Haemorrhoids—postulated pathogenesis and proposed prevention

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Summary

The very high prevalence of haemorrhoids in the most economically developed countries is contrasted with their low prevalence in rural communities in developing countries. Traditional concepts of causation are shown to be no longer tenable. It is argued from epidemiological, clinical and experimental evidence that the fundamental cause of piles is straining at viscid stools which are the result of fibre-depleted diets.

The size of the problem

Haemorrhoids have been referred to in literature dating back to the pre-Christian era, but the term seems often to have included other peri-anal conditions. Not a few great men in previous centuries were allegedly afflicted, but there is no information as to whether the poorer people suffered from the disease, nor about its overall prevalence. Haemorrhoids have for centuries been treated by ligature and cautery, but their cause has remained obscure.

Today, haemorrhoids are considered to be one of the most common ills of man, a judgment made by those with vision limited to the western world. The prevalence of haemorrhoids is still unknown and the lack of clear definition makes an assessment impossible. It has been suggested that at least one person in two in the western world over the age of 50 years has some degree of haemorrhoid formation (Goligher, 1967). Hyams and Philpot (1970) specifically questioned 1584 adults and concluded that about one in four of those over 30 years had some degree of haemorrhoids.

The cost to the community, both financial and in lost working days, is great and by any standards this condition must be considered a major health hazard.

In this communication only so-called idiopathic haemorrhoids will be considered.

Epidemiological evidence

Almost all those who have sought to account for the occurrence of haemorrhoids have incorrectly assumed western experience to be representative of all mankind, but this is far from the truth. Morgagni in the 18th century, and, more recently, other observers recognized that haemorrhoids did not

occur in animals. Morgagni attributed their development in man to his upright posture but others had insight (Graham-Stewart, 1962) to suggest that the absence of haemorrhoids in animals was because 'they seldom attempt to defaecate until they have a strong desire to go to stool', thus obviating excessive straining. Nevertheless, being unaware of the fact, no attention was drawn to the rarity of haemorrhoids in people living traditionally.

Geographical distribution

One of us (D.B.) during 20 years' surgical practice in Africa, was frequently confronted with haemorrhoids in the relatively small expatriate community but seldom in a much larger African practice. Cleave, Campbell and Painter (1969) and Dodd (1964) have drawn attention to the relative rarity of haemorrhoids in developing countries and Trowell (1960) recounted that in nearly 30 years' practice in Africa one of the only two African patients he had seen with severe anaemia due to bleeding piles was a prince taking a semi-European diet.

An attempt has been made to substantiate extensive anecdotal evidence of the relative rarity of haemorrhoids in rural communities in developing countries by a prospective study in which eighty-five rural hospitals in Africa and India have been reporting on a monthly questionnaire form the number of patients seen with bleeding or prolapsed piles. In rural Africa less than one case is seen, whether admitted or not, per 1500 hospital admissions, which represents approximately 15,000 outpatients. Haemorrhoids appear to be commoner in Nigeria and Ghana, both longer exposed to western influences, than in East Africa. They seem to be about three times more common in rural Indians than in Africans.

Even accounting for gross under-recording there can be little doubt that haemorrhoids are much less common in developing countries than they are in North America and western Europe.

Changing patterns

The biggest demonstration of the influence of a changed environment on the prevalence of haemorrhoids is the current comparable prevalence of the condition in black and white Americans in contrast

to its relative rarity in Africa, including the areas from which American slaves had originally been taken.

There is considerable evidence that haemorrhoids become commoner with progressive adoption of a more western way of life. Osman (1973) has drawn attention to their increasing prevalence in Khartoum, and Bremner (1971) to the increasing number of Africans requiring treatment in Johannesburg.

Relationship to varicose veins

Not only haemorrhoids but also varicose veins have their highest prevalence in more economically developed western communities and their lowest in communities least influenced by western culture. In almost every situation investigated, the prevalence of haemorrhoids appears to have risen before that of varicose veins. There are many instances where both conditions are common or both rare, and several where haemorrhoids are not uncommon but varicose veins are still rare.

