JUVENILE METROPATHIA—ITS INCIDENCE AND AETIOLOGY*

by

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Juvenile metropathia is dysfunctional bleeding occurring in young girls from puberty, i.e. from 12-14 years till the age of 20 and should include puberal bleeding and bleeding of adolescence.

Hamblen has divided the age incidence into two periods:

Years	white	coloured	Total
10-15	1.3%	0.3%	1.6%
15-20	9.8%	2.0%	11.8%

Mayer and Goldstein refer to only 17 cases of puberty bleeding. Mazer and Israel found puberal bleeding only in 3% of cases under the age of 20, while Sutherland reports an incidence of 3.8%. Both are of opinion that in general practice, the incidence must be much higher because at this age dysfunctional bleeding is self-limiting and only occasionally requires hospitalization. Most of you in Bombay, I am sure, will agree with me that the incidence should be even less than 3% — perhaps we see one or two cases per month, both in private and general hospitals, compared to the large number of women with functional bleeding after the age of 30. Though requested by the Chairman, I was unable to collect any definite data on the incidence of juvenile metropathia in schools, colleges or nurses' homes and hence it would be worthwhile for any of us to investigate into the matter in our own country. I do think however the incidence is higher in the poorer class of patients, in whom socio-economic factors and malnutrition play an important role and I do feel that if poverty, malnutrition and stress of socio-economic conditions are eradicated we will have much fewer cases of juvenile metropathia. In my practice, I have found a larger number of cases in the Muslim community, probably due to purdah system, less sunshine and lack of exercise, and chiefly due to poor nutrition, calcium deficiency and anaemia. It is less common in the Christian and Parsee communities and I do feel diet plays an important part.

Hypoproteinemia favours bleeding. In the non-vegetarians rich proteins can be easily had in meat, fish and eggs, while the vegetarians have to depend on large quantities of milk and rich pulses which the poor cannot afford. Constitution definitely plays an important role in the etiology of juvenile metropathia. It is more commonly found in the frail, emaciated, nervous and overworked girl, than in one who is well built, and emotionally stable. Sedentary occupa-

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tions and less exercise favour juvenile metropathia.

Amongst environmental factors, I think the village girl should suffer much less from this evil, than one in the town living in crowded localities, with all the hustle and bustle of a family life which increase emotion and stress. Unhappy home-life, either because of a step-mother or very strict parents or disagreement between family members disrupting the quiet and peace of home, is apt to influence the emotions of a growing girl, and be the underlying cause of her bleeding.

Hereditary and familial traits do predispose to juvenile metropathia, the most important being familial blood dyscrasias, e.g. haemophilia. In a large number of cases, the history indicates that there is bleeding tendency in the mother or the sisters but this exact history is difficult to obtain from our poor and illiterate patients. I had absolutely no occasion to study juvenile metropathia in twins and I would very much like to know if any member in the audience can enlighten us on the point. I have no figures to prove the facts, but I do not think that diseases of childhood have any actual bearing on juvenile metropathia, except tuberculosis, because this may leave behind a hidden tubercular endometritis which was found in 4% of cases reported by Sutherland.

Of the infectious diseases mumps may affect the ovaries but if the infectious disease immediately precedes puberty, it may leave the child weak and anaemic and thus poor general health might cause menorrhagia. Now a few words on aetiology.

Functional should apply to such bleeding as is caused by a disturbance of normal function, whether nervous, or endocrine or due to general systemic diseases, before any structural changes have begun. The limits of normal function are so wide, that perfect health may exist within these limits.

Menstruation depends upon 3 important factors: (a) normal internal genitals, (b) perfect endocrine control, (c) adequate general health. Any pathological finding of the internal genitals, palpable or otherwise — macroscopic or microscopic — would contribute to an organic cause of bleeding, while disturbances in the latter two would cause functional bleeding.

True functional bleeding is a disorder of menstruation which has a very complex mechanism.

Besides the histological changes of the endometrium, its stroma and glands, much attention is now focussed on the complex blood supply of the functional layer of the endometrium studied in detail by Markee in the anterior chamber of the eye of the rhesus monkey. Thirty-six hours before bleeding starts there is a marked slowing of blood in the spiral arterioles due to regression of corpus luteum and shrinking of the stroma which further increases the coils of the arterioles. At the same time many workers believe that arteriovenous anastomoses, connecting the coiled arteries with the parallel collecting venules, open up and so shunt the blood from the capillary network. The slowing of blood in the coiled capillaries leads to anoxaemia and necrosis of

the stromal cells. It is possible that these dead cells liberate a toxin called Necrosin which acts as a vasoconstrictor of the coiled arterioles and still further restricts the endometrial blood supply. This vasoconstriction of the arterioles may also be due to nervous stimulation, as nerve fibres have been described in the walls of the arterioles. During the spasm the endometrial cells of the capillaries become anoxaemic, degenerate and allow diapedesis of red cells into the stroma. After the wave of constriction passes off blood rushes in, ruptures the capillaries whose walls have become partly necrotic and form subepithelial haemorrhages which eventually leak into the uterine cavity and produce bleeding. This wave of constriction and relaxation of the arterioles acts on different parts of the endometrium at a time and therefore controls the amount of oozing. Excessive loss in the first 2 days might be due to imperfect constriction of the coiled vessels and relaxation of too many arteries at the same moment. The essentials of menstruation therefore are a falling hormone level of oestrogen and progesterone, and consequent thinning or shrinking of the endometrium, increased coiling and contraction of the arterioles, haemostasis in superficial layers, necrosis, bleeding and shedding of endometrium.

True functional bleeding, according to Sutherland, arises from: (1) endometrial hyperplasia in 31% of cases, (2) irregular shedding in 1.5% of cases, (3) irregular ripening of the endometrium in 3%, (4) atrophic endometrium in 2% of cases while in 73.5% the bleeding occurs from absolutely normal endometrium where the cause may have been some disturbance in the vascularity of the endometrium, e.g. abnormal constriction and relaxation of the arterioles due to some neurogenic cause as a result of stress or psychic disturbance, or due to some blood dyscrasia.

Amongst the disturbances in the endocrine chain the anterior pituitary at this age may be hypofunctioning producing chiefly less or continuous luteotrophic hormone resulting in the prolonged shedding of endometrium. Hypothyroidism is sometimes found; diagnosed by basal metabolic rate, protein bound iodine, etc. But many of these juvenile patients have blood dyscrasias like haemophilic tendencies, anaemia, thrombocytopenia, increased bleeding time, deficient clot retraction, etc. Lastly the aetiology may be found in malnutrition, hypoproteinemia and emotional stress.