarily the incidence and intensity of trichuriasis is on an average high in areas of high rainfall, high humidity and dense shade. The eggs do not survive long in temporary drought and heat. Direct sunlight and severe cold kills them. Cannot do well in decomposing organic matter or on heavy soil, cinder, ashes and dust either.

(*v*) *Hymenolepis nana* occurs more commonly in young age groups than in advanced age groups.

According to Stoll, 20·2 millions people of the world suffer from *hymenolepiasis* annually. It is said to be particularly common in Indo-Pakistan sub-continent. The incidence in the community under discussion, was found to be 4·49 per cent, which was considerably lower than what we have observed in routine examinations. However, we should not forget that both, the incidence and the intensity of the infection, are considerably high in children. In endemic areas the adults gradually develop resistance to its infection. There is definite resistance to super-imposed infections also. The continued high infections are attributed to *inter-auto-infections*.

(*vi*) *Taenia saginata* is responsible for beef taeniasis particularly in Muslim countries. According to Stolls' most conservative estimate, 38·9 millions persons of the world, annually fall a prey to the depredations of beef-tape-worm. Although an average Pakistani claims to consume thoroughly cooked beef, yet there is no dearth of such people (usually in urban population alone) who relish only smoked *tikka-kababs*, *seish-kababs*, pickled and salted hunter beef. The number of beef-tape-worm infections today are steadily increasing in those Lahorites, who have a fancy for these meat preparations. Most of the conservancy staff of the city Corporation of Lahore cannot either afford to eat *Kababs* or due to some religious restrictions and taboos (specially *Balmikis*) do not take meat at all. Negligible infection rate found in this community is a mere reference to the situation.

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TABLE VII

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED—CONTD.

Entamoeba histolytica	*Entamoeba coli*	*Iodamoeba butschlii*	*Endolimax nana*	*Giardia intestinalis*	*Hook worms*	*Hymenolepis nana.*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiura*	*Taenia saginata*	Age (in years)
..	..	..	..	..	..	..	2	..	..	..	16
..	..	R	..	R	..	..	..	..	..	..	16
R	..	R	..	R	..	..	3	..	..	..	16
R	..	..	..	..	..	..	..	..	..	..	17
..	R	..	..	..	1	..	2	..	..	..	17
..	..	..	..	..	1	..	1	..	..	..	17
..	R	R	..	R	..	..	2	..	..	..	17
..	..	..	..	R	..	..	..	R	..	..	18
R	..	R	..	..	2	..	3	..	..	..	18
..	R	..	..	..	..	..	1	..	..	..	18
R	..	..	..	..	..	..	2	..	..	..	18
..	R	..	..	..	..	..	1	..	..	..	18
..	R	..	..	..	1	..	..	..	..	..	18
..	..	..	..	..	1	..	2	..	..	..	18
..	..	..	..	R	..	..	1	..	..	..	18
..	..	..	..	R	..	..	1	..	..	..	18
..	..	..	..	..	..	..	1	..	..	..	18
..	..	..	..	..	..	..	1	..	..	..	18
..	..	..	..	..	1	1	2	..	..	..	18
..	..	..	..	R	..	..	2	1	..	..	18
..	R	..	..	..	R	..	..	..	..	..	18
..	..	..	..	R	1	..	..	..	..	..	18
..	..	..	..	..	R	..	..	..	..	..	18
..	R	..	..	..	..	..	2	..	..	..	18
..	..	R	..	..	R	..	..	..	..	..	18
..	R	..	..	R	..	..	1	..	..	..	18
..	..	..	..	..	1	..	1	..	..	..	18

TABLE VII—contd.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED.—CONTD.

Entamoeba histolytica	*Entamoeba coli*	*Iodamoeba butschlii*	*Endolimax nana.*	*Giardia intestinalis*	*Hook worms*	*Hymenolepis nana*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiuru*	*Taenia saginata*	*Age (in years)*
..	..	R	R	..	..	..	..	..	..	..	18
..	R	..	..	..	..	..	..	..	..	..	18
..	..	..	..	..	..	..	..	..	..	..	18
..	..	..	..	..	1	..	2	..	..	..	18
..	..	..	..	..	1	..	..	..	..	..	18
..	..	..	..	..	1	..	..	..	..	..	18
..	..	..	..	..	..	..	2	..	..	..	18
..	..	..	..	R	1	..	2	..	..	..	18
..	..	..	..	..	..	1	..	1	..	..	18
..	..	..	..	..	..	..	1	..	..	..	18
..	..	..	..	3	..	..	..	1	..	..	18
..	..	..	..	..	..	..	..	..	..	..	19*
..	1	..	..	..	2	..	..	..	..	..	19
..	R	R	..	..	1	..	..	..	..	..	19
..	R	..	..	R	..	..	1	..	..	..	19
..	..	..	..	..	..	..	1	..	..	..	19
..	..	..	..	..	1	..	1	..	..	..	19
..	..	..	..	..	..	..	..	..	..	..	19*
..	R	..	..	R	..	..	..	..	..	..	19
R	R	..	..	..	1	..	..	..	..	..	19
..	..	..	..	..	1	..	..	..	..	..	20
..	..	..	..	..	..	..	2	2	..	..	20
..	..	..	..	R	R	..	..	..	..	..	20
..	..	..	..	..	..	..	1	..	..	..	20
..	..	..	..	..	..	..	1	..	..	..	20
..	..	..	..	..	..	..	1	..	..	..	20
..	..	..	..	..	1	..	..	..	..	..	20

OOLS

TABLE VII—contd.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED—CONTD.

Age (in years)	Entamoeba histolytica	Entamoeba coli	Iodamoeba butschlii	Endolimax nana	Giardia intestinalis	Hook worms	Hymenolepis nana	Ascaris lumbricoides	Enterobius vermicularis	Trichocephalus trichiura	Taenia saginata
20	..	..	..	..	R	2	..	..	..	..	..
20	..	..	..	..	..	..	..	1	..	..	..
20	..	R	R	..	..	..	..	1	..	..	..
20*	..	R	..	..	R	..	..	1	..	..	..
20	..	..	..	..	R	..	..	..	1	..	..
20	..	..	..	..	R	..	..	..	..	..	..
20	..	..	..	..	..	1	..	..	1	..	..
20	5	16	9	1	18	23	2	31	7	..	..
21	..	..	..	..	R	1				..	..
22	..	..	3	..	..	..	1	..	..	..	..
22	..	..	..	..	..	1	..	..	..	..	..
22	..	R	..	..	..	..	..	..	..	..	..
22	..	..	R	..	..	1	..	..	..	..	..
22	..	..	..	..	..	..	1	..	..	..	..
22	..	..	..	..	..	1	..	1	..	..	..
23	R	R	R	..	R	..	..	..	1	..	..
23	..	..	..	..	R	..	..	..	..	..	..
23	..	..	..	..	..	1	..	..	..	..	..
23	..	..	R	..	..	..	..	..	..	..	..
23	R	R	4	..	R	..	..	..	2	..	..
24	..	R	..	..	..	1	..	2	..	..	..
24	..	R	R	..	..	..	..	..	..	..	..
24	..	..	R	..	..	1	..	..	..	..	..
24	..	R	..	..	..	4	..	..	..	..	..
24*	..	..	..	..	..	..	..	..	..	..	..
24	..	R	..	..	..	..	..	..	..	..	..

TABLE VII—contd.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED—CONTD.

Entamoeba histolytica	*Entamoeba coli*	*Iodamoeba butschlii*	*Endolimax nana*	*Giardia intestinalis*	*Hook worms*	*Hymenolepis nana*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiura*	*Taenia saginata*	Age (in years)	
..	..	R	..	R	..	..	1	..	..	..	24	..
..	..	..	..	..	2	..	..	..	..	..	24	..
..	..	R	..	R	1	..	..	..	..	..	24	..
..	..	..	..	..	..	..	1	..	..	..	24	..
..	..	..	..	..	..	..	1	..	..	..	24	..
..	..	..	..	R	1	..	1	..	..	..	24	..
..	..	..	..	..	..	..	1	..	..	..	24	..
..	..	..	..	..	1	..	R	..	..	..	24	..
..	..	R	..	..	..	..	..	..	..	..	25	..
..	..	..	..	..	..	..	..	..	..	..	25*	R
..	R	..	..	..	1	..	..	..	..	..	25	..
..	..	..	..	R	1	..	..	1	..	..	25	..
..	R	..	..	..	1	..	1	..	..	..	25	..
..	..	..	..	R	3	..	2	..	..	..	25	..
..	R	..	..	R	1	..	1	..	..	..	25	..
..	..	..	..	..	..	..	..	..	..	..	25*	1
..	..	..	..	..	..	..	..	1	..	..	25	
..	R	..	..	..	..	..	..	1	..	..	25	..
2	11	10	..	11	18	2	12	5	..	..	39 cases	..
..	1	..	..	..	2	..	..	..	..	..	26	..
..	..	..	..	..	..	..	1	..	..	..	26	..
..	1	..	..	..	1	..	..	..	..	..	26	..
..	..	..	..	..	1	..	1	..	..	..	27	..
..	..	..	..	..	..	..	..	1	..	..	27	
..	..	..	..	R	..	1	1	..	..	..	27	..
..	..	..	..	..	1	..	..	..	..	..	27	

TABLE VII—contd.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED—CONTD.

Age (in years)	*Entamoeba histolytica*	*Entamoeba coli*	*Iodamoeba butschlii*	*Endolimax nana*	*Giardia intestinalis*	*Hook worms*	*Hymenolepis nana*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiura*	*Taenia saginata*	Age (in years)
24	..	..	..	..	..	1	..	..	..	..	..	27
24	..	..	..	..	1	1	..	..	..	..	..	27
24	..	..	..	..	..	1	..	..	..	..	..	27
24	..	..	..	..	..	1	..	..	..	..	..	28
24	..	..	..	..	..	1	..	1	..	..	..	28
24	..	..	..	..	..	..	..	..	..	..	..	28*
24	..	..	..	..	..	..	..	..	..	..	..	28*
24	..	..	..	..	..	..	..	..	..	..	..	28*
24	..	..	..	..	..	1	..	..	..	..	..	30
24	..	..	R	..	..	..	..	..	..	..	..	30
25	..	..	..	..	..	..	..	..	.	..	..	30
25*	R	..	R	..	..	..	..	..	..	..	..	30*
25	..	..	..	..	R	..	..	..	..	..	..	30
25	..	..	..	..	..	..	..	2	1	..	..	30
25*	..	..	..	..	R	1	..	1	..	..	..	30
25	..	..	..	..	..	..	..	..	..	..	..	30*
25	..	..	..	..	R	1	..	..	..	..	..	30
25*	1	2	2	..	5	12	1	6	2	..	..	24 cases
25	..	..	1	..	..	1	..	1	R	..	1	31
25	..	R	..	..	..	1	..	..	..	..	..	35
39 cases	..	..	..	..	..	..	..	..	1	..	..	35
26	..	..	..	..	..	3	..	..	..	..	..	35
26	..	..	..	..	R	..	..	..	..	..	..	35
26	..	..	..	..	..	1	..	..	1	..	..	35
27	..	..	..	..	..	..	..	..	1	..	..	35
27	..	1	1	..	1	4	..	1	4	..	1	7
27												

TABLE VII—contd.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED CONTD.

Entamoeba histolytica	*Entamoeba coli*	*Iodamoeba butschlii*	*Endolimax nana*	*Giardia intestinalis*	*Hook worms*	*Hymenolepis nana*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiura*	*Taenia saginata*	Age (in years)
..	1	..	..	R	..	1	1	1	..	..	38
..	..	..	..	..	..	..	1	..	..	..	38
..	..	..	..	R	1	..	..	..	..	..	38
..	1	..	..	..	1	..	..	R	..	..	40
..	..	..	..	..	..	..	..	..	..	..	40*
..	..	..	..	R	1	..	..	..	..	..	40
..	2	..	..	3	3	1	2	2	..	..	6 cases
..	R	..	..	..	..	..	..	..	..	..	42
..	..	..	..	..	1	..	..	..	..	..	45
..	..	..	..	..	..	..	..	..	..	..	45*
..	1	..	..	..	1	..	..	..	..	..	45
..	..	..	..	..	1	..	..	..	..	..	45
..	..	..	..	..	..	..	..	..	..	..	45*
..	2	..		..	3	..	..	..	..	..	6 cases
..	..	R	..	..	..	..	..	..	..	..	50
..	..	..	..	R	..	..	..	..	..	..	50
..	..	R	..	..	..	..	..	..	..	..	50
..	..	..	..	R	..	..	1	1	..	..	50
..	..	..	..	..	..	..	..	..	..	..	50*
..	R	..	..	..	..	..	..	1	..	..	50

TABLE VII—concld.

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES IN THE STOOLS OF CONSERVANCY STAFF EXAMINED—CONCLD.

