high dust levels. These dust levels can be compared with 1 743 personal air samples collected between 1976 and 1981 from 205 foundries in the USA.[22] The median respirable dust level in the USA was 0,90 mg/m³ with a median percentage quartz in this dust of 9%. Our data, collected a decade or more later, show a foundry median for respirable dust of 1,9 mg/m³ which suggests that South African foundries had not yet attained USA standards of the 1970s.

Silicosis prevalences (Table IV) have the same generalisability constraints as do the dust measurements. The wide range of disease prevalence (0 - 38% for longer service workers) may be explained in part by factors inherent in the production processes (e.g. extent to which sand moulds are used) or by employment policies such as dismissal of workers with silicosis. Of importance is that most of the workers with pneumoconiosis identified were undiagnosed prior to the surveys and working in high-dust sections despite having pneumoconiosis.

In conclusion, this study provides convincing evidence of neglect of occupational health by the foundry industry. Inadequate dust control and monitoring of exposed workers are the norm and it is reasonable to conclude that conditions and hazards less well known than pneumoconiosis and dust receive even less attention.

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REFERENCES

1. American Conference of Governmental Industrial Hygienists. *TLVs Threshold Limit Values and Biological Exposure Indices for 1992 - 1993.* Cincinnati: ACGIH, 1992.
2. South African Institute of Foundrymen. *Foundrylog '89.* Johannesburg: South African Institute of Foundrymen, 1989.
3. Health and Safety Executive. *General Methods for Gravimetric Determination of Total and Respirable Inhalable Dust* (MDHS 14). London: HMSO, 1986.
4. Bradley AA. The determination of quartz in small samples by an x-ray technique. *Journal of Scientific Instruments* 1967; **44:** 287-288.
5. International Labour Office. *Guidelines for the Use of the ILO International Classification of Radiographs of Pneumoconioses.* Rev. ed. Geneva: International Labour Office, 1980.
6. National Centre for Occupational Health. *Interim Report on a Survey of Employees and the Working Environment in a Large Iron and Steel Foundry* (Report No. 11/83). Johannesburg: NCOH, 1983.
7. National Centre for Occupational Health. *Feedback on Foundry Respiratory Survey* (Report No. 50/86). Johannesburg: NCOH, 1986.
8. National Centre for Occupational Health. *Report on Environmental Conditions at a Foundry* (Report No. 8/86). Johannesburg: NCOH, 1986.
9. National Centre for Occupational Health. *Report on Respirable Dust Exposure at a Foundry* (Report No. 25/86). Johannesburg: NCOH, 1986.
10. National Centre for Occupational Health. *Report on Airborne Particulate Exposure at a Foundry* (Report No. 26/86). Johannesburg: NCOH, 1986.
11. National Centre for Occupational Health. *Report on Airborne Particulate Exposure at a Foundry* (Report No. 27/86). Johannesburg: NCOH, 1986.
12. National Centre for Occupational Health. *Report on Airborne Respirable Dust at a Foundry* (Report No. 32/86). Johannesburg: NCOH, 1986.
13. National Centre for Occupational Health. *Report on Dust Exposure at a Foundry* (Report No. 35/87). Johannesburg: NCOH, 1987.
14. National Centre for Occupational Health. *Report on Dust Exposure at a Foundry* (Report No. 33/92). Johannesburg: NCOH, 1992.
15. National Centre for Occupational Health. *Dust, Noise and Gas Levels at a Foundry* (Report No. 40/92). Johannesburg: NCOH, 1992.
16. Myers JE, Garisch D, Myers HS, Cornell JE. A respiratory epidemiologic survey of a small South African foundry. *Am J Ind Med* 1987; **12:** 1-9.
17. National Centre for Occupational Health. *Report on a Survey Conducted at a Foundry* (Report No. 2/89). Johannesburg: NCOH, 1989.
18. National Centre for Occupational Health. *Report on Radiological Surveys at Four Foundries* (Report No. 4/93). Johannesburg: NCOH, 1993.
19. National Centre for Occupational Health. *Report on Radiological Survey at a Foundry* (Report No. 7/93). Johannesburg: NCOH, 1993.
20. Lowe RE, Barron P, Steyn SD, Malekela LL, Steinberg MH, Reid G. Occupational health services in Johannesburg and Randburg. *S Afr Med J* 1990; **77:** 581-584.
21. Kistnasamy B, Yach D. Tuberculosis in commerce and industry in a western Cape suburb, South Africa, 1987. *Am J Ind Med* 1990; **18:** 87-93.
22. Oudiz J, Brown JW, Ayer HE, Samuels S. A report on silica exposure levels in United States foundries. *Am Ind Hyg Assoc J* 1983; **44:** 374-376.

