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Original Contributions

Dietary Fiber and Disease

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• **Many diseases common in and characteristic of modern western civilization have been shown to be related to the amount of time necessary for the passage of intestinal content through the alimentary tract, and to the bulk and consistency of stools. These factors have in turn been shown to be greatly influenced by the fiber content of the diet and by the amount of cereal fiber in particular.**

Mechanisms are postulated whereby these changes in gastrointestinal behavior could in part explain the occurrence of such common disorders as ischemic heart disease, appendicitis, diverticular disease, gallbladder disease, varicose veins, deep vein thrombosis, hiatus hernia, and tumors of the large bowel.

Calorie intake, speed of passage through the intestine, levels of intracolonic pressures, number and type fecal bacteria, as well as levels of serum cholesterol and changes in bile-salt metabolism have all been shown to be related to the amount of dietary fiber consumed.

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INFECTIVE diseases, previously responsible for most death and morbidity in North America, became much less prominent once highly effective drugs were available for their treatment. In contrast, noninfective diseases have greatly increased in prevalence and now dominate the mortality profile. Their prevalence in the affluent West contrasts with the rarity of such diseases in Africa, where two of us (D.P.B. and A.R.P.W.) have worked for many years, and they are in fact rare in all of the developing countries about which information is available[1] (Table 1[2-13]).

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Diseases Emerging in the Western World as Important Clinical Problems

Coronary artery disease was considered a rarity by Osler in 1910. It was still newsworthy in 1925 when Sir John McNee described to English physicians two cases of this "rare condition" that he had seen in the United States.[14]

Appendicitis, first described in England by Parkinson in 1812,[15] appeared to become common after 1880.[16-18]

There is abundant evidence that *diverticular disease of the colon* has become a major clinical problem only in the last 50 years.[8,19]

The emergence of *gallbladder disease* is more difficult to date. Operations for gallstones have increased by 350% in the Bristol Royal Infirmary, England, since 1940, although the total number of operations has slightly decreased.[20]

There is no evidence as to when *varicose veins* became common, but since epidemiological studies indicate that the distribution of varicose veins and *deep vein thrombosis* is closely similar, the frequency of both conditions may well have increased during the same period, as seems to occur in developing countries where prevalence of both of these conditions increases after contact with western civilization.[9] The rapid rise in frequency of pulmonary embolism at the Mayo Clinic at about the turn of the century suggests that deep vein thrombosis may have become more prevalent at about that time.[21]

Hiatus hernia has been recognized as a common condition during the last 30 years.

Hemorrhoids and *tumors of the colon and rectum* are common in the West, but there is very little evidence as to when they appeared as health problems. It may be noted that Stewart,[22] in an autopsy investigation into the prevalence of polyps at Leeds, reported a 14-fold rise between 1910 to 1912, and 1929 to 1931.

Table 1.—Prevalence of Noninfectious Diseases

Condition	In the United States	In Africa	Source
Ischemic heart disease	Responsible for a third of all deaths	Virtually unknown. Incidence just beginning to increase slowly in large cities	Trowell[2] Cleave et al[3] Seftel et al[4] Schrire[5]
Appendicitis	The most frequent of abdominal emergencies	Virtually unknown in rural areas. Incidence starting to rise in more westernized communities	Cleave et al[3] Burkitt[6] Walker et al[7]
Diverticular disease	The most common disease of the colon	Almost unknown	Painter and Burkitt[8]
Gallstones	Present in some 10% of the adult population	Exceedingly rare	
Varicose veins	Present in over 10% of the adult population	Present in probably under 0.1% of those living in a traditional manner. Increasing with adoption of western customs	Cleave et al[3] Burkitt[9]
Deep vein thrombosis and resultant pulmonary embolism	These make hospital life increasingly hazardous	Very rare	Cleave et al[3] Burkitt[9]
Hiatus hernia	Demonstrable in nearly half the population over the age of 50 years	Almost unknown	Burkitt[10]
Hemorrhoids	Demonstrable in nearly half the population over the age of 50 years	Rare or very rare according to degree of westernization	Burkitt[9]
Cancer of the colon and rectum	Second only to lung cancer as a cause of death from neoplasms	Rare	Burkitt[11]
Obesity	Nearly half the adult population is markedly overweight	Rare amongst those living wholly on traditional diets. Becomes common with urbanization and adoption of western foods	Cleave et al[3] Scotch[12] Walker[13]

A study of art over the centuries suggests that *obesity*, now the scourge of western society, was rare in the common man of Europe until about 200 years ago, as noted by H. C. Trowell (written communication, 1972).