Entamoeba histolytica	*Entamoeba coli*	*butschli*	*Endolimax nana*	*Giardia intestinalis*	*Hookworm*	*Hymenolepis nana*	*Ascaris lumbricoides*	*Enterobius vermicularis*	*Trichocephalus trichiura*	*Taenia saginata*	Age (in years)	
..	..	..	..	R	..	..	..	..	..	..	50	
..	..	..	..	R	..	..	3	..	..	..	50	
..	..	..	..	..	1	..	..	1	..	..	50	
..	1	..	..	..	..	..	..	..	..	..	55	
..	..	..	..	R	1	..	..	..	..	..	55	
..	..	..	..	..	1	..	..	..	..	..	56	
..	..	..	..	R	1	..	..	..	R	..	58	
..	..	..	..	R	2	1	..	..	..	..	60	
..	..	..	..	..	1	..	..	..	..	..	65	
..	2	2	..	7	6	1	2	3	1	..	15 cases	
8	36	24	1	45	69	7	54	23	1	1	140 Total Positive 89·75	
5·13	20·52	15·39	0·64	28·85	44·23	4·44	34·62	14·74	0·64	0·64	Percentages of total examined	
5·72	23·71	17·4	0·71	32·14	49·29	5·0	38·57	16·43	0·71	0·71	Percentages of total found infected.	

Abbreviations :—

*Cases free of all infections, absent, unknown.
R—one in several fields, *Light infection.*
1—one or two in each field, *Mild infection.*
2—two or three in each field, *Moderate infection.*
3—three or four in each field, *Heavy infection.*
4—four or more in each field, *Severe infection.*
Eh.—*Entamoeba histolytica*, (Dysentery amoeba).
Ec.—*Entamoeba coli*, (Colon amoeba).

En.—*Endolimax nana*, (Lesser amoeba).
Ib.—*Iodamoeba butschlii*, (Iodine-cysts).
Gi.—*Giardia intestinalis*, (Duodenal flagellate).
Hw.—*Ancylostoma duodenale*—(Old world Hook-worm).
Necator americanus —(New world Hook-worm).
Hn.—*Hymenolepis nana*, (Murine tape-worm).
Al.—*Ascaris lumbricoides*, (Giant Intestinal round-worm).
Ev.—*Enterobius vermicularis*, (Seat-worm).
Tt.—*Trichocephalus trichiurus*, (Whip-worm).
Ts.—*Taenia saginata*, (Beef-tape-worm).

B—Incidence of Protozoan and Helminthic parasites alone (Table VIII)

(*i*) *Incidence of intestinal protozoa*—This survey demonstrates that only 21 persons out of 140 found infected, were harbouring protozoan parasites. 14 of these cases were in 16—30 years age groups while only 7 fall in 31—65 years age group.

(*ii*) *Incidence of intestinal helminths*—This survey records that the number of persons harbouring helminthic parasites alone was almost twice the number carrying protozoan parasites alone. 34 of these persons were in 16—30 years age group, while 10 fall in 31—65 years age group.

(*iii*) *Incidence of mixed infections both protozoan and helminthic parasites*—The number of persons carrying mixed infection was more or less equal to the persons infected with protozoan and helminthic parasites separately. 62 persons harbouring both helminthic and protozoan parasites were in 16—30 years age group while 21 persons fall within 31—65 years age group.

TABLE VIII

SHOWING INCIDENCE OF PROTOZOAN OR HELMINTHIC PARASITES ALONE AND CASES CARRYING BOTH PROTOZOAN AND HELMINTHIC PARASITES.

Age groups (in years)	No. of cases examined	No. of cases positive ((per centages)	No. of cases carrying protozoan parasites alone (percentages)	No. of cases carrying helminthic parasites alone (percentages)	No. of cases carrying both helminthic and protozoan parasites (percentages)
16—20	59	56(94·94)	6(10·71)	23(41·07)	27(48·22)
21—25	39	35(89·74)	6(17·14)	1(31·43)	28(51·43)
16—30	24	19(62·51)	2(10·53)	10(53·68)	7(35·79)
16—30	122	110(90·16)	14(12·73)	34(30·91)	62(56·36)
31—35	7	7(100·00)	1(14·29)	4(57·14)	2(28·57)
36—40	6	5(83·33)	0(0·00)	1(20·00)	4(80·00)
41—45	6	4(66·66)	1(25·00)	2(50·00)	1(25·00)

TABLE VIII—contd.

SHOWING INCIDENCE OF PROTOZOAN OR HELMINTHIC PARASITES ALONE AND CASES CARRYING BOTH PROTOZOAN AND HELMINTHIC PARASITES—CONTD.

Age groups (in years)	No. of cases examined	No. of cases positive (percentages)	No. of cases carrying protozoan parasites alone (percentages)	No. of cases carrying helminthic parasites alone (percentages)	No. of cases carrying both helminthic and protozoan parasites (percentages)
45—65	15	14(93·33)	5(35·71)	3(21·43)	6(42·86)
31—65	34	30(82·35)	7(23·33)	10(33·33)	13(43·33)
Total	156	140(89·75)	21(15·00)	44(31·43)	75(53·57)
Age-groups			16-65 yrs.	16-30 yrs.	30-65 yrs.
Cases carrying protozoan parasites (percentages) ..			36(69·09)	20(66·66)	96(69·43)
Cases carrying helminthic parasites (percentages) ..			96(87·27)	23(76·66)	119(85·0)

C—INTENSITY OF INTESTINAL PROTOZOAN AND HELMINTHS (TABLE IX)

In all the cases carrying *E. histolytica*, the burden of protozoan appeared to be relatively low. In this connection we would call attention to periodicity in cyst formation and irregularity in laying of eggs. This interruption is an excitement to those who study the effect of drugs. The factors responsible for the cyclic behaviour of the parasites may be many, both of extrinsic and intrinsic origin. It is a regular phenomenon and has been studied in almost all parasites. In *E. coli* the periodicity is of 10—14 days, *E. histolytica* observes a period of 6—20 days and more and cyst production in *G. intestinalis* is also distinctly cylic in character (Ansari, 1953).

Out of 36 cases of *E. coli*, 29 had very light infection, while only 7 had mild infection. The picture is more or less similar for *I butschlii*. In this case 21 were light infections, 1 each was of mild, heavy and severe grade. There was only one case of *E. nana* and that too was of light infection. *G. intestinalis* was found to be of light grade in 43, mild and heavy in 1 each.

Hook-worm was found to be very predominating. The burden of the worm was light in most cases. It was of mild or heavy grade in one case each. *A lumbricoides* was also very common, mostly varying between mild and moderate infections. 3 persons, however were carrying very heavy infection. Infections was, however, light in one person. *E. vermicularis* was found in 23 cases. Mild grade infection was noticed in 18 persons. Light grade and moderate infection were found in 3 and 2 cases respectively. It was most unusual to find only one case of *T. trichiurus* in this series. The infection was of low grade. Mild infection of *H. nana* was found only in 7 cases. It also appeared to be unusual. A solitary case of mild grade infection of *T. saginata* was also detected.

TABLE
SHOWING NUMBER OF LIGHT, MILD, MODERATE, HEAVY AND SEVERE OF THE CONSERVANCY

Age (in years)	Entamoeba histolytica					Entamoeba coli					Iodamoeba butschli				
Age groups	Li	Mi	Mo	He	Se	Li	Mi	Mo	He	Se	Li	Mi	Mo	He	Se
16 to 20 ..	5	..	..	..	..	15	1	..	..	..	9	..	..	..	..
21 to 25 ..	2	..	..	..	..	11	.	..	..	..	8	..	..	1	1
26 to 30 ..	1	..	..	..	..	..	2	..	..	..	2	..	..	..	..
31 to 35 ..	..	..	.	..	..	1	..	..	..	..	..	1	..	..	..
36 to 40 ..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..
41 to 45 ..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..
46 to 65 ..	..	..	..	..	.	1	1	..	..	..	2	..	..	..	..
Total ..	8		..	..	..	29	7	..	..	..	21	1	..	1	1

IX

CASES OF INFECTION OF INTESTINAL PARASITES IN SEVEN DIFFERENT AGE GROUPS STAFF, LAHORE

Endolimax nana					Giardia intestinalis					Hook worms				
Li	Mi	Mo	He	Se	Li	Mi	Mo	He	Se	Li	Mi	Mo	He	Se
1	..	..	..	..	17	..	..	1	..	4	16	3	..	..
..	..	..	..	..	11	..	..	..	..	..	15	1	1	1
..	..	..	..	..	4	1	..	..	..	..	11	1	..	..
..	..	..	..	..	1	..	..	..	..	..	3	..	1	..
..	..	..	..	..	3	..	..	..	..	..	3	..	..	..
..	..	..	..	..	..	..	..	..	..	..	3	..	..	..
..	..	..	..	..	7	..	..	..	..	..	5	1	..	..
1	..	..	..	..	43	1	..	1	..	4	56	6	2	1

TABLE
SHOWING NUMBER OF LIGHT, MILD, MODERATE, HEAVY AND SEVERE
OF THE CONSERVANCY

Age (in years)	Hymenolepis nana					Ascaris lumbricoides				
Age groups	Li	Mi	Mo	He	Se	Li	Mi	Mo	He	Se
16 to 20	..	2	..	..	..	..	17	12	2	..
21 to 25	..	2	..	..	..	1	9	2	..	..
26 to 30	..	1	..	..	..	..	5	1		..
31 to 35	..	..	..	..	..	..	1	..	..	..
36 to 40	..	1	..	..	..	..	2	..	..	..
41 to 45	..	..	..	..	..	..	..	..	..	..
46 to 65	..	1	..	..	..	..	1	..	1	..
Total ..	..	7	..	..	..	1	35	15	3	..

IX—contd.

CASES OF INFECTION OF INTESTINAL PARASITES IN SEVEN DIFFERENT AGE GROUPS STAFF, LAHORE

Enterobius vermicularis					Trich ocephalustriohiurus					Taenia saginata				
Li	Mi	Mo	He	Se	Li	Mi	Mo	Ho	Se	Li	Mi	Mo	He	Se
1	5	1		..	..	..	..	..	..	..	..	..	..	..
..	4	1	..	..	..	..	..	..	..	..	..	..	..	..
..	2	..	..	..	..	..	..	..	..	..	..	..	..	..
1	3	..	..	..	..	..	..	..	..	..	1	..	..	..
1	1	..	..	..	..	..	..	..	..	..	..	..	..	..
..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
..	3	..	..	..	1	..	..	..	..	..	..	..	..	..
3	18	2	..	..	1	..	..	..	..	..	1	..	..	..

D. INCIDENCE OF VARIOUS INTESTINAL PARASITES WITH REFERENCE

Usually age influences the incidence of various intestinal parasites in a community. age groups are equally susceptible to the infection of various intestinal parasites. Some chance of exposure. Decline in incidence; due to real immunity to intestinal parasites Majority of the intestinal parasites are typically much more prevelant in children than in by the children, especially in the courtyard and around the house does not only pollute the en-and wallow in the same fields, pick up viable eggs and unknowingly ingest them. Ascaris manure obtained from human night-soil are another source of contamination. Eating of Hook worm infection occurs in fields. Children walking barefooted in the fields and more chances of contact with the infective stage. Here, it is primarily the occupation up to 30 years of age and marked decline after 40 years of age, *H. nana* is again a door

TABLE

SHOWING INCIDENCE OF VARIOUS PARASITES IN

Age groups (in years)	Total Examined	Total Infected with percentages	Eh.	Ec.	Ib.	En.
16 to 20 ..	59	56	5	16	9	1
		94·94	8·93	28·57	16·07	1·78
21 to 25	39	35	2	11	10	0
		89·74	5·71	31·43	28·57	0
26 to 30 ..	24	19	1	2	2	0
		62·51	5·26	10·53	10·53	0
31 to 35 ..	7	7	0	1	1	0
		100·00	0	14.29	14·29	0
36 to 40 ..	6	5	0	2	0	0
		83·33	0	40	0	0
41 to 45	6	4	0	2	0	0
		66·66	0	50	0	0
46 to 65 ..	15	14	0	2	2	0
		93·33	0	14·29	14·29	0
Total	156	140	8	36	24	1
Percentages of the total examined		89·75	5·13	20·52	15·39	0·64
Percentages of the total infected			5·72	23·71	17·14	0·71

TO AGE OF PERSONS (TABLE X.)

The epidemiological situation suggests, that not as a rule but as a matter of fact, all adults do not show high incidence of a parasite perhaps because they had lesser probably occurs in few. This immunuty is usually partial in nature and is an exception. adults not as a rule again but because they court infections. Promiscuous defaccation vironment but also cause of contamination of the same children in return. Children play Ascries is door yard and house-hold infection and this is how it spreads. Vegetables raised on Eating of imprefectly washed raw vegetables is one of causes of high incidence of intestinal parasites. play grounds contact the infective stage. The man behind the plough has comparatively of the victim which is responsible for the incidence. Its greatest incident may be expected yard infection and may be expected high in children than adults.

X

DIFFERENT AGE GROUPS (IN YEARS).

Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
18	23	2	31	7	0	0
32·14	41·07	3·57	55·35	12·50	0	0
11	18	2	12	5	0	0
31·43	51·43	5·71	34·29	14·28	0	0
5	12	1	6	2	0	0
26·84	63·16	5·26	31·58	10·53	0	0
1	4	0	1	4	0	1
14·29	57·14	0	14·29	57·14	0	14·29
3	3	1	2	2	0	0
60	60	20	40	40	0	0
0	3	0	0	0	0	0
0	75	0	0	0	0	0
7	6	1	2	3	1	0
50·00	42·85	7·14	14·29	21·43	7·14	0
45	69	7	54	23	1	1
28·85	44·23	4·49	34·62	14·74	0·64	0·64
32·14	49·29	5·00	38·57	16·43	0·71	0·71

The community under observation consisted of adults alone. We are, therefore, not competent to express our opinion or comment on the rate of incidence in age groups between one month to 15 years. For convenience of discussion we have split the community into seven series of five years each. The incidence of infection of both pathogenic and non-pathogenic protozoa and helminths for each 5 years group is presented in Tables XI—XVIII.