Relationship to stool characteristics

Communities in which intestinal transit times are short and stools normally large and soft, have a low prevalence of haemorrhoids and varicose veins, and also of certain other characteristically western diseases, for instance appendicitis, diverticular disease of the colon, large bowel cancer and hiatus hernia. In contrast, these diseases have their highest prevalence in communities who pass small firm stools and have prolonged transit times (Burkitt, Walker and Painter, 1972; Burkitt, 1973).

There are, however, apparent exceptions to this rule. In certain parts of India, haemorrhoids appear to be not uncommon where stools are customarily large. Bowel infections in these areas and their possible relationship to haemorrhoids will be discussed below.

Postulated causes examined in the light of epidemiological evidence

Numerous explanations have been offered to account for the occurrence of haemorrhoids. Bacon's (1949) list of postulated aetiological factors, given below, embodies most of the suggestions of those who have considered this problem: (1) heredity; (2) anatomical features; (3) nutrition; (4) occupation; (5) climate; (6) psychic factors; (7) senility; (8) endocrine changes; (9) irritation from drugs or food; (10) infection; (11) pregnancy; (12) increased intra-abdominal pressure from: (a) violent exercise, (b) constrictive clothing, (c) straining when coughing, sneezing or vomiting; (13) constipation.

He included in addition tumours, engorgement of the portal circulation and uterine proidentia, but haemorrhoids resulting from such conditions would

not be considered to be idiopathic. More bizarre explanations have been attempted, varying from sitting on hot radiators to riding horses. Many of these postulated aetiological factors can be eliminated as primary causes by measuring them against the yardstick of epidemiological evidence. This does not exclude their playing a contributory role.

1. Heredity

The similar situation in black and white Americans in contrast to that in Africa and the evidence of increasing prevalence following a change in life patterns, indicate that heredity is not a primary causative factor.

2. Anatomical features

The absence of valves in the portal venous system and the anatomical arrangement of haemorrhoidal veins and their surrounding structures have frequently been incriminated as being responsible for the occurrence of haemorrhoids. The similar anatomy in communities with a high or low prevalence invalidates such suggestions.

3. Nutrition

There is no evidence to suggest a relationship between haemorrhoid prevalence and nutritional status.

4. Occupation

This could be a contributory but not a primary factor, since haemorrhoids are not common amongst those in developing countries following occupations which are said in the west to be responsible for this condition.

5. Climate

Extremes of climate are experienced in East Africa but this does not appear to affect in any way the prevalence of haemorrhoids.

6. Psychic factors

Evidence seems lacking.

7. Senility

Although the prevalence of haemorrhoids, varicose veins, diverticular disease and hiatus hernia increases with age in western countries, none of these diseases is common amongst the elderly in developing countries.

8. Endocrine changes

These seem unrelated to the geographical and socio-economical distribution.

9. Irritation from drugs or food

Evidence seems lacking.

10. Infection

Evidence seems lacking.

11. Pregnancy

To consider this a primary factor would imply that mankind has failed to adapt to a common physiological condition. Moreover, any epidemiological relationship between birth rate and prevalence of haemorrhoids is inverse rather than direct.

12. Increased intra-abdominal pressure from:

(a) *Violent exercise*—both active exercise and sedentary occupations have been blamed. Neither suggestion gains epidemiological support.

(b) *Constrictive clothing*—the virtual elimination of constrictive clothing that has characterized women's fashions in recent decades has done nothing to reduce the prevalence of haemorrhoids.

(c) *Straining when coughing, sneezing or vomiting*—as will be pointed out below, all these forms of straining are accompanied by contraction of the anal sphincter.

Strangely, Bacon omitted the one form of straining most relevant to the problem, straining at stool. Other authors have, however, suggested this as a causative factor.

13. Constipation

This has frequently been linked with haemorrhoids, two main explanations being suggested: Cleave *et al.* (1969) consider the basic cause to be the direct pressure of hard faecal masses on the haemorrhoidal veins. Graham-Stewart (1963) and Goligher (1967), on the other hand, blamed the retrograde flow of blood in the haemorrhoidal veins caused by the intra-abdominal pressure resultant on straining at stool.