Prevalence of These Diseases in American Negroes and Immigrant Populations

The aforementioned diseases have a comparable prevalence in white and black Americans; such discrepancies as exist are small in comparison with those between Africans and American Negroes. In the latter, it is of particular interest that wherever the emergence of these diseases can be traced, the rise in incidence has lagged some 30 years behind that in American whites. Thus, coronary heart disease (as noted by G. E. Burch in a written communication in 1971), diverticular disease,[23] appendicitis,[24,25] and both benign and malignant tumors of the large bowel[26,27] were much less common in the black population until about a generation ago.

Many of the diseases enumerated are now much more prevalent in Japanese immigrants to Hawaii than they are in Japan (as noted by Stemmermann[28] in a published report in 1970 and a written communication in 1971). These diseases are also increasing in prevalence in Japanese urban communities.[29] Similar changes have been shown to occur in New Zealand Maori populations in contrast to the less westernized Polynesian Island peoples.[30]

Table 1 summarizes the findings concerning the differences in prevalence of noninfective diseases in United States and African populations.

Clues from Relationships

When two or more diseases tend to be closely associated with one another both geographically and in time of emergence, and sometimes also in individual patients; there is reason to suspect the operation of some causative factor common to both.[31-33] With the exception of appendicitis, the noninfective diseases of the large bowel—diverticular disease, both benign and malignant tumors, and ulcerative colitis—are closely associated epidemiologically, provided account be taken of the fact that appendicitis invariably becomes common in any community several decades before a major rise in frequency of any of the other noninfective bowel diseases.[11,33] Appendicitis appears to require a much shorter exposure to a new and noxious environment. It would thus seem that discovering the cause of one or more of these diseases might provide clues to the cause of the others, which may in turn throw light on the etiology of many of the diseases that are characteristic of western civilization and consequently tend to confirm an association with one another.

Epidemiological evidence clearly indicates that these diseases are more dependent on environmental than on genetic factors. Since fecal content must dominate the environment of the bowel mucosa, which in turn is largely determined by the type of food that is eaten and in particular by the fraction of this food that reaches the large bowel with the least change, this fraction deserves special attention. In any search for a common causative factor linking the noninfec-

tive diseases of the bowel, it would seem pertinent to examine, in different situations, the dietary changes that preceded by a short period the rise in the prevalence of appendicitis, and by several decades the rise in the prevalence of other bowel diseases.

Dietary Changes Preceding the Emergence of Appendicitis and Diverticular Disease

Although information is scarce and somewhat contradictory, it is estimated that in Britain and the United States before about 1890, some 450 gm of bread containing about 0.35% of fiber was consumed per person per day, giving a daily crude fiber intake of about 1.5 gm.[34,35] The introduction of new milling techniques, and in particular of the roller mills in 1870 that subsequently replaced most of the stone mills, resulted in greater availability of a low extraction-rate flour with a low fiber content, which in white bread today does not exceed 0.15%. The amount of bread consumed has fallen to less than one third that consumed before 1890, so that fiber intake from this source has probably fallen by more than 80%. The reduction in consumption of cereals in the form of porridge has also diminished, so that cereal fiber intake has probably fallen to one tenth of the pre-1870 figure.[34,36,37] Between 1889 and 1961, sugar consumption has more than doubled, but fat consumption has risen by not more than 20%.[35] Although consumption of fruit and vegetable fiber has increased, these sources apparently provide much less effect on bowel physiology than does cereal fiber.[38]

Similar dietary changes have occurred in Japanese immigrants to Hawaii. Not only has the proportion of carbohydrate to fat and protein fallen, but more refined carbohydrate has replaced less-refined carbohydrate as noted by Bennett et al[39] and W. Haenzel (written communication, 1971).