(*i*) *Incidence of infection in* 16—20 *years of age group* (*Table XI*)—Out of 59 persons examined, 56 persons were found infected either with protozoan, helminthic or both parasities.

Amongst the protozoan parasites, *G. instestinalis* (18), *E. coli* (16), *I. butschlii* (9), *E. histolytica* (5), and *E. nana* (1) occurred in the descending order of incidence mentioned each parasit e.The load of infection was light, except one case of *G. intestinalis* in which the infection was rather heavy.

A. lumbricoides—was found to be comparatively common helminthic infection. The load of infection was mild in 17, moderate in 12 and heavy in 2 cases.

Hook worm infection was found in 41 per cent cases. It was light in 4, mild in 16 and moderate in 3 .

Seat worm infection was found in 12·50 per cent cases. The infection load was more or less of mild nature.

Mild dwarf type-worm infection occurred in two cases.

TABLE XI

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES (PURE AND MIXED INFECTION) IN 16—20 YEARS OF AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh	1	1	3	0	2	1	0	1	1	0	0
Ec.	1	2	4	0	5	7	0	8	0	0	0
Ib.	3	4	0	1	3	4	0	4	1	0	0
En.	0	0	1	0	0	0	0	0	0	0	0
Gi.	2	5	3	0	1	5	0	10	3	0	0
Hw.	1	7	4	0	5	4	1	9	1	0	0
Hn.	0	0	0	0	0	1	0	1	1	0	0
Al.	1	8	4	0	10	9	1	10	2	0	0
Ev.	1	0	1	0	3	1	1	2	0	0	0
Tt.	0	0	0	0	0	0	0	0	0	0	0
Ts.	0	0	0	0	0	0	0	0	0	0	0
Total	5	16	9	1	18	23	2	31	7	0	0

(ii) *Incidence of infection in* 21—25 *years of age group* (*Table XII*)—There were 39 persons in this series, out of which 35 were found carrying one or the other protozoal/ helminthic or both parasites.

Light infection of *E. histolytica* was found only in two persons. Light *grade infection of E coli* and *G. intestinalis* occurred in 11 cases. 8 with light, one each of heavy and serious grades of infection were found of *I. butschlii.*

Hook worm infection again predominated in this group.15 mild and one each of moderate, heavy and serious grade infection were found.*A lumbricoides* was next in importance of light (1), mild (9), and moderate (2) grades.

TABLE XII

SHOWING INCIDENCE OF VARIOUS PARASITES (PURE AND MIXED) IN 21—25 YEARS OF AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh.	0	2	2	0	2	0	0	0	2	0	0
Ec.	2	2	3	0	3	5	0	3	3	0	0
Ib.	2	3	2	0	4	3	1	1	2	0	0
En.	0	0	0	0	0	0	0	0	0	0	0
Gi.	2	3	4	0	1	7	0	4	3	0	0
Hw.	0	5	3	0	7	3	0	7	1	0	0
Hn.	0	0	1	0	0	0	1	0	0	0	0
Al.	0	3	1	0	4	7	0	4	0	0	0
Ev.	2	3	2	0	3	1	0	0	1	0	0
Tt.	0	0	0	0	0	0	0	0	0	0	0
Ts.	0	0	0	0	0	0	0	0	0	0	0
Total	2	11	10	0	11	18	2	12	5	0	0

(*iii*) *Incidence of infection in* 26—30 *years age group* (*Table XIII*)—In this series 19 out of 24 were found infected either with protozoa/helminths or both.

Protozoan parasites were encountered in 9 persons. Light infection of *E. histolytica* occurred only in one case. Mild infection of *E. coli* occurred in two cases. *I. butschlii* was found in 2 persons. 4 light and a mild infection of *giardiasis* was also encountered.

16 persons in this series were found carrying helminthic parasites, Hook-worm infection of mild grade was recorded in 11 cases and in one person however, the incidence was of moderate nature. Mild grade infection of *ascariasis* was found in 5 persons, in one the infection was of moderate type. Mild infection of seat worm occurred in two persons . Mild infection of *H. nana* was detected only in one person.

TABLE XIII

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES (PURE AND MIXED INFECTIONS) IN 26—30 YEARS OF AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh.	**0**	0	1	0	0	0	0	0	0	0	0
Ec.	0		0	0	0	2	0	0	0	0	0
Ib.	1	0	**1**	0	0	0	0	0	0	0	0
En.	0	0	0	**0**	0	0	0	0	0	0	0
Gi.	0	0	0	0		3	1	2	0	0	0
Hw.	0	2	0	0	3	**5**	0	3	0	0	0
Hn.	0	0	0	0	1	0	**0**	1	0	0	0
Al.	0	0	0	0	2	3	1	**1**	1	0	0
Ev.	0	0	0	0	0	0	0	1	**1**	0	0
Tt.	0	0	0	0	0	0	0	0	0		0
Ts.	0	0	0	0	0	0	0	0	0	0	
Total	1	2	2	0	5	12	1	6	2	0	0

(iv) Incidence of infection in 31—35 *years age group* (*Table XIV*)—It was a small series of 7 person, all infected with protozoal/helminthic or both parasites.

Three persons had protozoan parasites. *E. histolytica* was conspicuous by its absence in this series. One mild case, each of *E. coli*, *I. butschlii* and *G. intestinalis*, was, however, encountered.

Helminthic parasites occurred in 6 persons. Four had hook worm, out of which 3 had mild grade and one was heavily infected. Mild infection of *A. lumbricoides* was encountered in only one person. 4 seat worm infections, one with mild and 3 with moderate infections, were also encountered.

A mild infection of *T. saginata* was discovered only in this series. It seems very possible that this parasite has a greater affinity for middle age group.

TABLE XIV

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES (PURE AND MIXED INFECTIONS) IN 31—35 YEARS OF AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh.	0	0	0	0	0	0	0	0	0	0	0
Ec.	0	0	0	0	0	1	0	0	0	0	0
Ib.	0	0	0	0	0	1	0	1	1	0	1
En.	0	0	0	0	0	0	0	0	0	0	0
Gi	0	0	0	0	1	0	0	0	0	0	0
Hw.	0	1	1	0	0	1	0	1	2	0	1
Hn.	0	0	0	0	0	0	0	0	0	0	1
Al.	0	0	1	0	0	1	0	0	1	0	1
Ev.	0	0	1	0	0	2	0	1	2	0	1
Tt.	0	0	0	0	0	0	0	0	0	0	0
Ts.	0	0	1	0	0	1	0	1	1	0	0
	0	1	1	0	1	4	0	1	4	0	0

(*v*) *Incidence of infection in* 36—40 *years age group* (*Table XV*)—Out of 6 persons in this group 5 persons were found having protozoan/helminthic or both parasites.

Out of 4 persons carrying protozoan parasites, 2 had mild grade infection of *E. coli*. Very light infection of *G. intestinalis* was recorded from 3 persons.

There was a preponderance of mild hook-worm infection in 3 persons out of 5 in this series. Mild infection of *A. lumbricoides* was encountered in 2 persons. One light and a mild grade infection of *E. vermicularis* was also detected. Light infection of *H. nana* occurred in one person.

TABLE XV

TABLE XV SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES (PURE AND MIXED INFECTIONS) IN 36—40 YEARS AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh.	**0**	0	0	0	0	0	0	0	0	0	0
Ec.	0	**0**	0	0	1	1	1	1	2	0	0
Ib.	0	0	**0**	0	0	0	0	0	0	0	0
En.	0	0	0	**0**	0	0	0	0	0	0	0
Gi.	0	1	0	0	**0**	2	1	1	1	0	0
Hw.	0	1	0	0	2	**0**	0	0	1	0	0
Hn.	0	1	0	0	1	0	**0**	1	1	0	0
Al.	0	1	0	0	1	0	1	**1**	1	0	0
Ev.	0	2	0	0	1	1	1	1	**0**	0	0
Tt.	0	0	0	0	0	0	0	0	0	**0**	0
Ts.	0	0	0	0	0	0	0	0	0	0	**0**
	0	2	0	0	3	3	1	2	2	0	0

(vi) Incidence of infection 41—45 years of age group (Table XVI)—Out of 6 persons in this series, only 4 had infection of either protozoa/helminthic or both. Protozoan parasites occurred in two cases. Both had the common colon amoeba. Out of helminthic parasities, only hookworm infection was detected in 3 cases.

TABLE XVI

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITIES (PURE AND MIXED INFECTIONS IN 41—45 YEARS AGE GROUP

	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	As.	Ev.	Tt.	Ts.
Eh.	**0**	0	0	0	0	0	0	0	0	0	0
Ec.	0	1	0	0	0	1	0	0	0	0	0
Ib.	0	0	**0**	0	0	0	0	0	0	0	0
En.	0	0	0	**0**	0	0	0	0	0	0	0
Gi.	0	0	0	0	**0**	0	0	0	0	0	0
Hw.	0	1	0	0	0	2	0	0	0	0	0
Hn.	9	0	0	0	0	0	**0**	0	0	0	0
As.	0	0	0	0	0	0	0	**0**	0	0	0
Ev.	0	0	0	0	0	0	0	0	**0**	0	0
Tt.	0	0	0	0	0	0	0	0	0		0
Ts.	0	0	0	0	0	0	0	0	0		0
Total	0		0	0	0	3	0	0	0	0	0

(*vii*) *Incidence of infection in* 46—65 *years as age group*—(*Table XVII*) Out of 15 persons in this series, 14 had either protozoan/helminthic or both infections.

Protozoan infection occurred in 11 cases. No infection of *E. histolytica* could be detected. Light to mild grade infection of *E. coli* and *I. butschlii* was found in two cases each. The preponderance of *G. intestinalis* infection in this series was rather unusual. The infection was of light grade in all the cases.

Helminthic infections were detected in 9 cases. When compared with other age group series and in the light of protozoan infection, this number is far too less. May we suspect that helminthic parasites have lesser affinity for advanced age groups. Or these persons had little chances of contact with the source of infection. However, there was still preponderance of hook worm infection in this series also. 5 mild and a moderate infections were detected. Mild infection of *E. vermicularis* was found in 3 persons. *A. lumbricoides* occurred in two cases, in one it was of mild grade, while in the other it was a heavy infection. A light infection of *trichuiriasis* occurred in this series alone. Mild infection of *H. nana* was also detected in one person.

TABLE XVII

SHOWING INCIDENCE OF VARIOUS INTESTINAL PARASITES (PURE AND MIXED INFECTIONS IN 46—65 YEARS OF AGE GROUP.

	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh.	**0**	0	0	0	0	0	0	0	0	0	0
Ec.	0	**1**	0	0	0	0	0	0	1	0	0
Ib.	0	0	**2**	0	0	0	0	0	0	0	0
En.	0	0	0	**0**	0	0	0	0	0	0	0
Gi.	0	0	0	0	**2**	3	1	2	1	1	0
Hw.	0	0	0	0	3	**2**	1	0	1	1	0
Hn.	0	0	0	0	1	1	**0**	0	0	0	0
Al.	0	0	0	0	2	0	0	**0**	1	0	0
Ev.	0	1	**0**	0	1	1	0	1	**0**	0	0
Tt.	0	0	0	0	1	1	0	0	0	**0**	0
Ts.	0	0	0	0	0	0	0	0	0	0	**0**
	0	2	2	0	7	6	1	2	3	1	0

E. Single Parasite Infections (TABLE XVIII).

It was not uncommon to find a person with a solitary intestinal parasite. It will be interesting thing to be noted here, that the stools of all pure infection cases were examined several times before recording the results. Pure infection were more of helminthic parasites than of protozoan parasites. There were 18 pure infection cases of protozoan as against 38 of helminths in this series.

Pure infection case of *E. histolytica* was encountered once. There were more or less equal number of pure infection cases of *E. coli* (6), *I. butschlii* (5) and *G. inetestinalis* (6).

Out of 69 hook worm cases, 17 persons had only this parasite and out of 54 round worm cases 16 were pure (single) infection cases. Four cases of *E. vermicularis* and one case of *H. nana* were also pure infection cases.

TABLE XVIII

SHOWING SINGLE PARASITE INFECTIONS IN VARIOUS AGE GROUPS (IN YEARS).

Age group	Eh.	Ec.	Ib.	Gi.	Hw.	Hn.	Al.	Ev.	Total : % ages	Total examined infected
16—20 ..	1	2	..	1	4	..	10	..	18: 32·14	59/56
21—25 ..	..	2	2	1	3	1	4	1	14: 40·00	39/35
26—30 ..	..	..	1	1	5	..	1	1	9: 47·37	24/19
31—35 ..	..	..	..	1	1	..	..	2	4: 57·14	7/7
36—40 ..	..	..	..	..	..	..	1	..	1: 20·00	6/5
41—45 ..	..	1	..	..	2	..	..	..	3: 75·00	6/4
46—65 ..	..	1	2	2	2	..	..	..	7: 50·00	15/14
Total ..	1	6	5	6	17	1	16	4	56: 35·90	156: 140
Percentage ..	0·71	4·28	3·57	4·28	12·14	0·71	11·43	2·85	56: 35·90	156/140

F. Multiple Infections of Intestinal Parasites (TABLE XIX—X)

Out of 140 positive cases, there were 56 pure infections, 52 double infections.