Anatomical and physiological considerations

Under ideal conditions, defaecation should be almost effortless and the faeces, which should be soft and bulky, are expelled by contraction of the gut and not by the muscles of the abdominal wall. If, however, there is little faecal residue the muscle of the gut cannot act effectively and voluntary straining is necessary. It is this straining, in the presence of a relaxed anal sphincter, which is the cause of haemorrhoids. This is a new concept in that it is recognized that both the nature of the stools and the nature of the straining are important.

Parks (1956) first realized to the full the crucial significance of the fact that venous dilatation in piles is confined to the superior haemorrhoidal plexus lying in the upper part of the anal canal; later one of us carried out further work on the aetiology of piles and described a vascular pump and tube-valve mechanism (Graham-Stewart, 1963).

The following conclusions, which were reached in this work on the cause of piles, were drawn from deductions based on observation and reasoning, and on experimental work.

Firstly internal haemorrhoids, which are to a varying extent varicosities of the superior haemorrhoidal plexus of veins, were divided into two main types—vascular and mucosal. Vascular piles occur most commonly in young persons, especially men, and the bulk of the pile consists of dark blue dilated venules which can be seen clearly through the thin mucous membrane. Mucosal piles, seen associated with pregnancy and in older people, appear to consist mainly of thickened mucous-membrane. These two types of piles have a different aetiology.

Vascular piles develop in the presence of a normal anal sphincter and are caused by straining at defaecation. The piles involve the superior haemorrhoidal plexus, which is situated at the opening of a muscular funnel formed by the pelvic diaphragm and the anal sphincter (Fig. 1). Straining during defaecation raises the intra-abdominal pressure and at the same time the anal sphincter relaxes, creating a pressure gradient. Because of its position this circular plexus of veins is subjected to very high pressure on defaecation. Physiological dilatation of these veins is present from childhood; excessive straining causes pathological dilatation of the plexus, in other words piles. On straining, the intra-abdominal pressure is exerted equally on the valveless portal vein and the inferior vena cava, effectively obstructing drainage by either route, the blood being trapped in a closed compartment. The point of pressure gradient in the portal system is at the level of the superior haemorrhoidal

FIG. 1. Anatomical relationships of superior haemorrhoidal veins and the anal sphincter.

FIG. 2. Distension of superior haemorrhoidal plexus of veins resultant on reversed blood flow caused by straining with simultaneous relaxation of the anal sphincter—the ampullary pump.

FIG. 3. Closure of the superior haemorrhoidal veins caused by further straining—the tube-valve effect.

plexus, whereas in the valved systemic system, it is situated at the point where the inferior rectal veins enter the abdominal cavity. On straining, in the presence of a relaxed anal sphincter, the blood in

FIG. 4. The contraction of the anal sphincter which accompanies other forms of abdominal straining.

the superior rectal venous radicles lying in the lower part of the ampulla of the rectum above the superior haemorrhoidal plexus is under very high pressure. It will, therefore, be forced into the superior haemorrhoidal plexus, the normal direction of blood flow being reversed—this mechanism has been termed the ampullary pump (Fig. 2). Once the blood in the junctional area has been forced into the superior haemorrhoidal plexus, further reversal of flow is prevented by tube-valve closure of the superior rectal venous radicles (Fig. 3). Closure of the veins, by lateral pressure on their walls (the tube valve effect), protects the ascending superior rectal veins from being subjected to a pressure gradient, so that they do not become dilated, and the degree of distension of the superior haemorrhoidal plexus is limited. For this reason piles never become large, and gross venous dilatation, similar to that seen at the oesophago-gastric junction in cirrhosis, never occurs.

Mucosal piles develop when the strength of the anal sphincter is impaired. Normally, sudden or transient increases in intra-abdominal pressure are countered by simultaneous strong contraction of the anal sphincter (Fig. 4). But in pregnant women and the aged the sphincter is unable to contract sufficiently to counteract these rises in pressure

effectively, resulting in herniation of the anal mucous membrane; in addition there is some dilatation of the superior haemorrhoidal plexus of veins, which is caused in part by straining.