It has been extensively documented that the occurrence of appendicitis was rare in Africans before they supplemented their diet with western food.[6,7,40] In South Africa, Walker et al[7] found the following prevalences of appendectomy in senior pupils and students 18 to 20 years of age: rural Negroes, 0.5%; urban Negroes, 1.4%; urban whites, 16.5%. Lubbe[41] has shown that when South African Negroes move from a rural to an urban environment, their intake of fiber falls to one fifth of its previous total. Much of the traditional carbohydrate food is replaced by white bread; moreover, there is a considerable increase in the consumption of sugar, fat, and meat.

The Effect of Food-Fiber on the Gastrointestinal Tract

Fiber has been a largely neglected component of food, mainly because it contributes little nutritionally. Its nature has been misunderstood and its important role in maintaining normal gastrointestinal function has not been appreciated. It is of obvious importance to estimate the effect of dietary fiber on both intestinal transit times and the nature and content of feces.

Various methods are available for measuring the transit time of food residues from mouth to anus. Millet seeds and carmine markers were used before barium meal studies became practicable.

Hinton et al[42] devised a method that used the ingestion of radiopaque pellets followed by roentgenography of the stools. It is more accurate than previous methods and gives reproducible results. The pellets consist of plastic impregnated with barium sulfate. These "shapes" are about the size of rice grains (cubes with sides of 2 to 3 mm). Twenty-five pellets are swallowed, usually before a main meal. Each volunteer is given five or six plastic bags that are numbered and marked to identify the subject, who passes his or her next five to six consecutive stools into the bags provided. The time at which each stool is passed is recorded. The bags are weighed and roentgenograms of them are taken, so that the number of "shapes" in each stool can be counted, and the time elapsing between swallowing and evacuating the pellets, or any proportion of them, is then measured. The "transit time" is taken to be the time required to pass 20 (80%) of the markers, for it has been found that one or two markers may not be passed for many days.

Subjects	Country	Race
Naval enlisted men and wives	U.K.*	White
Teenage boarding school pupils	U.K.	White
Students	South Africa	White
Nurses	South India	Indian
Urban school children	South Africa	African
Manor House Hospital patients	U.K.	White
Senior boarding school pupils	Uganda	African
Vegetarians	U.K.	White
Rural school children	South Africa	African
Rural villages	Uganda	African

*U.K. Indicates United Kingdom.
†Made of four different groups.

One of us[43] has modified the method of Hinton: consecutive stools are collected in cartons, weighed, and then forced in turn through a stiff copper wire sieve with the aid of a wooden spoon and water. The pellets are held back and can be counted. This method is economical, as it can be used without roentgenographic facilities. Table 2 summarizes studies carried out or initiated by the authors, in which radiopaque markers were used.

These results show inverse relationships between stool weight and intestinal transit time and also between intake of fiber and intestinal transit times (Fig 1 and 2). Those consuming high-residue diets tend to pass large, soft, often unformed stools.

The modern astronaut makes use of the observation that refining of food leads to fewer and smaller stools, and so he is fed a diet almost free of fiber; this results in extreme constipation, with five or six days elapsing between bowel actions.[44]

Effect of Restoring Fiber to Western Diets

The relationship between fiber intake and bowel behavior is confirmed

Table 2.—Transit Times of Digesta as Shown by Hinton's Method

Type of Diet	No. of Subjects	Time of Appearance of First Pellets, hr		Transit Time, hr		Weight of Stools Passed Per Day, gm		Comments
		Variation	Mean	Variation	Mean	Variation	Mean	
Refined	15	22-110	45.7	44-144	83.4	39-223	104	Shore-based personnel
Refined	9	18-103	57.4	35-120	76.1	71-142	110	Institutional diet together with cakes, sweets, etc, from school shop
Refined	100†	13-54	30.5	28-60	48.0	120-195	173	These ate more fruit than is usual in the United Kingdom
Mixed	13	9-34	27.6	23-64	44.0	...	155	Less refined diet than that of western world
Mixed	500‡	9-40	28.5	24-59	45.2	120-260	165	Partly Europeanized diet
Mixed	6	15-24	22.0	27-48	41.0	128-248	175	U.K. diet plus wholemeal bread and added bran
Mixed	27	4-54	27.6	22-118	47.0	48-348	185	Traditional Ugandan diet plus refined sugar, white bread, jam, butter and meat
Mixed	24	8-49	22.0	18-97	42.4	71-488	225	Note similarity of values to those of African groups
Unrefined	500	5-28	12.8	20-48	33.5	150-350	275	Traditional diet
Unrefined	15	4-32	19.8	19-68	35.7	178-980	470	Villagers not yet supplementing their diet with processed foods of western type

by the result of adding fiber in the form of unprocessed bran to the diet. One of us (N.S.P.) measured intestinal transit times in five British subjects who were consuming a high-residue diet with added bran to relieve the symptoms of diverticular disease. Eighty percent of the markers of these subjects were passed within 27 to 48 hours, and their daily stool weights averaged 175 gm.