24 triple infections, 3 quadruple and 5 quintuple infections. The relevant information is presented in Table XIX. The protozoan and helminthic parasites involved in various combinations are given in Table XX.

(*i*) Amongst the double-infection cases, in 5 cases the association was involved within protozoan parasites, in 32 cases protozoan were associated with helminths, while in 15 cases only helminthic parasites were associated.

(*ii*) There were 24 triple infection cases in this group. The association more or less predominated between two helminthic parasites and a protozoan parasite (usually between *G. intestinalis*) throughout the series. There were 6 cases in which the association was established between two protozoans and a helminth (usually hook-worm).

There was only one solitary case, however, where the association was found established between three protozoan parasites (*E. histolytica*, *I. butschlii* and *G. intestinalis*).

(*iii*) In the three quadruple infections, association in two cases was established between two protozoan and two helminths. In one case, however, round worm was associated with three protozoa. It was interesting to find *A. lumbricoides*, *G. intestinalis* and *E. coli* in all the three cases. Hook worm occurred in two and in one it was replaced by *E. histolytica*.

(*iv*) In 5 quintuple infections, hook worms are involved in two cases, round worms are involved in three cases, *E. vermicularis* appears in four cases while *T. saginata* and *H. nana* are involved once each. *G. intestinalis*, *I. butschlii* and *E. coli* were conspicuous in four of these cases. *E'. histolytica* occurred in two cases only. In two cases the association existed between one helminth (*E. rmicularis*) and four protozoas (*E. histolytica*, *E. coli*, *I. butschlii* and *G. intestinalis*), in one the association existed between two helminths (hook-worms and Round-worm) and three protozoas (*E. coli*, *I. butschlii* and *G. intestinalis*), in one case two protozoas (*E. coli* and *G. intestinalis*) and three helminths viz., Hook-worms, Round-worms, and Seat-worms had joind hands in a war against the victim, in another case the

association existed between four helminths viz., Hook-worms, Round-worms, Seat-worms and Beef tape-worms and one protozoa—*I. butschlii*.

TABLE XIX

SHOWING NUMBER OF PURE, DOUBLE, TRIPLE, QUARDRUPLE, AND QUINTUPLE INFECTIONS IN DIFFERENT AGE GROUPS (IN YEARS).

Age groups (in years)	Total examined	Total positive	Single infection	Double infection	Triple infection	Quadruple infection	Quintuple infection
16—20 ..	59	56 (94·94)	18 (32·14)	24 42·86)	11 (19·64)	2 (3-57)	1 (1·79)
21—25 ..	39	35 (89·74)	14 (40·00	11 (31·43)	7 (20·00)	1 (2·86)	2 (5·71)
26—30 ..	24	19 (62·51)	9 (47·37)	8 (42·11)	2 (10·52)	.. ..	.. ..
31—35 ..	7	7 (100·00)	4 (47·14)	2 (28·57)	.. ..	.. ..	1 (14·29)
36—40 ..	6	5 (83·33)	1 (20·00)	2 (40·00)	1 (20·00)	.. ..	(20·00)
41—45 ..	6	4 (66·66)	3 (75·00)	1 25(·00)	.. ..	.. ..	.. ..
46—above ..	15	14 (93·33)	7 (50·00)	4 (28·57)	3 (21·43)	.. ..	.. ..
Total ..	156	140	56	52	24	3	5
Percentage infected (of the total examined).	..	89·75	35·90	32·70	15·40	1·90	3·85
Percentage infected (of the total positive).	..	100	40·00	36·48	17·14	2·15	4·28

TABLE

SHOWING THE PARASITES INVOLVED IN SINGLE, DOUBLE, TRIPLE AND

Type of infection	Age groups		
	16—20	21—25	26—30
Single	Eh. (1) Ec. (2), Gi. (1), Hw. (4), Al. (10),. (18:32·14)	Ec. (2), Ib. (2), Gi. (1), Hw. (3), Hn. (1), Al. (4), Ev. (1), (14:40·00)	Ib. (1), Gi(1), Hw. (5), Al. (1), Ev. (1), (9: 47·37)
Double	Ib. En. (1), Gi. Ec. (1), Gi. Ev. (2), Hw.Ib.(1),Hw. Al.(5), Hw. Ev. (1), Hw. Gi. (3), Hw. Ec. (3), Al.Ev. (1), Al. Ec. (2), Al. Gi. (3), Hn. Ev. (1). (24: 42·86)	Ec. Ib. (1), Ib. Gi. (1), Hw. Al. (2), Hw. Ec. (2), Hw. Gi. (2), Hw. Ib. (1), Hn. Ib. (1), Al. Ec. (1), (11:31·43)	Eh. Ib. (1), Hw. Al. (2), Hw. Gi. (2), Hw. Ec. (2), Al. Ev. (1), (8:42·11)
Triple	Eh. Ib. Gi. (1), Eh. Ib. Ev. (1), Eh. Ec. Hn. (1), Hw. Hn. Gi. (1), Hw. Gi. Al. (1), Hw. Ib. Ec. (1), Al. Ev. Gi. (4), Al. Ib. Ec. (1), (11:19·64)	Hw. Al. Gi. (2), Hw. Al. Ec. (2), Hw. Gi. Ev. (1), Hw. Gi. Ib. (1), Al. Gi. Ib. (1), (7:20·00)	Hw. Al. Gi. (1), Al. Hn. Gi. (1). (2:10·52)
Quadruple	Hw. Al. Gi, Ec. (1), Al. Gi. Ec. Eh. (1). (2:3·57)	Hw. Al. Gi. Ec. (1). (1:2·86)	.. ..
Quintuple	Hw. Al. Gi. Ec. Ib. (1). (1:1·74)	Eh. Ec. Ib. Gi. Ev. (2). (2:5·71)	.. ..
Total infected (with percentages)	56 (94·94)	35 (89·74)	19 (62·51)
Total examined ..	59	39	24

XX QUADRPULE AND UINTUPLE INFECTIONS (NUMBER OF CASES) IN DIFFERENT AGE GROUPS IN, (YEARS)

Age Groups			
31—35	36—40	41—45	46 and above
Gi. (1), Hw. (1), Ev. (2). (4:57·14)	Al. (1). (1:20·00)	Ec. (1), Hw. (2). (3:75·00)	Ec. (1), Ib. (2), Gi. (2), Hw. (2). (7:50·00).
Hw. Ec. (1), Hw. Ev. (1). (2:28·57)	Hw. Gi. (2). (2:40·00)	Hw. Ec. (1). (1:25·00)	Hw. Ev. (1), Hw. Gi. (1), Al. Gi. (1), Ev. Ec. (1). (4:28·50)
.. ..	Hw. Ev. Ec. (1). (1:20·00)	.. ..	Hw. Gi. Hn. (1), Hw. Gi. Tt. (1), Al. Gi. Ev. (1). (3:21·43)
.. .. ..	.. .. ..	.. .. ..	.. .. ..
Hw. Al. Ev. Ts. Ib. (1). (1:14·29)	Al. Ev. Hn. Gi. ä Ec. (1). (1:20·00)	.. .. ..	.. .. ..
7 (100·00) 7	5 (83·33) 6	4 (66·66) 6	14 (93·33) 15

G. Incidence of Intestinal Parasites according to the Locality (Table XXI)

The incidence of intestinal parasites varies with the cultural and aesthetic standards of the people. Our people are not conservative. They are readily co-operative and very enthusiastically take up any thing useful. As the saying goes, "Money makes the mare go,"* is terribly involved. The common man hasn't has enough resources and he hasn't has proper guidance either. Governmental agency formerly did not approach the common man as a guide but as a boss. The common man was not involved into any programme as a partner. The project was usually thrust upon him without giving him a chance to understand the planned undertaking. The programmes flopped because common man; for whose interest it was, avoided to be involved because of ignorance. Why do we have slums? Why do the ill-planned colonies still sprout in this country? Why do our people think that improvement of environmental sanitation is somebody's else job? We know the answer, but what we are going to do about it. We can only remind of *all help is for those who can help themselves.* The hatcheries of the intestinal parasites can stop functioning if there are missionaries to look after environmental sanitation.

Else where in this paper, we have endeavoured to give brief discriptions of the areas from where the conservancy staff of the City Corporation hails. Table XXI gives the distribution of the intestinal parasites in this staff with respect to their residence.

TABLE XXI

TABLE XXI SHOWING DISTRIBUTION OF INTESTINAL PARASITES ACCORDING TO THE RESIDENTIAL QUARTERS OF THE CONSERVANCY STAFF

Sectors	Eh.	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
1. Ravi Road ..	2	8	3	..	7	10	1	9	4	1	..
2. Badami Bagh ..	1	8	3	..	7	12	2	9	6	..	..
3. Misri Shah ..	2	1	3	..	4	2	..	4	1	..	..
4. Balal Ganj ..	..	3	3	..	8	8	2	15	1	..	..
5. Krishan Nagar ..	3	8	5	..	13	21	2	9	6	..	..
6. Pakki Thatti ..	..	3	4	1	1	11	..	4	2	..	1
7. Mozang, etc. ..	..	5	3	..	5	5	..	4	3	..	..
Total ..	8	36	24	1	45	69	7	54	23	1	1
Percentages of the total examined.	5·13	20·52	15·39	0·64	28·85	44·23	4·49	34·62	14·74	0·64	0·64
Percentages of the total infected.	5·72	23·71	17·14	0·71	32·14	49·29	5·00	38·57	16·43	0·71	0·71

Jedhi kothi dune, usde kamle wi siyane.

Highest number of cases of *E. histolytica* were recorded from Krishan Nagar, Ravi Road area and Misri Shah. No case of this parasite was encountered in those coming from Balal Ganj, Pakki Thatti and Mozang sector.

Highest number of the cases of *E. coli* were from Ravi Road area, Badami Bagh and Krishan Nagar sectors. Five cases were found from Mozang. Three cases of each were found from Balal Ganj, Pakki Thatti. Misri Shah gave us only one case.

The number of *I. butschlii* cases were the same from almost all the sectors except from Krishan Nagar and Pakki Thatti, which had 4 and 5 cases, respectively.

Thirteen cases of *giardiasis* were recorded from Krishan Nagar, Ravi Road and Badami Bagh gave 7 cases each, Bilal Ganj sector gave 8 cases, Mozang and Misri Shah gave us 5 and 4 cases, respectively, while Pakki Thatti gave only one case.

Hook-worm infection was highest in Krishan Nagar, it gave us 21 cases as against 12 from Badami Bagh, 11 from Pakki Thatti, 10 from Ravi Road and 8 from Balal Ganj. From Misri Shah and Mozang sectors there were 2 and 5 cases only.

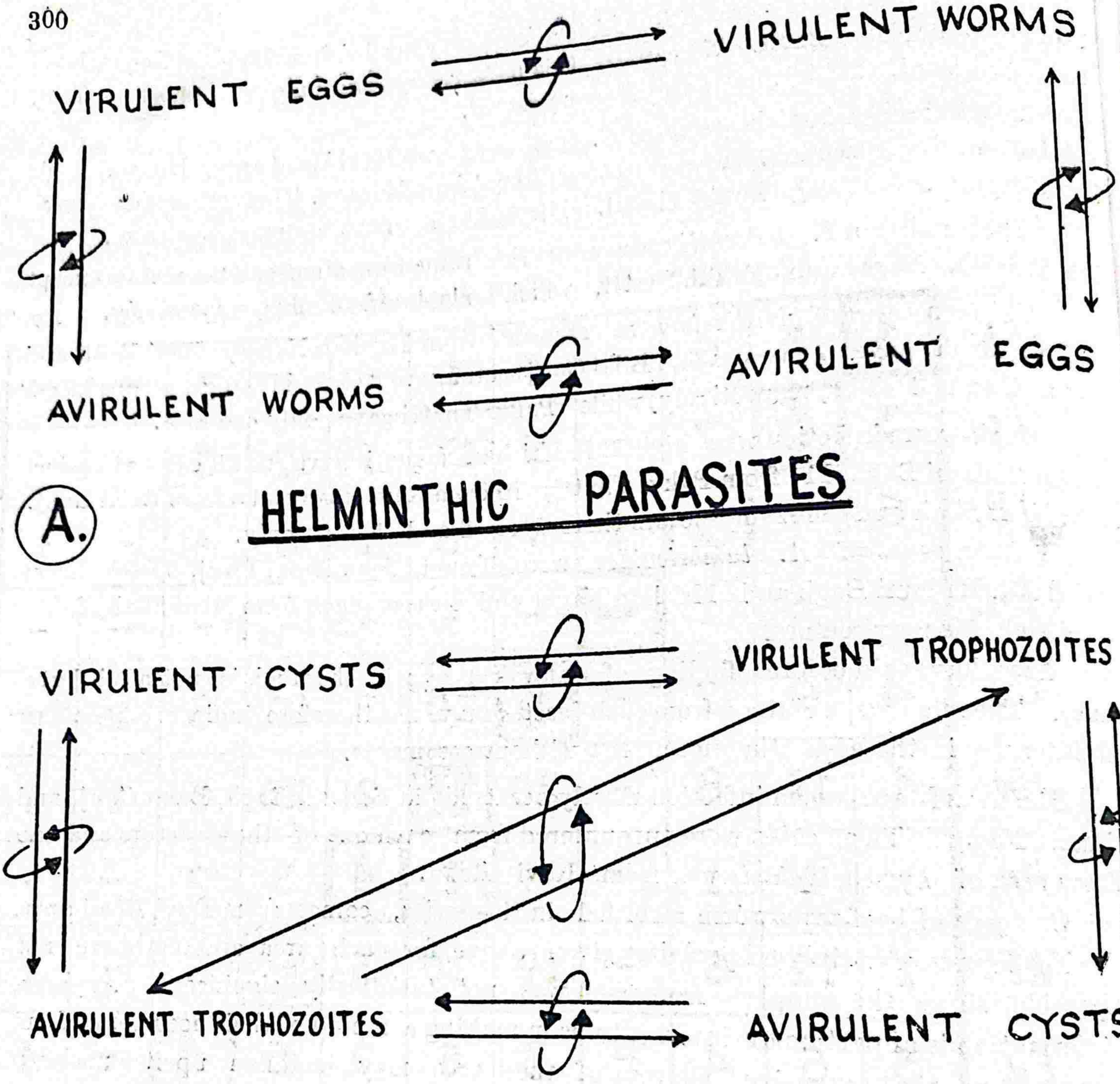
Fifteen cases of *A. lumbricoides* were found from Balal Ganj, 9 each from Ravi Road, Badami Bagh and Krishan Nagar and 4 cases each from Misri Shah, Pakki Thatti and Mozang sectors.

