Medical History

Dalzeil's disease—66 years on

JAMES KYLE

British Medical Journal, 1979, **1**, 876-877

"I have pleasure in drawing your attention to this condition, which, I think, has not yet been fully described." With these words T Kennedy Dalzeil drew the attention of members of the British Medical Association to the intestinal disease that he called chronic interstitial enteritis.[1] The address, accompanied by a demonstration of resected specimens, was given to the Annual Meeting of the BMA in Glasgow in July 1913. It is clear from his opening statement that Dalzeil (figure) realised that he was describing a new condition. Towards the end of his paper he again states: "As far as I know the disease has not been previously described." If credit for discovering a new disease should be given to the person who first recognised it as a separate entity, then Dalzeil should be accepted as the discoverer of transmural chronic inflammatory bowel disease. The first world war broke out a year after Dalzeil had described chronic interstitial enteritis. The condition was apparently lost sight of until almost 20 years later (in 1932) it re-emerged under a different name before the American Medical Association in New Orleans.[2]

Discovery and rediscovery

T Kennedy Dalzeil (pronounced dee-yell) was born at Penport, Dumfriesshire, in 1861. He attended school there and later went to study medicine at Edinburgh University, where he graduated in 1883. Dalzeil became house surgeon to Sir William Macewen at the Royal Infirmary, Glasgow, in 1884. Five years later he was appointed to the staff of the Western Infirmary. He was with the 3rd Scottish General Hospital in the first war, and there clearly was more concerned with gunshot wounds than inflammatory bowel disease. He received a knighthood in 1917 and died seven years later.

Dalzeil's description of the condition that later mistakenly became known as Crohn's disease is both comprehensive and surprisingly modern. He saw his first patient, a doctor, in 1901; Crohn did not see his until 1930. Dalzeil had operated on nine patients, while the Mount Sinai workers had collected 14 examples. Eponymous fame does not, however, depend on the number of cases reported. Conn[3] described only one patient with primary aldosteronism, while Zollinger and Ellison[4] had dealt with only two patients with the syndrome that perpetuates their names. Dalzeil operated on all his patients himself. For some reason that has never been clear,[5] the surgeon, A Berg, who operated on 13 out of the 14 New York cases, declined to have his name associated with the 1932 paper that reawakened interest in Dalzeil's disease. The lesions recognised at the Mount Sinai Hospital were all in the terminal ileum, while Dalzeil had examples in jejunum, middle and lower ileum, and transverse and sigmoid colon. In his two fatal cases both large and small intestines were extensively affected. Dalzeil's clear description of lesions in the colon is noteworthy. Morson's classical account of so-called Crohn's disease[6] of the colon was not to appear until nearly half a century later.

Gastroenterology Research Unit, Royal Infirmary, Aberdeen AB9 2ZB

JAMES KYLE, DSC, FRCS, consultant surgeon

Sir T Kennedy Dalzeil, FRFPSG (1861-1924). (The portrait originally appeared in the *Glasgow Medical Journal*, 1924, and is reproduced by courtesy of the editor of the *Scottish Medical Journal*.)

Aetiology and symptomatology

It was when discussing aetiology that Dalzeil gave the memorable description of the macroscopic appearance of the intestine: "The affected bowel gives the consistence and smoothness of an eel in a state of rigor mortis, and the glands, though enlarged, are evidently not caseous." Tuberculosis was rife in Glasgow and New York. Both Dalzeil and, decades later, Crohn were at pains to exclude it as the cause of the lesions that they observed. In 1907 McFadyean[7] gave an account of Johnne's disease in cattle, and noted an acid-fast bacillus in the animals. Dalzeil followed up this veterinary discovery and decided to keep an open mind about the role of a long-acting organism. The search for such bacterial variants or particles is still being pursued.[8 9]

For nearly 25 years after Hadfield[10] in 1939 gave his detailed account of the histological changes in chronic interstitial enteritis, few pathologists were willing to diagnose the condition unless granulomas and giant cells could be detected. Hadfield

had not intended such a rigid interpretation to be adhered to, but a static view of the inflammatory process developed. Sixty-six years ago Dalzeil stated that his various specimens "form a graded series in which all the stages from acute to chronic may be traced." This concept of a spectrum of changes is universally accepted today. Unfortunately, we do not know the names of the Glasgow pathologists who examined Dalzeil's specimens. Crohn and his colleagues described lesions in the terminal ileum. The proportion of patients with isolated ileal lesions has diminished in Scotland in recent years, while the proportion with extensive colonic disease or with disease affecting both parts of the intestinal tract has increased. The sites of disease today resemble those observed in Glasgow during the early years of this century.

Diarrhoea is the commonest symptom of Dalzeil's disease, followed by intestinal colic and loss of weight. From reading the case histories provided by Dalzeil, and the fuller, and more orthodox, description of symptomatology given by the Mount Sinai Hospital workers, it is clear that both groups were seeing patients at a more advanced stage of the disease than would be the case nowadays. Many of Dalzeil's patients had subacute intestinal obstruction. All those included in Crohn's series apparently had masses in the right iliac fossa and nearly half of them had developed internal fistulae. In addition, an unknown number had external fistulae on to the abdominal wall; this complication is not mentioned by Dalzeil, but a master surgeon does not expect to produce such fistulae.[11]

Prognosis and treatment

When considering prognosis and treatment Dalzeil stated: "As far as I am aware the prognosis is bad except in cases where the disease is localised, and even then seems rather hopeless unless operation be had recourse to. . . ." He advocated radical resection for localised disease. In a postscript to his main report Dalzeil mentioned a woman on whom he had performed a total colectomy for chronic inflammatory bowel disease. He was proposing, as a second stage, to anastomose the caput caeci to the rectum—that is, the Oxford operation, the details of which were given by Truelove *et al* in 1965.[12]

Although the years of the peak incidence of Dalzeil's disease may have passed, all gastroenterologists and surgeons must share the hope expressed by Dalzeil in his concluding remarks 66 years ago: "I can only regret that the aetiology of the condition remains in obscurity but I trust that ere long further consideration will clear up the difficulty."