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HISTORY OF MEDICINE

Constipation, purgatives or the knife?

A 19th-century dilemma

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Research into the history of laxative abuse requires analysis of prevailing attitudes towards, and definitions of, constipation, both 'lay' and 'professional'. A review of the medical literature of the last decade of the 19th century reveals concern with the causes and treatment of constipation, and while there are cautions against the indiscriminate use of laxatives, no studies were found on laxative abuse as such.

Constipation was found in about 60% of patients, was more common in women,[1] and affected young and old, 'peasant' and 'potentate', although it was 'somewhat aristocratic in its tendencies, showing a special fondness for the highborn and the wealthy, not to be wondered at when their diet and inactive habits are remembered'. Furthermore, their luxurious water-closets distracted them from 'exclusive attention to the act', and 'calls to stool' were possibly neglected or postponed by 'false modesty'.[2] Was this 'aristocratic tendency' noted merely because the 'highborn' could afford and therefore more often consulted the doctor?

There was general agreement by the profession that constipation was caused by diminished muscularity and mucosal secretions of the bowel and defective innervation, and most authors recognised the primary roles played by diet and exercise. Constipation was defined as an 'abnormal condition of the great colon and entire intestinal tract manifested by unusual retention of excrementitious material both as to quantity and time of evacuation',[1] or 'modification of the alimentary canal resulting in the retention of faecal matter'.[2]

However, there was not consensus about regularity, and while some doctors accepted a wide range of normal function, claiming that 'remarkable differences in individual habit may not be incompatible with ordinary health',[2] there were hardliners who insisted on 'an easy regular movement every morning, so soon as they have breakfasted', making those persons 'meek, affable, gracious, kind, and "no" from their mouth comes with more grace than "yes" from the mouth of one who is constipated'.[1]

Dr E. S. Pettyjohn[1] further alarms the public by declaring that 'since the fecal matter is toxic, how can the guilty (those who do not believe in defecation), or even the innocent escape destruction?' One wonders what effect those moral judgements of one's bowel habits must have had on the constipated — committing them shamefacedly to lifelong surreptitious addiction to Merrie & Bright laxative sweets (containing phenolphthalein), Allen & Hanbury's

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Bynocascada, Bishop's Carlsbad Sulphate of Soda, Laymons' Castoria, Karlsbader Sprudelsalz, McKesson & Roberts's refreshing and mild fruity laxative (Analax), Hawley's Cascara, Bishop's granular effervescent citrate of magnesia, JBL cleansing powder for internal bathing, or H. K. Mulford Co.'s Cremo Magnesium. Such a wholesome and harmless-sounding menu must have appealed to a public made avid for moral and physical cleanliness. The few records of adverse side-effects found during that decade included a reference[3] in the *BMJ*, by the Director-General of the Medical Department of the Navy, to the mechanical or 'shot bag' treatment which he tried because he found constipation 'a very frequent annoyance for purgatives soon lose their usual effect, and when given stronger often bring on troublesome diarrhoea'.

Another reference by Thomas D. Savill[4] in the *Lancet* describes the plight of 250 patients in the Sick Wards of the Paddington Workhouse who suffered from particularly obstinate constipation, which was attributed to the routine administration of compound rhubarb pills distributed wholesale by the attendants. 'This medication after temporarily relieving the constipation was usually succeeded by constipation more pronounced than before; a chronic constipation which bye-and-bye, in the course of a few months, defied all but the most violent purgatives.' It would be instructive to know what facilities these poor patients had for exercise and how conducive their diet and toilets were to the 'divulsion of the sphincters'.

Constipation was incriminated in the nutrition of the brain and the nervous system, supplying infective nutriments to the brain, in the production of cerebral anaemia and nervous disability followed by brain exhaustion, in the production of retrograde changes in the quality of the blood and diminution of the red blood corpuscles, and in creating conditions favourable for the maintenance of putrefaction and atrophy, degeneration in the nerve substance itself, and even transient mania.[1] Small wonder that the laxative industry responded enthusiastically to a market clamouring for relief from 'intestinal auto-intoxication'.