As we have said before, finding of seat worms eggs in the stools is not a rule but a chance. The figures obtained from each sector may not therefore, indicate endemicity and intensity in the area therefore, left without comments.

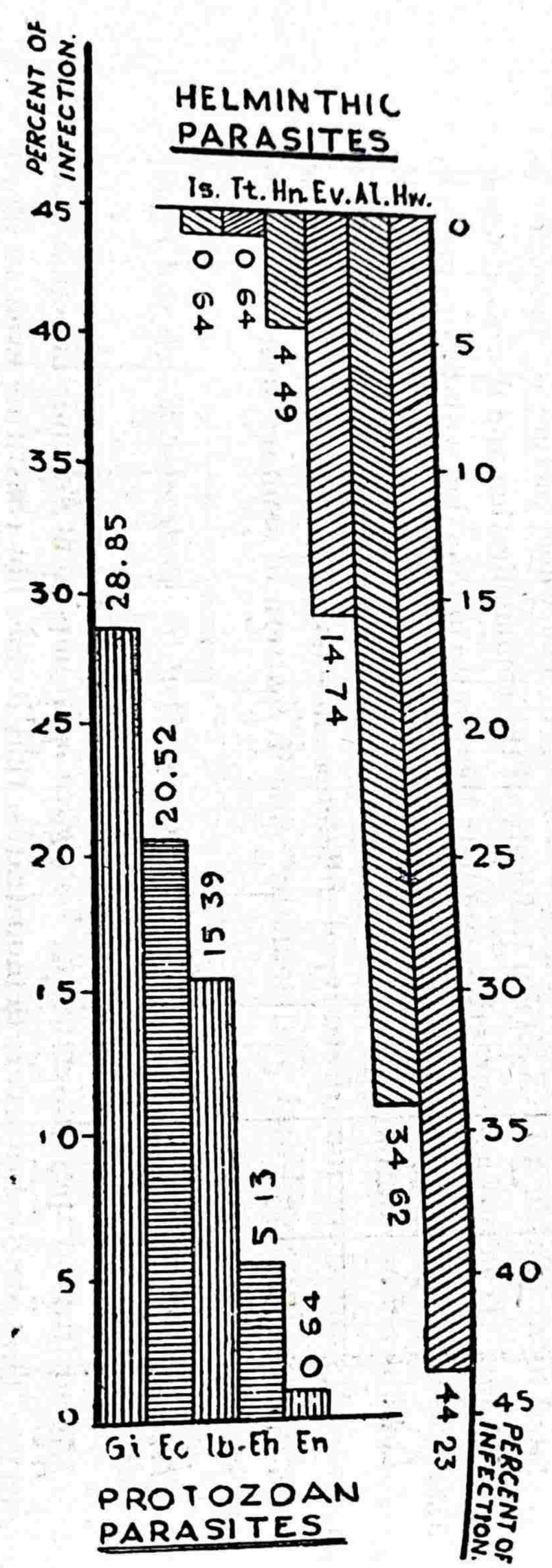
The dwarf-tape-worm infection mostly occurred in Badami Bagh, Balal Ganj and Krishan Nagar. Two cases were encountered from each one of these sectors and one solitary case of *hymenolepiasis* was from Ravi Road.

One case of beef-tape-worm recorded in this series, comes from Ravi Road area.

The figures of individual parasites given above, for each area and sector are not comparable since the sample from each was not statistically significant. Anyhow, the information is of particular interest to us in making a record of their occurrence from these areas, so that extensive surveys of these areas may be taken up at some later date and reliable approximations of the true incidence of the intestinal parasites are known.


VIRULENT EGGS
VIRULENT WORMS
AVIRULENT WORMS
AVIRULENT EGGS
A.
HELMINTHIC PARASITES
VIRULENT CYSTS
VIRULENT TROPHOZOITES
AVIRULENT TROPHOZOITES
AVIRULENT CYSTS
B.
PROTOZOAL PARASITES

	Eh	Ec.	Ib.	En.	Gi.	Hw.	Hn.	Al.	Ev.	Tt.	Ts.
Eh	1	3	6	-	4	1	-	1	3	-	-
Ec	3	6	7	-	9	17	1	12	6	-	-
Ib.	6	7	5	1	7	8	1	6	4	-	1
En.	-	-	1	-	-	-	-	-	-	-	-
Gi.	4	9	7	-	6	20	3	19	8	1	-
Hw.	1	17	8	-	20	17	2	20	6	1	1
Hn.	-	1	1	-	3	2	1	3	2	-	-
Al	1	12	6	-	19	20	3	16	6	-	1
Ev.	3	6	4	-	8	6	2	6	4	-	1
Tt.	-	-	-	-	1	1	-	-	-	-	-
Ts.	-	-	1	-	-	1	-	1	1	-	-


HELMINTHIC PARASITES
Is. Tt. Hn. Ev. Al. Hw.
0 64
0 64
4 49
14.74
34 62
44 23
PERCENT OF INFECTION.
0
5
10
15
20
25
30
35
40
45
28.85
20.52
15 39
5 13
4 90
Gi Ec Ib-Eh En
PROTOZOAN PARASITES

TIS HARD TO SAY IF GREATER WANT TO SKILL APPEAR IN WRITING OR IN JUDGING ILL:
(POPE)

1. Data are presented on the intestinal protozoan and helminthic parasites of a sample of conservancy staff of the City Corporation of Lahore. 156 persons were examined during the period commencing from February 16 and ending on March 28, 1963. The conservancy staff consists of economically poor section of our population. Opportunities of exposure and re-exposure in this section are relatively abundant. They live under slum, crowded environments, eat un-wholesome food and soiled vegetables, drink opaque or bad water which has direct or open communication with effluent, usually use unclean utensils and have filthy hands. Human faeces is always on their clothes. They are discharging the duties of a scavanger since 2000 years or more before Christ.

2. This data is based on the examination of one single, usually formed stool, from each case. The examinations were made both by direct and concentration methods.

3. The data was analysed by age groups. The whole picture is presented in tabulated form (Table VII-XXI) in the body of the communication and discussed briefly *ad seriatum*. It is not attempted to compare these rates of infection with figures given by other workers from else where. These figures are derived from a different type of population and are not comparable with figures collected from any other kind of population because of short duration of study, of difference in examination technique, of variance in age groups involved and of diagonally opposite pattern of sanitary standards of the homogenous community.

4. From the data before us, it is concluded that *G. intestinalis*, *E. coli* and *I. butschlii* are common protozoan infections.

According to the clinicians, *E. histolytica* is an important endemic infection of our country. This study however, demonstrates, that it does not constitute even an important fraction of the total parasitic infections as far as the conservancy staff of city corporation is concerned. We did not do mensuration, therefore, the race of *E. histolytica* involved could not be ascertained. We also did not make any permanent slides and ascertain the presence of *E. hartmanni*. This appears to be a serious lecunae in these studies. We would very much like to find answers to these question in our future surveys.

The rate of infection with *E. coli* was high. It was more or less four times the infection of *E. histolytica*, amounting to 20·5 per cent in persons examined.

The infection rate with *E. nana* was unexpectedly very low in this community.

I. butschlii was found less common than *E. coli* and *G. intestinalis* and none of the positive cases complained of any abdominal distress.

G. intestinalis is primarily an infection of children. In the adults examined, its incidence was unexpectedly and greatly in excess, giving cause to suspect it an endemic infection in Lahore. It was found in about 29 per cent persons examined.

5. The eggs of the helminthic parasites, viz., *A. duodenalis*/*N. americanus*, *A. lumbricoides* and *E. vermicualris* were encountered very frequently and it was concluded that

worm burden forms serious public health problem of this community. The hook-worm infection was found in more or less 45 per cent persons examined. From a study of socio-economic level of this community, their cultural pattern and the physical environments in which they live, one may say that high rate of hook-worm infection is not a surprise. It will be fine if these findings persuade some one to carry out studies on the nutritional deficiencies of this group of people.

Both epidemiologically and clinically, *A. lumbricoides* is probably one of the most important parasites of Lahorites. There are known permanent foci in congested localities. Greater incidence may be expected in areas having suburban conditions with unsati factory sanitary provision for the disposal of human excreta and improper water supply. Facilities for propogation are sure and sound. Effective and uninterrupted transmission takes place from door yards, courtyards, contaminated water supply, dust storms, flies and raw vegetables. Promiscuous defaecating habits have made extensive "seed-beds" of *ascaris* worms around the town. The incidence was found to be 34·62 per cent. It appears a very conservative figure. A detailed consideration of those harbouring males, young females, quiescent and spent females of the worm would probably give us significantly increased figures.

E. vermicularis is notorious for group infections. Huddling together and congestion helps in its propogation not within a family but also within a community. It is an infection which cannot be fully estimated from coprological examination. An incidence of 14·74 per cent, based on stool examination alone indicates that it is very widely spread parasite.

6. Choice of laboratory method, out of many methods available, is an individual decision. Perfection comes with practice and experience. No comments are, therefore, added on laboratory proceedings.

7. Essential clinical features, associated with different parasitic infections, have always remained shortcuts to the identification. Although signs and symptoms have no diagnostic value, yet in the absence of bedside identification of parasites, remained an advantage. A summary, of signs and symptoms in volved with different parasites, therefore, has been given in the body of the communication for ready recknoning.

ACKNOWLEDGEMENT

Our thanks are due to Dr. Mazhar Hussain, M. B., B. S., D. P.H., W. P.H. S.(I), Chief Medical Officer of Health, City Corporation of Lahore, for unstinted help and cooperation rendered during the collection of the stool samples from the conservancy staff, Lahore.

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ETHIONAMIDE IN THE TREATMENT OF CHRONIC PULMONARY TUBERCULOSIS

By

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With the appearance of resistant strains of tubercle bacilli against the standard chemotherapeutic drugs, *i.e.*, streptomycin, P.A.S. and isoniazid, a need has always been felt for some new anti-tuberculous drugs which could be effective and do not show cross resistance with other agents.

Encouraging results were reported with the use of ethionamide in vitro against isoniazid resistant strains of tubercle bacilli by Rist and others (1958) and later by Brouet and others (1959). Since then, many more reports have appeared regarding the efficacy of this new oral anti-tuberculous drug.

This paper reports the results of seven patients treated with ethionamide in combination with a standard drug. All these patients had gross lesions with multiple cavitation, and their tubercle bacilli had become resistant to two out of the three standard drugs. These patients had been treated for long time previously with standard drugs but their sputum still showed tubercle bacilli. Therefore, each of these patients was given ethionamide plus one of the three standard drugs to which the tubercle bacilli were still sensitive. The supply of the drug being limited, only seven cases could be included in this series.

Clinical material—Each of these cases was subjected to sensitivity tests against streptomyein, P. A. S., isoniazid and ethionamide. They all were sensitive to ethionamide but each one of them was sensitive to only one of the three standard drugs as mentioned above and therefore their combinations were as follows :—

1. M.A. Females, age 27 years—Ethionamide 1 Gm+Isoniazid 300. mgms. daily.
2. A.W.-Male, age 50 years—Ethionamide I Gm. +Isoniazid 300 mgms. daily.
3. G. B.-Male, age 45 years—Ethionamide I Gm.+Isoniazid 300 mgms. daily.
4. S. A.-Female, age 28 years—Ethionamide I Gm +P.A.S. 12 Gms. daily.
5. Z.A.-Male, age 45 years—Ethionamide I Gm+P.A.S. 12 Gms daily.

6. A.M.-Male, age 35 years—Ethionamide IGm+Isoniazid 300 mgm. daily.

7. F.K.—Male, age 38—Ethionamide I Gm+Streptomycin I Gm. Daily.

Sensitivity testing—Sensitivity tests were done in all the seven cases before the treatment was started. The techinque used was as follows.

Growths from primary cultures were emulsified in 0.5 ml sterile distilled water. Using a standard loop of 3 mm. diameter, one loopful of emulsion was spread over the Lowenstein—Jensen medium slopes containing respectively 5, 10, 20, 40, 80, 160 micrograms/ml. ethionamide. The results were reported as a resistant ratio. H37 RV and other sensitive strains are commonly inhibited by 10 mgm per ml. This observation is supported by the results of sensitivity of more than 75 strains of tubercle bacilli done in this laboratory on ethionamide. In none of the cases included in this trial and even in others not included in the trial, the resistence ratio was more than one.

