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Autonomic denervation and the origins of chronic Western diseases

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SUMMARY

Many chronic Western diseases result from lifestyles that include refined diets, poor bowel habits, limited physical exercise and suboptimal patterns of childbirth. Western diets result in reduced stool weights, increased bowel transit times and persistent physical efforts during defaecation. Prolonged physical efforts during defaecation and childbirth cause latent, or direct, injuries to branches of the cardiac (thorax), coeliac (abdomen) and hypogastric (pelvis) plexi. Injuries to autonomic nerves result in impaired visceral function including visceral dysmotility, tissue hypoplasia and hyperplasia, increased susceptibility to infection, and, aberrant reinnervation with sensitisation of the central nervous system (CNS). These unrecognised injuries are vulnerable to the long list of causes of autonomic Dysfunction, e.g. stress, alcohol, drugs, infection, trauma, cancer, etc. Specific injuries at different anatomical locations in midline autonomic pathways give rise to a wide range of Western diseases from infancy to old age, through diverse and cumulative mechanisms.

Introduction

D.P. Burkitt recorded different distributions of chronic disease in Western and non-Western societies in association with marked differences in diet and stool weight [1,2], (Table 1). He was unable to define the underlying relationships between refined, high-calorie Western diets and diverse Western disease. Typical Western diets result in increased, bowel times (80 h vs. 34 h), reduced stool weights (110 g vs. 454 g each day) and constipation [1]. "Constipation" may mean small or hard stools, infrequent passage of stools, or, persistent, physical efforts during defaecation [3], though most clinical studies do not distinguish these different interpretations. Overall, it affects 2–27% of Western adults and is more prevalent in women than men, children than adults, and, in the elderly [4]. Physical efforts during defaecation complicate 20–30% of Western bowel movements with 1% of adults opening their bowels less than once each week, and, 0.3% less than once each month [5]. Rates of constipation between the sexes are roughly equal until adolescence when there is a progressive and sustained increase in female constipation through the reproductive years into old age [4].

Sympathetic nerves, arising from thoracolumbar segments of the spinal cord (T1-12, L1-2), pass through paravertebral, sympathetic chains to visceral plexi while parasympathetic nerves have craniosacral origins from four cranial nerves (III, VII, IX, X) and three sacral segments of the spinal cord (S2–S4). Some organs receive autonomic nerves directly from the sympathetic chain and the vagus nerve while others pass through intervening plexi and ganglia with, or without, synapses (Fig. 1). The three great autonomic plexi are the cardiac (thorax), coeliac (abdomen) and hypogastric (pelvis) that supply extrinsic innervation to their respective viscera. Intrinsic visceral innervation varies from the complex interactions of Auerbach's, Meissner's and Henle's plexi in small bowel to the subserosal and submucosal plexi in solid organs such as uterus and prostate. Immersion in formalin destroys fine, autonomic nerves so that many, twentieth century physicians have been unfamiliar with the fine, anatomical detail of the major autonomic plexi described by Robert Lee and other, nineteenth century anatomists [6,7] (Fig. 2a–c).

Autonomic dysfunction results from a long list of possible causes including alcohol, drugs, trauma, infection, cancer, etc., that has not previously included the complications of persistent physical efforts in childbirth or defaecation [8], (Table 2). Many of these traditional causes will exacerbate the effects of specific injuries to autonomic nerves. New, immunohistochemical reagents provide reliable markers for denervation or subsequent, aberrant reinnervation that have been recorded in many, unrelated and unexplained, subspecialist contexts in recent years. Description of the range of gynaecological disease resulting from injuries to branches of the hypogastric plexi in the female pelvis offers a template for the consequences of denervation and reinnervation in cardiac, coeliac, and secondary autonomic plexi.

The inferior hypogastric plexus and gynaecological disease

Pre-aortic, nerve plexi divide into bilateral superior hypogastric plexi that converge on large, multilayered, nerve plates on the

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Table 1
D.P. Burkitt's diseases of Western civilization, BMJ 1973;1:274–278.

Diseases of the bowel
Appendicitis, diverticular disease, ulcerative colitis, polyps, cancer of the large bowel
Diseases of cholesterol metabolism
Coronary disease, gallstones, obesity
Raised intra-abdominal pressure
Hiatus hernia, haemorrhoids, herniae, varicose veins
Venous disorders
Deep venous thrombosis, pulmonary embolism
Autoimmune diseases
Type 1 diabetes, thyrotoxicosis, pernicious anaemia, rheumatoid arthritis, multiple sclerosis