Dr A. B. Cooke[2] observes in 1897 that constipation is the great curse of 'modern life' but that it is a preventable disease. He advocates more concern from the profession and education of the laity. The aetiologies he claims are diet, leaving too much or too little residue, and overindulgence in tea and alcoholic beverages; the hurry and turmoil of modern life, where disease is the harvest of neglect; defective innervation, decreased peristalsis or diminution of secretion; habitual use of drastic purgatives, which is alarmingly prevalent and increasing (the drugs of this class merely serve to increase the condition which calls for their use); diseased or debilitated states of general health; mechanical obstruction (he places malignant growths in the 'list of rectal diseases for which constipation is at least in a sense responsible'); and painful affections of the rectum and anus, 'from which human nature shrinks'.

The noteworthy reference to the habitual use of drastic purgatives as the cause of disease needs to be elaborated on, as there are other cautionary messages in the literature of that time. Thomas M. Southworth, M.D.,[5] advocates in his lecture on the treatment of habitual constipation in infancy that dietetic measures are the best, and that 'temporary relief or masking of symptoms by therapeusis, when the aetiological morbid condition may be permanently removed by more painstaking application to our problem, approaches dubiously near to Charlatanism', and cautions that 'the abuse of enemata and purgatives, will eventually diminish the sensitivity of the mucous membrane and produce atony of the muscular coats of the intestine'.

Once diet and habits of life have been corrected Cooke[2] advocates the use of purgatives, although 'their proper field of application is a very limited one. It may be tersely expressed: In the beginning, to relieve the costive condition, afterward, only when it recurs and other methods fail. Wisely selected, medicines are useful, but not to be relied upon.'

Pettyjohn[1] insists that massage, gymnastics, faradic and galvanic currents and hydrotherapeutics produce constitutional and local effects and more satisfactory results than medicines, and that when the patient recovers, he stays well!

The hardliner George Herschell[6] (an advocate of the daily evacuation) recommended careful history-taking to get to the bottom of the problem. He commenced treatment by making certain that the large intestine was empty. He expedited this with hot soup and water enemas, olive oil and ox-gall solvents or continuous irrigation with a Turck's double colon tube. He proceeded with douches, cascara, belladonna and electrodes, and finally, in order to restore 'tone to the muscular and nervous tissues of the bowel itself', he prescribed hydrotherapy, electricity, massage and its substitutes, gymnastics, drugs and regulation of the diet with lots of fibre.

In the maintenance of 'these cases of constipation only, which are complicated with the dilated or atonic stomach, it is good scientific practice to give a daily laxative drug'; he adds that 'in the treatment of chronic constipation drugs are of extreme use', and categorises them into five groups. Those which relieve spasm, excite peristalsis, increase the secretions of the gastro-intestinal tract and have a beneficial effect upon the neurasthenic condition he distinguishes from laxatives, which, he says, must only be used in special circumstances. 'We must make up our minds to abandon the use of purgatives.'

Unfortunately the early warnings against laxative abuse cast the constipated between the devil and the deep blue sea, where some fell into the hands of surgeons busying themselves with justifying the surgical treatment of constipation. For example, after several unsuccessful surgical procedures on a young but 'feeble and emaciated' woman at Guy's Hospital, Mr Arbuthnot Lane[7] decided 'to open the ileum into the sigmoid, and so throw the remainder of the large bowel out of use'. In 1896 the Professor of Surgery at the New York Post-Graduate Hospital, Charles B. Kelsey,[8] described the 'weak and dilated sphincter' of an incontinent lady 'in the higher social scale, being a person of intelligence accustomed to taking the best possible care of her health'. This lady in napkins 'blames the doctor who operated upon her for the stricture of the rectum which she never had'.

Finally, one returns to the recommendations of Cooke[2] in some despair and not a little confusion. It is unlikely that his call for education of the laity would have produced the desired effect that 'purgatives will give place to intelligent prophylaxis', considering the near-hysteria surrounding the concept of auto-intoxication, prevailing Victorian mores, the mystical power of the medical language with its moralising tone, and the availability of an array of 'harmless' and 'refreshing' laxatives.