Duration of treatment—All these cases were in-patients at the time of commencement of trial. One of these left the hospital against medical advice but had developed toxic symptoms before leaving the Hospital. She was reported to have died a few days later.The remaining six cases were hospitalized for periods ranging from four to eight month and were followed up later in the clinic during the course of the treatment and for some time afterwards.

Dosage—Treatment was started with 250 mgm of Ethionamide morning and evening; and the dose was increased to 500 mgm. twice daily within a week according to the tolerance of the drug by the individual patient. The companion drug was either stremptomycin I gram daily or P. A. S. 4 grams thrice a day or isoniazid 150 mgm twice daily, depending upon the sensitivity result as already discussed.

Radiographic extent of disease—Before the start of treatment radiographic assessment was made by a postero-anterior film of the chest. All cases showed moderately advanced disease with multiple cavitation. One case had unilateral disease while all others had bilateral disease.

Laboratory Tests—Besides culture and sensitivity tests for tubercle bacilli done on monthly basis, as described earlier, weekly examinations of urine and direct smear sputum microscopy were carried out regularly. Blood urea estimation, B.S.R., total and differential white cell count and X-Ray examination of chest were done every month.

Toxic reactions—The main complications encountered were malaise, nausea,vomiting,anorexia, upper abdominal pain and skin reactions. One patient also developed pain in the small joints of hands and feet, The complications are listed in table I.

TABLE I

(SHOWING COMPLICATIONS ENCOUNTERED WITH ETHIONAMIDE)

Complications	Case No. 1	2	3	4	5	6	7
1. Malaise ..	+	+	..	+	..	..	+
2. Nausea ..	+	..	..	+	..	..	+
3. Vomiting ..	..	..	..	+	..	..	+
4. Anorexia ..	+	..	..	+	..	..	..
5. Upper abdominal pain	+	+	..	..	..	..	..
6. Peripheral sensory changes.	..	..	..	..	..	..	+
7. Skin reactions ..	+	+	..	..	..	..	+
8. Joint pains ..	..	..	..	..	..	+	+

Case Summaries—

Case 1—M.A. female 27 years. Extensive fibro-caseous cavitary disease of three years duration. General condition poor. Sputum culture showed tubercle bacilli resistant to streptomycin and P. A. S. Treated with ethionamide 500 mgm. twice daily and isoniazid 150 mgm. twice daily. Gastro-intestinal symptoms appeared as metallic taste in the mouth, nausea and abdominal discomfort. There was generalised itching, and burning sensation in the body. These toxic manifestations disappeared with stoppage of drug. Drug restarted and above symptoms reappeared. Drug continued for 5 months. No change in general condition or X-ray chest. However sputum converted into negative after 2 months treatment.

Case No. 2—A. W., male, 50 years, bilateral fibro-caseous cavitary disease of $2\frac{1}{2}$ years duration. Treated with ethionamide and isoniazid. He developed mild symptoms e.g., metallic taste, abdominal discomfort, itching and burning sensation but later tolerated the drug well. Very slight improvement in general condition and radiologically. Sputum converted negative in 2 months and remained so thereafter.

Case 3—G. B., male 45 years. Unilateral cavitary disease of 3 years duration. Treatment with ethionamide and isoniazid. No toxic symptoms noted. Slight improvement in general condition. No significant change in X-ray,. Sputum convertion did not take place uptil 6 months of treatment.

Case 4—S. A., female, 28 years. Extensive cavitating bilateral disease of $3\frac{1}{2}$ years duration. General condition was poor. Sputum culture showed tubercle bacilli resistant to streptomycin and isoniazid. Treatment started with ethionamide and P. A. S. She developed severe nausea and vomiting and drug had to be stopped

after a week. She refused to restart the drug and left the hospital.

Case 5—Z. A. male, 45 years, Fibro-caseous disease of 2 years duration. Tubercle bacilli resistant to streptomycin and isoniazid. Treated with ethionamide and P. A. S. He developed mild gastro-intestinal symptoms in the beginning, otherwise tolerated the drug well. No change radiologically but slight improvement in general condition. Sputum convertion after two months treatment.

Case 6—A. M., male, 35 years. Bilateral cavitating disease of chronic nature and 3 years duration. Had extensive treatment at home. Treated with ethionamide and isoniazid in dosage described earlier.He had mild gastro-intestinal symptoms and skin reactions such as tingling and sensation of pins and pricks but drugs continued. No radiological change noticed but there was slight improvement in his general condition. Sputum was converted negative after two months treatment.

Case 7—F. K., male 38 years. Fibrocaseous disease right lung with advanced multiple cavitated lesion in the left lung of long duration. Resistant to P. A. S. and isoniazid. Treated with ethionamide 500 mgm. twice daily and streptomycin 1 gram daily. Had mild gastro-intestinal symptoms such as metallic taste, nausea, loose motions, itching and burning sensation in whole body. After 5 months treatment he developed pains in smaller joints of hands and feet which subsided on stoppage of drug. Slight improvement clinically and radiologically. Sputum converted into negative after 4 months.

Discussion

Whatever favourable results were obtained, can be attributed to ethionamide in combination with one of the standard drugs. Since almost all the standard drugs had been given to these cases for a long period prior to this trial, therefore it is not difficult to conclude that ethionamide must have played a major role in the changes noticed in this trial. The result in one case is inconclusive, because she left the ward and died soon after. Out of the remaining six cases sputum became negative in five of them and remained so for periods varying from five months to ten months depending upon their follow up.

The clinical, radiologizal and bactenological changes are given in Table II.

TABLE II

Showing Changes in general condition, chest-x-ray and sputum conversion

Case No.	General condition	Radiological changes	Sputum conversion
1. M. A. ..	No improvement ..	No change ..	Yes.
2. A. W. ..	Slight improvement ..	Slight improvement ..	Yes.
3. G. B. ..	Slight improvement ..	No change ..	No.
5. Z. A. ..	Slight improvement ..	No change ..	Yes.
6. A. M. ..	Slight improvement ..	No change ..	Yes.
7. F. K. ..	Slight improvement ..	Slight improvement ..	Yes.

These changes are not much encouraging. Bearing in mind, the chronic nature of disease and extensive radiological evidence not much changes could be expected in these cases. However, the results indicate that the drug has a definite value in bacteriological convertion of sputum which should be considered as a great helping factor in chronic cases.

SUMMARY

Seven patients with chronic active cavitating pulmonary tuberculosis were treated with ethionamide in combination with one of the standard drug. In one case result could not be ascertained. Sputum conversion was obtained in five, out of six cases. General improvement of slight degree was noticed in five cases. The drug was reasonably tolerated with minor toxic symptoms except in one case. Though a small series, but conclusion can be reached that ethionamide may be valuable in sputum conversion.

Our thanks are due to Messrs. MAY AND BAKER (Pakistan) Limited for the supply of Ethionamide (Trescatyl).

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IMMUNIZATION IN CHILDREN

by

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In developing countries like our own, many a million still fall a prey to the devastations of diseases which could well be prevented by active measures of immunization. These measures have already led to virtual disappearance of terrible afflictions like smallpox, cholera, diphtheria and tetanus in advanced countries and have caused a remarkable fall in the incidenceof many other diseases like Poliomyelitis, Whooping cough and Tuberculosis, etc.

The only way towards progress lies in early control of these diseases by prompt measures. This becomes a very vitally important issue when it is realised that how quickly various diseases like Smallpox etc., can be transmitted across the Globe due to facilities of modern travelling. Most effective measures to achieve such an ideal would be to prevent the diseases by active immunization at an early age or halter it by passive immunization soon after its onset.

Immunization in children can be described under two headings (1) Active Immunization, (2) Passive Immunization.

1. ACTIVE IMMUNIZATION

(*a*) *Diphtheria*—At least 70 per cent of the population must be protected to ensure control of diseases by herd immunity. All currently available antigens are satisfactory, but alum containing toxoids such as A.P.T. and P.T.A.P. are no longer used in view of the reports that they are more likely than non-absorbed preparations to provoke paralysis in the injected limbs of the patients who contract Poliomyelitis in the ensuing month. (Medical Research Council, 1956)

When Diphtheria immunization is being given separately it is now recommended that two injections of 1 ml. formol toxoid be given not less tha n one month apart at 8-9 months of age with reinforcing doses of 0.5 ml. at 10 years and 0.25 ml. at 15 years. (Lancet 1957). Many health authorities have found that these separate antigens are impracticable under present day conditions.

If Diphtheria is present in the community and rapid primary immunization is required, three injections of 1 ml. fluid Diphtheria toxoid should be given at 10 days interval. For children previously immunized a single reinforcing dose as small as 0 2 ml. fluid formol toxoid will raise the anti body titre to a safe level in a few days. Large doses may give rise to re-actions in adolescents and adults, though young children tolerate formal toxoid well; it it is far less irritant than A. P. T. and P. T. A. P.

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The importance of susceptibility test *i. e. Schik Test* should not be forgotten. Schick test is a biological test to find out if a person has already developed his or her own Diphtheria anti-toxin and so is no longer susceptible to the disease. It consists of an intradermal injection of Schick toxin. The dose is 0.2 ml. (1 Schick dose) inoculated intradermally into left arm. 0.2 cc heated toxin (control) is inoculated similarly into right arm. Read at 48 hours, and 5 days.

Positive—Redness and swelling in test arm only.

Negative—No reaction.

Schick positive and swab negative cases should be considered susceptible to the disease and for such cases immunization is essentially required.

(*b*) *Triple Antigen Immunization Against Diphtheria Pertus,is and Tetanus*—1 cc. triple antigen by deep I/C injection at intervals of at least one month beginning at the age of two months or soon after is well recommended schedule. In exceptional circumstances, the first interval may be extended to three and the second to six months without restarting the course. A booster injection of triple antigen is advised at 15-18 months of age.

Risk from *whooping cough* is greatest in the 1st six months of life. At this time the risk from Diphtheria is negligible. For the first few months babies of immune mothers may continue to carry inherited Diphtheria anti-toxin, which interferes with response to immunization. The anti-toxin is excreted at the rate of about 15% per week, so that Diphtheria inoculations should theoratically be delayed until it has fallen below 0·04 units per ml. at 4·5 months.

Inoculations rather earlier can, however, be considered with virtual disappearance of the disease abroad, few mothers have high anti toxin levels to pass on to their offsprings many investigators have found that it is possible to immunize the great majority of infants between 2 and 5 months and an early booster injection will protect them all.

(*c*) *Tetanus*—Primary vaccination against tetanus is generally combined with vaccination against enteric fever(for adults) and with vaccination against Diphtheria (for childeren). In exceptional circumstances however, it may be carried out separately by injecting tetanus toxoid (T. T.) simultaneously. In such cases T. T containing 20 Lf. doses per ml. is usually used in the following dosage irrespective of age and sex.

1st Injection 0·5 ml. ..	2nd Injection 0.5 ml. ..	3rd Injection 0·5 ml
	6—12 weeks after 1st injection ..	6—12 month after 2nd injection.

Maintenance of Immunity—Except for the first two maintenance doses for children for which D.T. will normally be given, uncombined tetanus toxoid will be used for maintaining immunity to tetanus. Under normal conditions a maintenance dose of 0.5 ml. is given by subcutaneous injection every five years.

(*d*) *Enteric Group of Fevers*—Infants under one year old will not be inoculated against Enteric. Children of from one to 12 years inclusive will be inoculated subcutaneously with diluted typhoid paraty phoid vaccine (diluted T. A. B.). Diluted vaccine contains 1000 millions killed organisms (400 million Salmonella-typhi, 300 millions Salmonella para typhi and 300 millions Salmonella para typhi B per, ml.). Intradermal injection of children is not easy and the advisibility of using that route for them depends on a future assessment of the degree of liability to skin sensatization by it. The first and third doses for primary vaccination and all subsequent doses are 0·2 ml. The second dose for primary vaccination will generally be 0.4 ml. though for undersized children, or following any thing more than a mild reaction through the first dose, the second dose also should not be greater than 0.2 ml.

(Alcoholised vaccine is no longer used). Intramuscular route should be avoided.

Degree, duration and maintenance of immunity—Inoculation does not give full protection agianst enteric and though case incidence should be lessened, overt infections may still occur in those who are inoculated. When this happens the severity of the disease is not likely to be mitigated by the inoculation. It is hard to assess the duration of immunity following injections of T. A. B. vaccine. Protection is assumed to be maintained for one year and basal immunity to exist for three years after primary immunization or a maintenance dose. Those at risk are given annual doses to preserve protection and those not at risk are given doses every three years to maintain basal immunity.

(e) *Cholera*—Babies under one year are not normally immunized against cholera as the risks of contracting this infection are slight at this age. For primary vaccination two doses of vaccine are injected simultaneously at an interval of 7—28 days (preferably the interval should be 14—28 days though as one of only 7 days is undesirably short and should be avoided except in emergency). Adults and children over five years should be given 0·5 ml, as first dose and 1·0 ml. as second dose Children of 1—5 years should be given 0·25 ml. as first dose and 0·5 ml. as second dose. Immunity duration is short lived and maintenance doses are needed every six months for those at risk.