Payler[45] found that by feeding school boys wholemeal instead of white bread and adding bran to their diet, intestinal transit times were reduced by a third and stool weights increased by a quarter.

Interpretation of Results in Relation to Disease

Bowel Diseases Associated With Small Stools and Prolonged Transit-Times.—*Diverticular Disease of the Colon.*—We believe this to be a deficiency disease caused by lack of fiber in the diet that results from the refining of carbohydrate foods.[3,8,19] This conclusion is consistent with the evidence that the disease is almost unknown in communities where individuals eat a high-residue diet, that a high-residue diet relieves symptoms in the majority of patients,[46] and that the condition can be produced experimentally by feeding rats and rabbits a low-residue diet.[47,48]

Appendicitis.—Half a century ago, Short[17] produced evidence that indicated that appendicitis was related to fiber-depleted foods, and this has since been substantiated by extensive epidemiological studies.[6,7] Appendicitis may well be the result of a low-residue diet that increases the viscosity of the feces. This can result in the formation of fecaliths and also, it is postulated, in excessive segmentation of the appendix, either of which may obstruct the lumen of the appendix. Obstruction might raise the intraluminal pressures sufficiently to devitalize the appendicular mucosa and to allow bacterial invasion.[6]

Benign and Malignant Tumors of the Colon and Rectum.—The following hypothesis is consistent with epidemiological and other evidence. The numbers and type of fecal bacteria found in populations in countries where there is a high incidence of bowel cancer are reported to differ from those found in stools of persons from low-risk areas. These bacterial changes, which have been postulated as related to fat intake, result in a greater degradation of bile salts to potential carcinogenic substances.[49,50] Not only would the fecal arrest and small stool-mass associated with a low fiber diet provide more time both for bacterial proliferation and for their degrading action on bile salts, but it has also been shown[51] that the addition of fiber to the diet in the form of bran inhibits the bacterial degradation of bile salts. Moreover, any noxious substances formed by lack of fiber would be held against the bowel mucosa for a prolonged time in a concentrated form.[11,33,43,52,53]

The incidence of benign tumors may be influenced by similar factors, since many authorities consider that some common factors must be involved in the induction of both benign and malignant tumors of the bowel.[54]

Diseases That May Owe Their Origin to Straining at Stool.—Cleave[55] was the first author to relate varicose veins to constipation, basing his evidence on epidemiological studies. Recently, in a prospective study, varicosities have been related to diverticular disease in individual patients.[56] This suggests that some causative factor is common to each. We believe that a possible mechanism is that intra-abdominal pressure, raised unnaturally when