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REFERENCES

1. Pettyjohn ES. Constipation: some of its effects and its non-medicinal treatment. *JAMA* 1896; **27:** 247-249.
2. Cooke AB. Constipation and rectal diseases. *NY Med J* 1897; 11 Sep: 356-359.
3. Beatty HB. The mechanical treatment of constipation. *BMJ* 1892; **1 Oct:** 735.
4. Savill TD. A note on the therapeutics of saline laxative mineral waters. *Lancet* 1895; **23 Nov:** 1286-1288.
5. Southworth TS. The causes and treatment of habitual constipation in infancy. *Arch Paediatr* 1898; **15:** 418-426.
6. Herschell G. Constipation and its modern treatment. *Clin J* 1898; **13** (3, 9 Nov): 41-112.
7. Lane A. The Operating Theatres, Guy's Hospital. Operation for chronic obstruction produced by constipation. *The Medical Press & Circ* 1903; **126** (76, 20 July): 118.
8. Kelsey CB. The surgical treatment of constipation: a clinical lecture at the New York Post-Graduate Hospital. *NY Med J* 1896; 16 May: 633-634.

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Fatal *Capnocytophaga canimorsus* (DF-2) septicaemia

A case report

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A 45-year-old man died 2 months after being bitten on the hand by a dog. He developed the rare but characteristic clinical picture of fulminant septicaemia and peripheral gangrene caused by a Gram-negative bacillus, *Capnocytophaga canimorsus,* previously known as dysgonic fermenter type 2 (DF-2), which is an occasional commensal in the oral flora of dogs and cats. This disease must be anticipated and dog bites appropriately managed to avoid the mortality associated with infection by this micro-organism. Initial treatment includes appropriate prophylactic antibiotics and debridement, while early exchange transfusion and emergency amputation may be of value in fulminant cases.

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Dysgonic fermenter type 2 (DF-2) was an earlier name given to a slow-growing Gram-negative bacillus transmitted by dog bite and producing fulminant septicaemia with marked peripheral ischaemia and a high mortality rate.[1] This organism has recently been renamed *Capnocytophaga canimorsus,*[2] and severe infection may be more common than previously reported. The disease, first described by Butler[3] in 1977, is characterised by the history of a dog bite followed by the clinical features of a malar rash, peripheral ecchymosis, symmetrical gangrene, disseminated intravascular coagulopathy and acute tubular necrosis with renal failure. The organism must be identified on a peripheral blood smear, since it is difficult to culture using routine culture methods.[4] This case highlights the potentially catastrophic result of an apparently insignificant dog bite and delayed medical intervention.

Case report

A 45-year-old previously healthy man, resident in Cape Town for only 1 month, was admitted to Groote Schuur Hospital in multiple organ failure. He had a history of heavy smoking and ethanol consumption for many years. One week before admission he had sustained a seemingly insignificant dog bite to the 5th digit and hypothenar eminence of his right hand. He had not sought medical attention for this injury and it was not the stated reason for his presentation to hospital. The puncture wounds on the hand were not obviously septic and no other septic focus was identified. He reported mild myalgia and fever for a few days, but his condition had deteriorated acutely during the 24 hours before admission. He had experienced profuse watery diarrhoea with approximately 15 bowel actions during the day before admission, associated with nausea, vomiting, abdominal discomfort, fever and dizziness. In addition he had noted numbness of the fingers and toes, a petechial rash on the legs, and haematuria.

On presentation the patient was ill, shocked, clinically dehydrated, deeply jaundiced, and had peripheral ischaemia. There was also evidence of recent spontaneous bleeding with petechial lesions on the forearms and thighs. He had obvious vascular compromise of the nose, fingers and toes, manifest by a well-demarcated bluish discoloration, hypothermia, a prolonged capillary filling time and decreased sensation with areas of hyperaesthesia. Most severely affected was the 5th digit of the right hand, with early gangrene. He had tachycardia and was hyperventilating in response to metabolic acidosis. Initial arterial blood gas analysis revealed pH 7,4, partial arterial oxygen tension (Pa_{O_2}) 20,4 kPa on a fractional inspired oxygen concentration (Fi_{O_2}) of 0,4, partial arterial carbon dioxide tension (Pa_{CO_2}) 2,6 kPa, standard bicarbonate 16,4 mmol/l and base excess –10,2. The lungs were clinically and radiographically normal. The abdomen was tender without evidence of peritonism. Palpation revealed a 6 cm firm, tender, non-pulsatile hepatomegaly with a 2 cm splenomegaly. The total bilirubin value was 215 µmol/l, conjugated bilirubin 68 µmol/l, alkaline phosphatase 108 IU/l, aspartate aminotransferase 1 141 IU/l and alanine aminotransferase 339 IU/l. Urinary catheterisation produced 10 ml of heavily bloodstained urine. The serum urea value