(f) *B.C.G. Vaccination (Immunization against T.B.)*—B.C.G. vaccination of children can be considered under three categories :—

I. New born infants of Tuberculous mothers

(i) *No contact with mother after birth*—A new born infant of a tuberculous mother should be removed from its mother at birth. No Heaf or other Tuberculin test is then needed , and the infant should be vaccinated with B.C.G. on the fourth or fifth day after birth. The baby should be kept segregated from the mother for a further six weeks after which a Heaf test will be carried out. If this is positive the child may be returned to the mother who should in the mean time has been told how to protect the baby from infection. Annual follow up Heaf tests are necessary. If the post-vaccination test is negative separation from the mother is continued for a further three weeks before a second Heaf test is made. If this is positive the child is returned to mother. If negative the baby is revaccinated, further action being as after the first vaccination.

(ii) *Contact between baby and mother after birth*—If there has been any contact between mother and infant after birth the child is segregated from mother for six weeks. A Heaf test is then carried out and the child vaccinated if this is negative. Action after vaccination is as in (i) above.

II. Older children who are contact cases

Action required is as for infants who have been in contact with tuberculous mothers except that :

(i) a Heaf test is done before the prevaccination segregation.

(ii) segregation must be supervised but need not

The Heaf test may be carried out at any age but which is most suitable and the age at which vaccination of negative reactors is required depend upon prevailing circumstances. When it is known that the tuberculin positive rate in children in a locality is high (30 per cent or more) children of such a locality should be Heaf tested at an age (*e.g.* 3—5 years) when they may be exposed outside the home and be vaccinated forthwith if tuberculin negative. On the other hand when the tuberculin positive rate among children under five is very low, B.C.G., vaccination is not at once necessary even if the Heaf test has been carried out at this earlier age and is negative. Vaccination interferes with the future diagnostic value of tuberculin tests and may well be deferred until the child is ten years old; when, of course, B.C.G. must still not be given without a repetition of Heaf test, with a negative result.

(*g*) *Poliomyelitis*—This is not legarlly required but is strongly recommended when it is available. It should precede smallpox vaccination by at least 2 weeks (and yellow-fever preferrably by 3—4 weeks).

It is paradoxical that the public health measures which have done so much to reduce or eliminate other infective diseases should have increased the risk of parlytic poliomyelitis. In communities living under primitive conditions infant inherit protection from their mothers under its cover they begin to acquire a natural immunity so that during the first few years of life most of them develope antibodies to all the three virus types. Paralysis is very rare at any age. Gelf and et al. (1957) studying a mixed community in a highly endemic area in Louisiana, found that Polio-viruses of all the three types were circulating continually, the transmission being regulated by seasonal limitation of contact and by personal hygiene. In this area nearly three-quarters of the children were immune by school age, the greatest spread of infection being among children aged 2-5, from whom it extended to other members of the family. In the most advanced society virus dissemination is reduced and exposure is delayed. For reasons not fully understood., this creates an explosive epidemiological situation and greatly increases the chances of any infection that does occur being paralytic or even fatal.

There is a convincing evidence from large scale trials in America, Canada and Britain that immunization with Salk type vaccine is 70 per cent effective (Med. Research Council 1957 B) but like other vaccines it does not provide complete protection. Cases of poliomyelitis have been reported in triple vaccinated subjects.

Primary Vaccination—Primary vaccination consists of 4 doses each of 1.0 ml. injected intramuscularly into the deltoid (usually of the left arm). The interval between the first and the second dose should be at least 3 weeks and the third for at least 6 months These intervals may, if necessary, be extended to 3 and 18 months respectively. A fourth dose is given 6 to 12 months after the third.

Maintenance Doses—Following the fourth dose further maintenance doses are probably required every five years.

Oral Route Administration of Vaccine—Living attenuated Poliomyelitis vaccine has been prepared recently and successfully tried in Russia. It is given by mouth and the Oral Route Administration provides certain additional advantages. It enables the vaccine to act on the intestinal mucosa so possibly to render the mucosal cells resistent to infection as well as nearly preventing the development of paralysis in those infected, and the pro-

vocation of paralysis which may possibly result from the injection of inactivated vaccine in the presence of an epidemic, is not to be feared. Oral immunization also reduces the total number of injections and individuals needs and the vaccine is cheaper to make.

(*h*) *Smallpox*—A strong argument usually forwarded in respect of vaccination against smallpox in infancy is based on the fact that the complications of this malady have been proved to be severer and more frequent when vaccination is carried out at a later age period than when it is done in infancy. Further more risks of infecting rest of the population in the world due to increased international travel can be decreased only by immunization against smallpox in infancy. Certificate becomes valid after 8 days of successful primary take and on the day of revaccination and remains so for 3 years.

Now smallpox vaccine is also prepared in dry form in freezed dried vacuum sealed preparation of living vaccinia virus which is reconstituted just before use with the diluent provided with it. It is supplied in 25 dose and 100 dose packs. Dried vaccine lasts indefinitely at refrigerator temperature (2°*C* to 10° *C*), for a year when kept in a cool dark place (not over 15° *C*); or for one month at tropical temperatures. The reconstituted vaccine should be used at once but will retain potency for one week if kept in the refrigerator.

Doses—The number of pressures to be made depends on whether the vaccination is primary or a revacination (and if the latter after how long an interval), on the age of the subject, whether it is a repeat following an immediate previous failure and on the imminence of risk, as shown in the table below:—

	Degree of risk	Nature of vaccination	Number of drops of vaccine	Number of pressure per drop	Remarks
Infant ..	Normal ..	Primary ..	1	30	
	Epidemic, or undue prevalence of smallposx	Primary ..	2 or 3	30	Drops on 2 or 3 separate areas an inch apart.
	Normal ..	Revaccination (after first infant vaccination).	1	10	Primary response likely.

Response to vaccination can be given in stages as mentioned below:—

	Papule	Vesicle	Pustule	Scab	Scab off
Primary ..	4 days	5 days	8 days	11 days	21 days.
Vaccinoid or Accelerated ..	2 days	3 days	4 days	5 days	8 days.

Immediate or Immune Papule under 1 day (usually no vesicle. Fades within 3 days) The time given may vary considerably.

Immunization of Contacts—All persons who are known to have been in contact with a case of smallpox should be vaccinated or revaccinated at once. Those who have never been vaccinated before or whose vaccination history is considered unsatisfactory should also be given post-vaccinal gamma globulin by intramuscular injection. This material is obtained from the plasma of recently vaccinated individuals and contains antibodies to vaccinia virus. The best dose has not yet been determined. In general 1·0 to 1·5 gms. may be given to adults and to children alike, but doses of 2·0 gms. or more may be desirable for unvaccinated close contacts. Vaccination should when practicable precede injection of the gamma globulin by 12 to 48 hours then interference with active immunization by the injected antibodies is unlikely and the vaccination should be succesful, if it is not, however, it should be repeated.

For chalking out immunization programme designed to protect children; any of the following two types of schedules can be adopted against five diseases. The Schedule A is usually preferred;—

SCHEDULE A

Age	Visit	Vaccine	Injection	Interval
5 weeks 6 months	1	Pertussis ..	1	4 weeks or more between each.
	2	Pertussis ..	2	
	3	Pertussis ..	3	
7-10 months	4	Poliomeolitis ..	4	4 weeks or more
	5	Poliomylitis ..	5	
	6	Diphtheria and Tetanus ..	6	4 weeks
	7	(Diphtheria, Tetanus and Pertussis) ..	7 8	
15-18 months	8	Diphtheria and Teanus	9	
		Poliomyelitis* ..	10	
		Small pox sometimes during the first 5 years		
School entry	9	Diphtheria and Tetanus ..	11	
8-9 years	10	Diphtheria and Tetanus	12	
		Smallpox (re-vaccination)		
10-15 years	11	B.C. .G ..	13	

*There is no doubt that a fourth dose of poliomyelitis vaccine will be necessary but the exact timing of this has not yet been decided.

SCHEDULE B

Age	Visit	Vaccine	Injection	Interval
2.6 months	1	Triple (Diphtheria, Tetanus and Pertussis).	1	4 weeks or more between each.
	2	Triple (Diphtheria, Tetanus and pertussis)	2	
	3	Triple (Diptheria, Tetanus and Pertussis).	3	
7-10 months	4	Poliomyelitis	4	4 weeks or more.
	5	Poliomyeltis	5	
15-18 months	6	Triple (Diphtheria, Tetanus and pertussis) Piliomyelitis*	6	
		Smallpox sometimes during the first 5 years	7	
School entry	7	Diphtheria and Tetanus	8	
8-9. years	8	Diphtheria and Tetanus Small-pox (re-vaccination)		
10-15 years	9	B.C.G.	10	

There is no doubt that a fourth dose of poliomyelitis vaccine will be necessary, but the exact timing of this has not yet been decided.

2. Passive Immunization

(*a*) *Tetanus*—Tetanus anti toxin (A.T.S.) is prepared by immunizing horses with tetanus toxoid followed by tetanus toxin. The plasma or serum collected from these animals is refined and concentrated.

Tetanus anti-toxin should be given to prevent tetanus developing following wounds or injuries when the patient is non-immune and the injuries are such as to give rise to risk of tetanus.

Doses—Non immune persons should be given a dose of 1500 international units of tetanus anti-toxin by intramuscular injection after a sensitivity test. Except in the case of an individual who has had previous injections of serum (including serum vaccine preparations, *e.g.*, Diphtheria T.A.F.), when the dose may be raised to 10,000 units, this should generally suffice. However, similar doses may be given at weekly intervals, if considered necessary, in the light of clinical progress.

Duration of effect of tetanus anti-toxin, after treatment—The protective effect of injection is generally at its heigh in two or three days. It may be adequate for about 2 weeks, which has become slight before three weeks have passed. Since one treatment with anti-toxin is liable to render the future use of anti-toxin of less value the last dose of it given on such a first occasion should be followed after 6 to 8 weeks, by the first

dose of vaccine for primary vaccination against tetanus. (If vaccine is given earlier, remaining anti-toxin may interfere with the active response).

(b) *Diphtheria*—Passive immunization against diphtheria may be required in the following circumstances.

(*i*) For contacts of cases of diphtheria unless such contacts are known to be immune.

(*ii*) For persons who are not known to be immune and who are at risk during the explosive outbreaks of diphtheria.

Prophylactic dosage of diphtheria anti-toxin—500—2000 units of diphtheria anti-toxin may be given intramuscularly after sensitivity test.

Duration of Passive Immunity after treatment—The protection afforded by the above-mentioned dose usually lasts for about 2 weeks. In a person sensitized by previous injection of serum the duration of protection may be much shorter perhaps only 2 or 3 days.

(c) *Role of Gamma Globulin in Passive Immunization*—The gamma globulin fraction of the serum proteins contains almost all the antibodies against bacteria and viruses. In clinicial use it has the advantage that the equivalent doses only about 1/20th that of whole serum, as well as greater stability and virtual freedom from risk of transmitting serum hepatitis. The principal uses of it can be mentioned in the following diseases:—

(*i*) *Measles*—To abort or modify the disease in young or debilitated children and to control institutional outbreaks. As a rule modification is preferable since this is followed by permanent active immunity. Given within 6 days of exposure, 250 mg. will usually prevent the disease in infants and modify it in children over one year (McDonald and Cockburn, 1954). The protection lasts about 3 weeks.It is ineffective once the rash has appeared.

(*ii*) *German Measles*—To protect women exposed in the first four months of pregnancy and prevent damage to the foetus 1.5 g. should be given without delay, desirably within a week of exposure.

(*iii*) *Poliomyelitis*—To provide protection for about 5 weeks for young children, pregnant women and tonsillectomised subjects. It is effective when given before exposure, but its value in home contacts is negligible because they have probably already been infected when the first case is diagnosed.

(*iv*) *Infective hepatitis*—To control institutional outbreaks and to protect, for example pregnant women, nurses or certain children with in 7 days of exposure for subjects below and above the age of 10 years dosage is respectively 250 and 300 mg.

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DEMOGRAPHY AS A SCIENCE AND IT'S EXISTENCE IN PAKISTAN

By

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Demography as a science has to play a very important and significant role in the development of Pakistan or we can say that without a sound knowledge of this science we cannot keep pace with the rapidly advancing world. Demography provides us a real picture of the nation's health and its socio-economic status. In other words demography is concerned with the growth, development and movements of population as a whole. Demography constitutes most important part of statistics and a good statistical system is the back-bone of human activities. In order to have an idea of national economy and its impact upon the social status of the people, it is very necessary to have an accurate statistical data. In this atomic age of ours proper planning in unthinkable without a sound knowledge of statistical methods and in Pakistan like all other countries the success of national projects hinges on the quality of statisticians to a large measure.

For all types of demographic studies we are mainly concerned with the details of human population of a particular area. Information about age, sex births, deaths, migration, marriages, divorce, infant mortality, occupation of different earning members, type of household, education, locality and many other socio-economic problems is elicited and interpretted through the science of demography. Without knowing the demographic characteristics of the population of a country we can-not proceed further with proper and methodical planning. In Pakistan the importance of statistics especially of demography has been realised by the Government and the Health Department is trying its best to equip the medical men with this science.