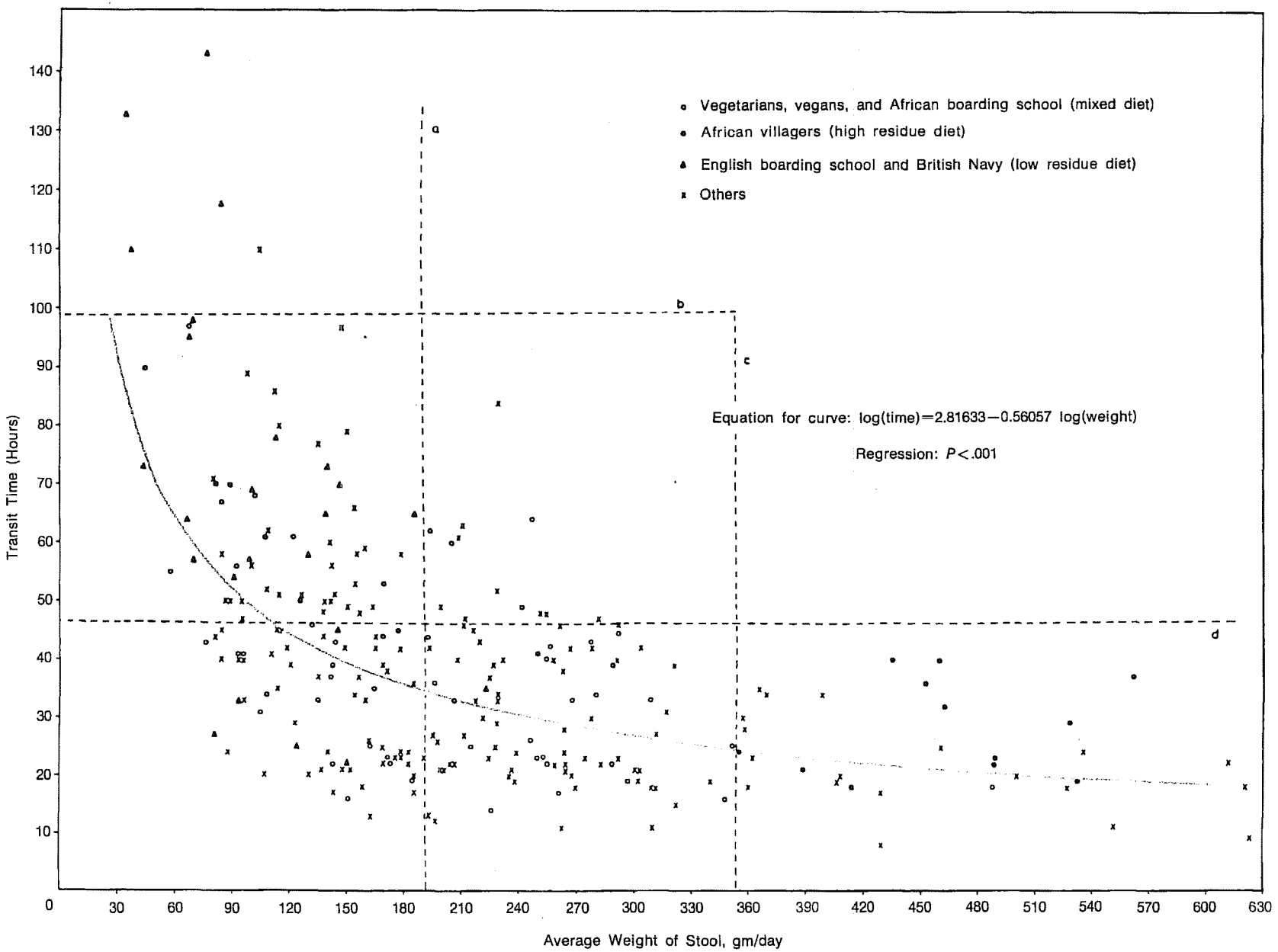

Fig 1.—Relationship between fiber intake, intestinal transit time, and stool weight.