pelvic side wall (inferior hypogastric plexi) before distributing nerves to the pelvic viscera (Fig. 2a). Large bundles of autonomic nerves converge on the junction of the uterus and vagina where they are susceptible to injury, particularly during childbirth. Two forms of injury to pelvic autonomic nerves result in two patterns of aberrant reinnervation; chaotic proliferative reinnervation and perivascular nerve fibre proliferation (Fig. 3a–d). In the first pattern, intrapartum injuries to pelvic nerves as they enter the uterine isthmus, result in re-growth of nerves from the proximal stump, and, chaotic patterns of aberrant reinnervation [9–12]. Some years later light touch causes pain or discomfort (allodynia) (Fig. 3b). Typical "allodynic" symptoms include some forms of vulvodynia [14], dyspareunia [15], chronic pelvic pain [16,17], dysmenorrhoea [18], rectal hypersensitivity [19] and irritative bladder symptoms [20,21] i.e. benign gynaecology. In the second pattern, persistent, physical efforts during defaecation produces perivascular nerve fibre proliferation (Fig. 3d). Injured nerves regrow along blood vessels encasing them in multiple, circumferential layers of abnormal nerves. Symptoms relate to increases in blood flow during the second half of the menstrual cycle resulting in premenstrual uterine, vulval, vaginal and vesical pain [11]. Similar patterns occur in myocardium [22], nasal turbinates [23] and intervertebral discs [24].

Injuries at different sites in the lower genital tract result in different forms of gynaecological pathology. Focal injuries to

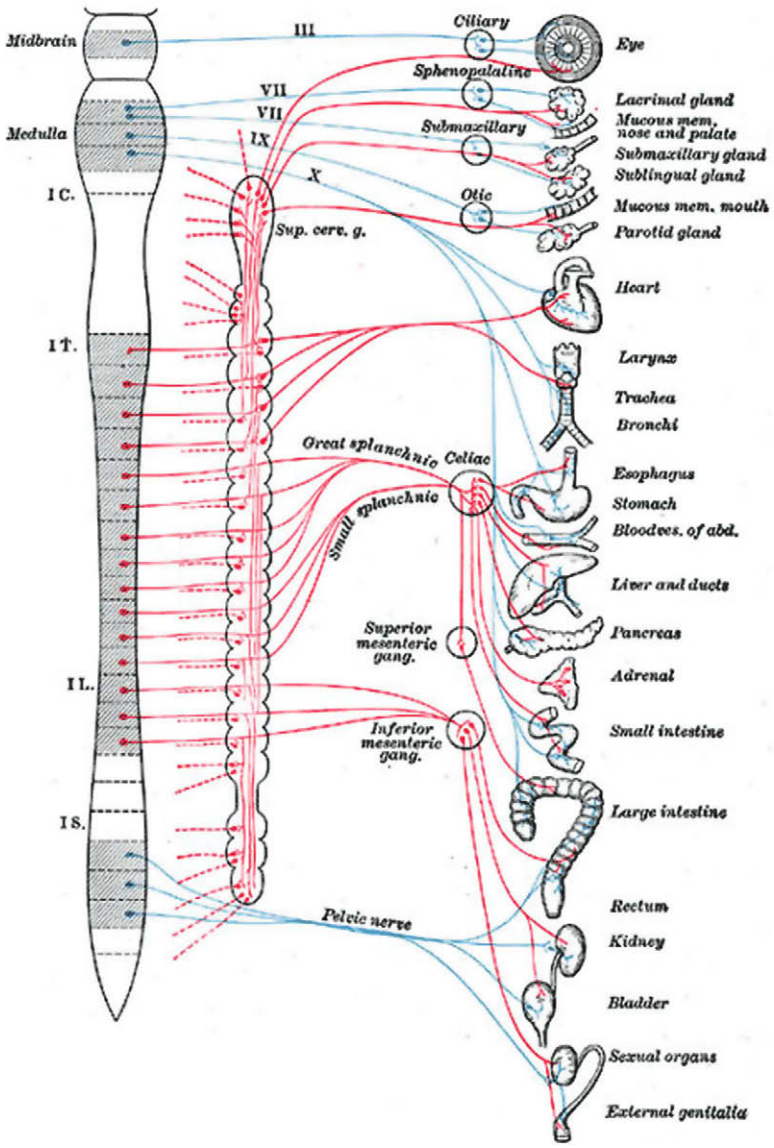

Fig. 1. Neuroanatomical "wiring" of the autonomic nervous system. Parasympathetic nerves have a craniosacral outflow (III, VII, IX, X) whilst sympathetic nerves have thoracolumbar origins (T1-12, L1-2). Sympathetic nerves may, or may not, synapse in the sympathetic chain; intermediary neurons may diminish, or amplify, the effects of sympathetic stimulation, parasympathetic nerves tend to synapse in, or adjacent to, the viscus. Some autonomic nerves have short anatomical courses, e.g. pancreatic nerves, whilst those supplying the small bowel traverse the mesentery, and, those supplying pelvic viscera traverse pre-aortic plexi and the pelvic side wall.