Demographic activities may be called (Human Book-Keeping, for the work is concerned with the maintenance of the charateristics of the population from birth till death. In Pakistan Public Health authorities, relying on system functioning irregularly or inefficiently, remain ignorant of the physical conditions of the people, of the revailing diseases and their age, sex, local and seasaonl distribution. Generally speaking it may be said that no infectious disease except cholera, small-pox, fevers and plague are reported from rural areas. For other diseases in the rural areas, few reports are made. In the light of the information available, the authorities will lack accurate knowledge indispensible to the framing of future legislation in the interest of public health. No one with a little common sense would venture to invest money where accounts are not properly maintained and regularly audited. It is only by such means as the statistical data, that the directors of successful commercial enterprises are kept informed of the exact state of affairs at all times of all branches of their business. Similarly details characterising health, disease and medical policies if not submitted to regular and objective audits throughout the year, effective plans cannot be made and functions successfully.

The present day procedure in official demgoraphy undoubtedly owes much to William Farrthan to any other person. So far he can safely be regarded as the greatest medical statistician ever born and lived. The application of statistical methods to the study of biological problems began with Galton. He may be said to have been the founder of

science of biometry. (Biometry is the branch of science which studies the quantitative aspects of vital phenomena.)

Pakistan is an over populated country with population, excluding Jammu and Kashmir, Gilgit and Baltistan, Junagarh, Manavader and Pakistan enclaves in India on 1st February, 1961 was 9,38,31,982 persons including 1,11,369 Non-Pakistanis. There are 4,93,08,645 males and 4,44,11,968 females in Pakistan. In 1951 the population of the corresponding territory was 7,58,79,165 including 2,066,69 non-Pakistanis and also the estimated population of Gawadar (13,000) which become a part of Pakistan in 1958. The population in 1961 has thus registered an increase of 23·70 per cent of the total population, (23·00 per cent of males and 24·4 per cent of females). The total area of Pakistan is 8,65,529 square miles. West Pakistan with 84·9 per cent of the total area has 45·8 per cent of the population. On the other hand, East Pakistan with only 15·1 per cent of the total area has 54·2 per cent of the population. The brief comparison of the rate of growth within the two wings of Pakistan indicates large scale population movements and greater development opportunities in West Pakistan and a rather immovable population with little room to shift the pressure in East Pakistan. The average density of 253 persons per square mile for the whole country makes an extraordinary wide range of variation between the two wings, more especially between large administrative areas within the provinces. For every person on a square mile of land in West Pakistan, East Pakistan has about seven.

In sharp contrast, 10 out of 17 districts of East Pakistan have density of more than 1,000 persons per square mile. Briefly the distribution and concentration of population in Pakistan makes a fascinating study bringing out in bold relief the problems of land utilisation, prospects of diversification of occupation and the trends of pop ulation movement in different parts of the country.

The ration of men to 100 women for Pakistan as a whole was 113 in 1951 and 111 in 1961. This sex ratio for rural area in Pakistan is 109 and for urban area 129 as shown by the Census of 1961. The higher number of males in the urban population is mostly due to the fact that there have always been more males than females among the migrants. Among different types of communities, the sex composition is not uniform. The ratio is considerably higher is urban area than rural area. In 1961 census it was Reported that in urban population there are 29 per cent more males than females.

In 1951 Census as a result of high mortality and high fertility, Pakistan had extremely young population. Their age pyramid revealed a very broad base with quickly attenuated top. It stood in marked constrast to the kind of pyramid found in countries with low fertility and mortality. Slightly more than the half of the population is represented by the age group 10—39. In Pakistan as in the rest of the world, the number of boys at birth was greater than that of girls but, owing to their higher mortality rate this numerical advantage was lost before they were one year old. The shortage of girls began at the age of ten and became greater during the age period 40—50 and after the age of 55 women regained their superiority in number.

The above given figures provides us a sound ground that the population of Pakistan is increasing very rapidly. If this rate of expansion is allowed to continue unchecked we

will have to suffer from many socio-economic problems which are already creating innumerable trouble in the proper development and advancement of the country. The main cause of these difficulties is the lack of statistical data regarding Pakistan especially the East Pakistan and the former Punjab, which had been divided at the time of partition. The division of the sub-continent and its attendant aftermath made it impossible to get sound reliable statistical information. As it has already been pointed out, in Pakistan between the census of 1951 and 1961 our population has increased, at the rate of 2·37 per cent per year as compared to the rate of increase of 1·7 per cent per year for the rest of the world. This rapid multiplication is leading us to serious socio-economical and political problems which in turn have a very grave effect on the public health. Due to this abnormal increase in population we have not been fully able to fulfil the objectives of the First and Second Five Year. Plans. In order to maintain any population grounup adjusted to the surroundings we require a simultaneous increase in our food-grain, housing, water supply, public utilities, services and other human necessities. Our resources are limited and are not increasing as rapidly as our population is. The Pakistan Planning Board in its preliminary recommendations has stated that even if all the resources were exploited to raise the food production, the rate of increase of food production-he will lag behind the rate of increase of population. To the limitation of family size there is much of opposition. Some religious groups may oppose family limitation, on moral principles. Other groups believe that children add much to the joy of living and bringing comfort, social security and joy in the old age. Other people feel and advocate that children should be given a full education and various opportunities and advantages of modern society and so favour the family limitation. From the health point of view it is important to improve our social and economic standards which are related to the population. If health has to be improved, growth of population must be controlled and for that family limitation needs be advocated.

It is encouraging to note that quite a number of statisticians and technicians and others are conducing study surveys to find out the sources of unreliability and defficiency existing is the statistical data. There are two types of population statistics :(*i*) census (*ii*) registration of events as and when they occur. In addition it is also possible to obtain both types of information by means of special sampling survey. This was the technique used by Social Sciences Research Centre of the Panjab University, Lahore, while collecting the requisite information about socio-economic studies and fertility experiences for-the citizens of Lahore in collaboration with the population Council, New York. The contribution of Mr. Khalid Hayat Khan to the developing of Demographic Science in Pakistan is tremendous. Khan's distribution is the result of his efforts and dedication to the cause of this science. The efforts, of Dr. A. H. Awan in this field are also appreciable. A study of some of the population characteristics in the area of Nicholson Road, Lahore, was conducted by Mr. S. M. Anwar and Dr. Siddiqui. Dr. Zia-ud-Din read a paper "Demographic study in West Pakistan" in the International Conference at Vienna. A study was also conducted in Qilla Gujjar Singh area of Lahore to find out relationship between economic-status, environmental sanitation and infant health. It was found that infant mortality decreased as the *per capita* income increased and it was less in those families where the standards of

environmental sanitation were good. A study was conducted for the village Budhopur (District Gujranwala) by six men from the University of Cambridge. In this study the influence of social organisation on the individual groups, on village politics, social grouping, mutual alliance, marriages etc., has been examined with great thoroughness. The standard of cultivation implements used, crops growing, yield obtained and income earned have also been observed. This information, it is, visualised will be of far reaching importance when projected to those high up in the hierarchy. There is still further need for thorough and unbiased studies.

Vital events, that is age sex, births, deaths, migration, marriages, etc., coming from registration are mostly inaccurate biased, mileading and unsuitable from the statistical analytical registeraview point. While analysing the available information for morbidity, mortality and mig- ration, etc., in Pakistan, we should be very careful and should not take these values as free from bias.

In municipalities, the local authority and in rural a areas village headman (Chowkidar) are the usual reporting agencies, In those areas where police is responsible for the registration and the reporting of vital events the information obtained from the villages falling under the jurisdiction of a particular Thana, is limited to some days of the month, weeks or fortnights. Moreover if the policeman collecting informaton is not properly entertained with 'Nazrana', birth will seldom be registered. The result being that birth, death, and cases of infectious diseases are frequently registered much later than the actual date of occurrence. The Chowkidar's records of birth, death are often incomplete. Usually the registers are not maintained by the Chowkidars themselves but are got done by literate villagers, and the chances of false informaton creeping in are too great. Registers so maintained cannot be relied upon to give complete records of annual births and deaths on the following grounds :—

(1) Not all Chowkidars are conscientious in recording all births, nor all fathers always notify the birth of a child who dies almost immediately.

(2) Some women come from other villages to their mothers for child-birth and these are also entered in the register. Some women going out to their mothers for child birth are not recorded. The discrepancy is great.

In the absence of life table we do not know the average duration of life enjoyed by a person and the expectation of life. Computation of life table generally take 80 to 90 years, but in Pakistan even in this period it will not be possible to frame an accurate life table, because the reporting of the age is mostly inaccurate.A life table for the population Lahore is about to be relaased but this life table based on census figures will not be free from criticism. Life table is the-back-bone for the successful operation of insurance companies and many other commercial enterprises.

Taking into consideration the ambiguities and discrepancies of the vital events; question arises as to what line of action should be channelised in order to get maximum possible accurate and reliable statistical figures.For this purpose, the following few suggestions are put forward and it is hoped a considerate approach will certainly bring good results.

I. Chowkidar (as vital events reporting agent) may be replaced by more efficient reporting agency. The following can be useful alternatives :—

(i) Employment of health officials, i. e., sanitary inspector or health visitor for

birth registration.

(*ii*) Prescribing certain minimum qualification for the persons holding the post of Registrar.

(*iii*) These duties of the Chowkidar may be transferred to the Basic Democrats because they can collect much better information than Chowkidars. This system will be of greatest benefit. The living example of marriage registration and the efficient manner in which this business is being conducted calls for the Basic Democrats to share this vital issue as well.

II. At the time of each birth and death a certificate may be issued. This certificate of birth must be produced whenever it is required or asked for. Record of these certificates may be retained in the Union Councils Offices. This certificate will be of material value while determining age, etc.

III. Birth registration should include two new items in respect of each birth : (*i*) age of mother (*ii*) number of pregnancy. The first is required for calculation of "fertillity" and the second for the study of maternal and child mortality.

IV. Voluntary registration Act of Births and Deaths must be replaced by compulsory registration Act and for that the new legislation is warranted.

V. Private Medical Practitioners if join hands and supervise the reporting of vital events with special reference to cause of death things can be improved considerably without any burden on the national exchequer.

VI. Question of transference of vital events record to the places to which certain persons or groups of persons belong requires consideration.

VII. Another important factor is that well concerted and ceaseless battle need be fought by all ranks of people jointly to rid the country of illiteracy. With the percentage of literacy coming up quite a few problems get automatically solved.

VIII. The role of useful and proper publicity, too, can be of far reaching importance. All the available means of propaganda to enlighten the laity about the inherent virtues of the programme be exploited and untiring efforts be made that the message reaches most of the public. Any lacuna in the programme means failure to get near the targets.

IX. The training and quality of the field workers, is still another very vital factor which needs considerate attention. After all the entire outcome of any programme is significantly dependant upon the way, the field workers take their jobs. Here we require people of high moral integrity and selfless devotion. The behavioural pattern of people' their socio-cultural and religious biasis should be carefully viewed before attempting to elicit any information. The approach must differ with every different individual for each individual is an institution in itself.

To overcome these difficulties in Pakistan, an effective welfare programme has to be launched. For this purpose constant hardwork, co-operation from all members of society, good planning and sound leadership is required.

The impact of this information on the national projects is very very great indeed. It is thus ripe time that an all-out attempt is made to get reliable vital statistics. It is only

then and then alone that we can think of ushering in an era of real happiness, joy, satisfaction and patriotism, through well-planned and scientifically calculated projects of nation-building.

I am confident this article will contribute in its own humble way to the benefit of motherland.

Book Review

CLINICAL PHYSIOLOGY edited by Campbell, E. J. M., Dickinson, C. J., Slater S. D. H. (Pp. xiii 605, 57s 6d), and Ed, 1963. Oxford: Blackwell Scientific Publications.

This book has been throughly revised and brought up to date. It is a well written book and well prove to be useful companion for these who wish to understand 'physiology gone wrong'. Lists of useful references have been appended at the end of each chapter. It is recommended to both undergraduates and General practitioner.

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Leading Pakistani Malariologist awarded the Darling Prize

Colonel Manowar Khan Afridi, Honorary Consultant in Malariology to the Ministry of Health, Pakistan, has been awarded the Darling Foundation Medal and Prize for his contribution to the control and eradication of Malaria over the past 30 years and for his laboratory and field studies in many aspects of practical malariology. The recommendation of the award, which was made by the Darling Foundation Committee, was endorsed by the WHO Executive Board during its 33rd session, held in Genava early this year.

This is the ninth award of the Foundation, created by private funds to honour the memory of Dr. Samuel Taylor Darling killed in an automobile accident in 1955 during a study mission of the League of Nations, Malaria Commission. It consists of a bronze medal and a fixed sum of one thousand Swiss francs.

Colonel Afridi, the first Darling Laureate from an Eastern Mediterranean country, is a leading contributor to malaria control and research work. He began his career with the Indian Army Medical Corps in 1924 and later specialized in bacteriology and serology. After the Second World War, Dr. Afridi was appointed Director of the Malaria Institute of India, and then of the Malaria Institute of Pakistan. Later he served as Director of Health Services of the North-Western Frontier Province until his retirement in 1955.

In 1958, Colonel Afridi was appointed Vice-Chancellor of Peshawar University and served in that capacity until 1962 when he became the Honorary Consultant to the Ministry of Health, Government of Pakistan.

A holder of high degrees from Pakistan and United Kingdom Universities, and a former WHO Deputy Regional Director for the Eastern Mediterranean, Colonel Afridi is a member of the WHO Advisory Panel on Malaria. He has held a number of consultantships for the World Health Organization, was elected Chairman of its Executive Boards 30th Session in May 1962, and is presiding over the 17th World Health Assembly, currently meeting in Geneva.

The laureate will receive the Darling Foundation Medal and Price during a plenary meeting of the Assembly.

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