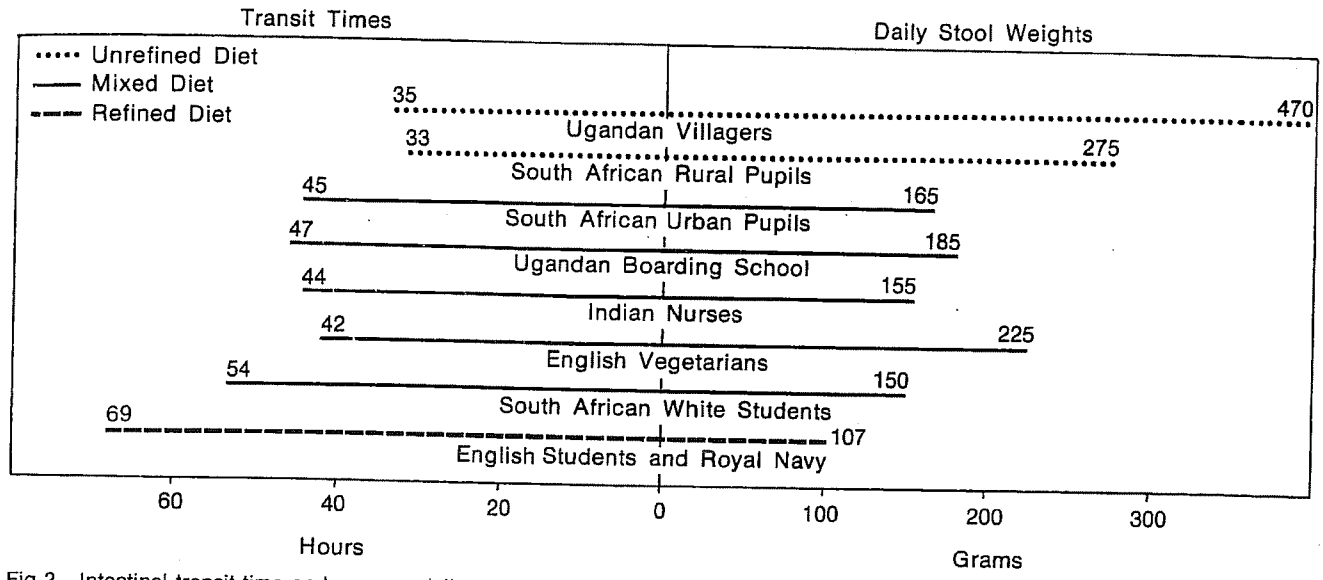

Fig 2.—Intestinal transit time and average daily stool weights in different communities.

straining at stool, could well damage the proximal valves of the leg veins. In view of the fact that raised intra-abdominal pressures are readily transmitted down the leg veins only when the valves are incompetent, intact valves must sustain these pressures.[9] Since valve incompetence is followed by dilatation and varicosity of superficial veins, it seems probable that changes may also occur in deep veins that could induce venous stasis and predispose to thrombosis during enforced recumbency. A factor that may contribute to the rarity of these venous disorders in developing countries may be the protection from raised abdominal pressures afforded to leg veins when defecation occurs in the traditional squatting position. Changes in clotting time or fibrinolysis activity may also contribute to, but would not explain, the virtual exemption from varicosity found in the upper limb.

Increased intra-abdominal pressures would also favor the dilation of the hemorrhoidal veins and might contribute to the production of hiatus hernia, a condition almost unknown in less civilized countries.[57]

Diseases That May be Indirectly Related to a Fiber-Depleted Diet.—Epidemiologically, and often clinically, these bowel diseases, venous disorders, and hiatus hernia are associated with obesity, diabetes mellitus, and coronary heart disease. Cleave and his colleagues[3] have pointed out that the removal of fiber from carbohydrate foods inevitably leads to overconsumption or too rapid absorption of the refined end-product. They have presented a great deal of epidemiological evidence that fiber-deficiency associated with excess refined cereal and sugar consumption may be the primary cause of this triad and some other western ailments.

Evidence is accumulating that shows that the removal of fiber from the diet raises serum cholesterol levels, a process that predisposes to coronary heart disease.[58,59] Intake of food rich in fiber[60] and the addition of cellulose to the diet[61] have been shown to protect against hypercholesterolemia. There is some evidence that a high-residue diet increases fecal excretion of bile acids.[62] This may account for the close association between coronary disease and diverticular disease,[63,64] both being related to the consumption of refined carbohydrate foods. These conditions are often associated with gallstones, the formation of which Heaton[20] believes to be related to refined carbohydrate diets.

Conclusions

Many of the diseases of western civilization have appeared only in the last century. We believe that they owe their origin, at least in part, to the removal of indigestible fiber from the carbohydrate foods that constitute the major part of our diet. In the past, the physiological function of this fiber has been almost completely ignored, probably because it contributes no calories and has scarcely any nutritional value. This attitude should be questioned, as cereal fiber is necessary not only for the "bulk" it provides in the intestine but also for its effect on the chemical and bacteriological processes that take place in the intestine. Evidence has been presented to show that its removal from the diet may, directly or indirectly, cause certain diseases that are becoming an increasing problem in western countries.

References

1. Burkitt DP: Some diseases characteristic of modern western civilization. *Br Med J* 1:274-278, 1973.

2. Trowell HC: *Non-infective Diseases in Africa.* London, England, Edward Arnold (Publishers) Ltd, 1960.

3. Cleave TL, Campbell GC, Painter NS: Diabetes, Coronary Thrombosis and the Saccharine Disease, ed 2. Bristol, England, John Wright & Sons Ltd (1969).