The advantage of this concept over previous theories of aetiology is that, although straining may be limited to a period of minutes each day, this mechanism comes into action each time the patient strains and it becomes effective whether the rectum is either full or empty and when, for instance with tenesmus, defaecation is attempted without success. This might explain the presence of piles in some communities not prone to constipation. If these conclusions are accepted most, if not all, the old theories of aetiology are no longer tenable: the detailed evidence in support of the view that piles are due to straining is available (Graham-Stewart, 1962), but it is beyond the scope of this paper.

Direct and ultimate causes

As has been argued above, the direct cause is believed to be raised intra-abdominal pressure effected by straining to evacuate hard stools. Both epidemiological and physiological studies have related the prevalence of haemorrhoids to the size and consistency of stools. This observation can account for the relationship between haemorrhoids and varicose veins since it has been argued that the latter are also the result of raised intra-abdominal pressure, but in this case transmitted to the veins of the legs and exerting pressure on the valves (Burkitt, 1972). The observation that the prevalence of haemorrhoids usually, if not always, appears to rise before that of varicose veins could be explained by the fact that the leg veins are protected not only by valves but also by external pressure in the traditional squatting position for defaecation.

As mentioned above, haemorrhoids can apparently be associated with bowel infections, when the straining associated with tenesmus, and an empty rectum, has a similar effect on the pressures within the superior haemorrhoidal venous plexus. This, however, is an exception to the usual association with constipation.

Deductive reasoning has, however, tended to stop at the level of suggesting constipation as a direct cause, without going on to consider the fundamental cause of constipation. This has presumably been due to the failure to recognize that intestinal behaviour in the western world is far from normal. From time to time some workers have recognized this and Best and Taylor in 1939 wrote: 'Our rich, low-residue diets often necessitate prolonged straining of the abdominal and pelvic musculature. This may last over ten seconds and raise intra-abdominal pressure from 20 to 200 mm Hg.'

Extensive studies in many countries have related stool size and consistency and intestinal transit times to the fibre content of the diet (Walker, 1947; Burkitt *et al.*, 1972). Deficiency of dietary fibre appears to be the fundamental cause of constipation (Avery Jones and Godding, 1972) which implies sluggish transit times and the evacuation of small firm stools. It would appear that where food is rich in plant fibre the source of the fibre is not of crucial importance. However, where dietary fibre is deficient, available evidence suggests that the fibre of cereals, and to a lesser extent that of legumes, is more effective in restoring gastrointestinal behaviour than the fibre of fruits and green vegetables, unless these are taken in very large quantities. The fundamental cause of constipation is held to be the large proportion of calories consumed in the western world in the form of fibre-depleted carbohydrate foods and of white flour and sugar in particular.

Potential prevention

If the argument outlined above is accepted then haemorrhoids are, as we firmly believe, largely preventable. Stool characteristics approaching those of communities with a much lower prevalence, not only of haemorrhoids but also of many other characteristically western diseases, can be readily acquired by the addition of some 2–4 g of cereal crude fibre to daily food. A change from white to whole meal bread is considered to be of prime importance, but the addition of two to four heaped dessertspoonsful of miller's bran to daily diet will normally revolutionize bowel behaviour. This is cheap and effective.

Not only are these dietary measures proposed as a preventive policy, but surgeons are increasingly recognizing that for less advanced haemorrhoids they can be curative. Patients thus treated while awaiting admission often subsequently do not require inpatient treatment.

When emphasizing the prime importance of straining at stool as the direct cause of haemorrhoids one of us (Graham-Stewart, 1963) concluded that there is no evidence that any other aetiological factor is of significance in the causation of piles. We would like now not only to reiterate this but to repeat it slightly altered with regard to the role of fibre-depleted carbohydrate foods as the major cause of straining at stool. 'There is no evidence that any other aetiological factor is as significant in the causation of constipation.'

Napoleon's piles were troubling him during the battle of Waterloo (Bryant, 1971). It is interesting to speculate the effect on history had his attention not been so diverted from the course of the battle. Had the problem been then understood, a pound of bran might have been worth a regiment of cavalry.

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