4. Seftel HC, Dew MC, Bersohn I: Myocardial infarction in Johannesburg Bantu. *S Afr Med J* 44:8-12, 1970.

5. Schrire V: Heart disease in southern Africa with special reference to ischaemic heart disease. *S Afr Med J* 45:634-644, 1971.

6. Burkitt DP: The aetiology of appendicitis. *Br J Surg* 58:695-699, 1971.

7. Walker ARP et al: Appendicitis, fibre intake and bowel behaviour in ethnic groups in South Africa. *Postgrad Med J* 49:187-193, 1973.

8. Painter NS, Burkitt DP: Diverticular dis-

ease of the colon: A deficiency disease of western civilization. *Br Med J* 2:450-454, 1971.
9. Burkitt DP: Varicose veins, deep vein thrombosis, and haemorrhoids: Epidemiology and suggested aetiology. *Br Med J* 2:556-561, 1972.
10. Burkitt DP: Geographical Pathology related to Diet, in *The Medical Annual.* Bristol, England, John Wright & Sons Ltd, 1972.
11. Burkitt DP: Epidemiology of cancer of the colon and rectum. *Cancer* 28:3-13, 1971.
12. Scotch NA: A preliminary report on the relation of sociocultural factors to hypertension among the Zulu. *Ann NY Acad Sci* 84:1000-1009, 1960.
13. Walker ARP: Overweight and hypertension in emerging populations, editorial. *Am Heart J* 68:581-585, 1964.
14. Sinclair H: Modern diet and degenerative disease, in Waller R (ed): *Just Consequences.* London, England, Charles Knight & Co Ltd, 1971.
15. Short AR: *The Causation of Appendicitis.* Bristol, England, John Wright & Sons Ltd, 1946.
16. Treves F: *Allbutt's System of Medicine.* T. C. Allbutt (ed), New York, the Macmillan Co, Publishers, 1902, p 3.
17. Short AR: The Causation of Appendicitis. *Br J Surg* 8:171-186, 1920.
18. Elliot-Smith A: Changing patterns of Disease, in Waller R (ed): *Just Consequences.* London, England, Charles Knight & Co Ltd, 1971.
19. Painter NS: Pressures in the colon related to diverticular disease. *Proc R Soc Med* 63:144-145, 1970.
20. Heaton KW: *Bile Salts in Health and Disease.* Edinburgh, Scotland, Churchill Livingstone, 1972.
21. Wilson LB: Fatal postoperative embolism. *Ann Surg* 56:809-817, 1912.
22. Stewart MJ: Precancerous lesions of the alimentary tract. *Lancet* 2:669-675, 1931.
23. Kocour EJ: Diverticulosis of the colon. *Am J Surg* 37:430-436, 1937.
24. Boland FK: Vitamin deficiency as a factor in etiology of surgical disease of digestive system. *Ann Surg* 115:939-944, 1942.
25. Boyce FF: Acute appendicitis in the aging Negro. *Ann Surg* 133:631-643, 1951.
26. Steiner PE: *Cancer: Race and Geography.* Baltimore, Williams & Wilkins Co, 1954.
27. Quinland WS, Cuff JR: Primary carcinoma in the Negro. *Arch Pathol* 30:393-402, 1940.
28. Stemmermann GN: Patterns of disease among Japanese living in Hawaii. *Arch Environ Health* 20:266-273, 1970.
29. Wynder EL, Shigematsu T: Environmental factors of cancer of the colon and rectum. *Cancer* 20:1520-1561, 1967.
30. Prior IAM: The price of civilization. *Nutrition Today* 6:2-11, 1971.
31. Burkitt DP: Related disease—related cause. *Lancet* 2:1229-1231, 1969.
32. Burkitt DP: Relationship as a clue to a causation. *Lancet* 2:1237-1240, 1970.
33. Burkitt DP, Walker ARP, Painter NS: Effect of dietary fibre on stools and transit-times, and its role in the causation of disease. *Lancet* 2:1408-1411, 1972.
34. Robertson J: Changes in the fibre content of the British diet. *Nature* 238:290-292, 1972.
35. Antar MA, Ohlson MA, Hodges RE: Perspectives in nutrition. *Am J Clin Nutr* 14:169-178, 1964.
36. Kent NL: *Technology of Cereals.* Oxford, Pergamon Press, 1970, p 163.
37. Hollingsworth DF, Greaves JP: Consumption of carbohydrates in the United Kingdom. *Am J Clin Nutr* 20:65-72, 1967.
38. Hoppert CA, Clark AJ: Digestibility and effect on laxation of crude fiber and cellulose in certain common foods. *J Am Diet Assoc* 21:157-160 (1945).
39. Bennett CG, Tokuyama GH, Bruyere PT: Health of Japanese Americans in Hawaii. *Pub Health Rep* 78:753, 1968.
40. Janssens PG, DeMuynck A: Appendicular pathology in the African Negro. *Trop Geogr Med* 18:81-96, 1966.
41. Lubbe AM: Dietary evaluation. *S Afr Med J* 45:1289-1297, 1971.
42. Hinton JM, Lennard-Jones JE, Young AC: A new method for studying gut transit times using radio-opaque markers. *J Br Soc Gastroenterol* 10:842-847, 1969.
43. Walker ARP: Diet, bowel motility, faeces composition and colonic cancer. *S Afr Med J* 45:377-379, 1971.
44. Winitz M, et al: Evaluation of chemical diets as nutrition for man-in-space. *Nature* 205:741-743, 1965.
45. Payler DK: Food fibre and bowel behaviour. *Lancet* 1:1394, 1973.
46. Painter NS, Almeida AZ, Colebourne KW: Unprocessed bran in treatment of diverticular disease of the colon. *Br Med J* 2:137-140, 1972.
47. Carlson AJ, Hoelzel F: Relation of diet to diverticulosis of the colon in rats. *Gastroenterology* 12:108-115, 1949.
48. Hodgson J: An animal model to study diverticular disease. *Br J Surg* 59:315, 1972.
49. Aries V, et al: Bacteria and aetiology of large bowel cancer. *Gut* 10:334-335, 1969.
50. Hill MJ, et al: Bacteria and aetiology of cancer of large bowel. *Lancet* 1:95-99, 1971.
51. Pomare EW, Heaton KW: Alteration of bile salt metabolism by dietary fibre (bran). *Br Med J* 4:262-264, 1973.
52. Oettle AG: Primary neoplasms of the alimentary canal in whites and Bantu of the Transvaal, 1949-1953: A histopathological series, in *Tumours of the Alimentary Tract in Africans.* National Cancer Institute Monograph 25. Bethesda, Md, National Cancer Institute, 1967, p 97.
53. Wynder EL, et al: Environmental factors of cancer of the colon and rectum. *Cancer* 23:1210-1220, 1969.
54. Morson BC, Bussey HJR: Predisposing causes of intestinal cancer. *Curr Prob Surg,* February 1970, pp 1-46.
55. Cleave TL: *On the Causation of Varicose Veins.* Bristol, England, John Wright & Sons Ltd, 1960.
56. Latto C, Wilkinson RW, Gilmore OJA: Diverticular disease and varicose veins. *Lancet* 1:1089-1090, 1973.
57. Burkitt DP, James PA: Low-residue diets and hiatus hernia. *Lancet* 2:128-130, 1973.
58. Trowell H: Ischemic heart disease and dietary fiber. *Am J Clin Nutr* 25:926-932, 1972.
59. Trowell H: Dietary fibre and coronary heart disease. *Rev Evr Etucl Clin Biol* 17:345-349, 1972.
60. Mathur KS, Khan MA, Sharma RD: Hypocholesterolaemic effect of Bengal gram: Long term study in man. *Br Med J* 1:30-31, 1968.
61. Shurpalekar KS, et al: Effect of inclusion of cellulose in an "atherogenic" diet on the blood lipids of children. *Nature* 232:554-555, 1971.
62. Antonis A, Bersohn I: The influence of diet on faecal lipids in South African white and Bantu prisoners. *J Clin Nutr* 11:142-155, 1962.
63. Schowengerdt CG, et al: Diverticulosis, diverticulitis and diabetes. *Arch Surg* 98:500-504, 1969.
64. Trowell H, Burkitt DP, Painter NS: Aspects of the epidemiology of ischaemic heart disease and diverticular disease. *Am J Dig Dis,* to be published.