References

1 Dalzeil, T K, *British Medical Journal*, 1913, **11**, 1668.
2 Crohn, B B, Ginsburg, L, and Oppenheimer, G D, *Journal of the American Medical Association*, 1932, **99**, 1323.
3 Conn, J W, *Journal of Laboratory and Clinical Medicine*, 1955, **45**, 3.
4 Zollinger, R M, and Ellison, E H, *Annals of Surgery*, 1955, **142**, 709.
5 Crohn, B B, *Gastroenterology*, 1967, **52**, 767.
6 Lockhart-Mummery, H E, and Morson, B C, *Gut*, 1960, **1**, 87.
7 McFadyean, J, *Journal of Comparative Pathology and Therapeutics*, 1907, **20**, 48.
8 Parent, K, and Mitchell, P, *Gastroenterology*, 1978, **75**, 368.
9 Sharon, P, and Ligumsky, D, *Gastroenterology*, 1978, **75**, 632.
10 Hadfield, G, *Lancet*, 1939, **2**, 773.
11 Atwell, J D, Duthie, H L, and Goligher, J C, *British Journal of Surgery*, 1965, **52**, 966.
12 Truelove, S C, Ellis, H, and Webster, C W, *British Medical Journal*, 1965, **1**, 150.

(*Accepted 16 January 1979*)

A 16-year-old girl who is asking for oral contraception has a father with type 4 hyperlipidaemia and hypertension; he had a stroke when aged 35. A maternal uncle also has type 4 hyperlipidaemia and the paternal grandmother died of heart disease in her 50s. The girl herself has normal cholesterol and triglycerides. What advice should I give her?

The question is really in two parts. Does the 16-year-old girl have a genetic predisposition to hypertriglyceridaemia and, if so, will oral contraceptives have an adverse effect on this? In putative monogenic hypertriglyceridaemia, phenotype expression in young progeny of affected individuals is less than would be expected (25% of expected frequency),[1] therefore the finding of normal lipid levels in a 16-year-old girl does not rule out the presence of an abnormal gene. In answer to the second part, it has been shown in some older women that where mild hypertriglyceridaemia exists oral contraceptives exacerbate this condition to produce a grossly hypertriglyceridaemic state.[2] Even in apparently normolipaemic subjects, oral contraceptives produce a rise in mean triglyceride levels of up to 48% in young women[3]; this is thought to reflect the dosage and nature of the oestrogenic component. For this girl, with a family history of ischaemic heart disease as well as hypertriglyceridaemia, it would be wise to monitor triglyceride and cholesterol levels before and after starting an oral contraceptive course.

1 Goldstein, J L, *et al*, *Journal of Clinical Investigation*, 1973, **52**, 1544.
2 Zorilla, E, *et al*, *Journal of Clinical Endocrinology and Metabolism*, 1968, **28**, 1793.
3 Wallace, R B, *et al*, *Lancet*, 1977, **2**, 11.

What investigations are advised in a patient with psychosis thought to be due to temporal lobe epilepsy?

The interictal psychoses of temporal lobe epilepsy arise about 14 years after the onset of the seizures. Investigation should be directed towards establishing the cause of the epilepsy and to ensuring that the effects of anticonvulsant treatment have not caused metabolic abnormalities which may have precipitated an abnormal mental state. Investigation of the epilepsy should consist of an EEG to determine whether the psychosis is related to a continuing epileptic discharge or is unrelated to an ictus, and to define the area and origin of the patient's epileptic attacks. An EMI scan is essential to rule out a slow-growing glioma or other tumour of the temporal lobe, as hamartomas are seen in association with temporal lobe epilepsy and psychoses. Psychometry would help to establish whether the right or left or both temporal lobes have been damaged. Estimation of the serum anticonvulsant concentrations to ensure that they are within the therapeutic range and that the patient is not showing toxic effects is important. Anticonvulsants may reduce the serum folate and red cell folate concentrations and a low folate concentration may precipitate an abnormal mental state. Routine haematological and biochemical screening is important to check on kidney, thyroid, and liver functions, which may have been altered by long-term anticonvulsant treatment.

A Caucasian woman of 30 contracted onchocerciasis in the French Cameroons. She has been treated twice with diethylcarbamazine (Banocide) but suffers severe reactions, and she still has severe pruritus. Is there another drug that can be used that will not cause unpleasant side effects?

Three courses of Banocide are often necessary before there is any pronounced improvement, which is often not acceptable to the patients because of side reactions. The best drug to kill the adult worm, and indeed the only one that works, is suramin but, unfortunately, this occasionally has some alarming side effects. It is nephrotoxic, and some people have an idiosyncracy to the drug and collapse. Occasional deaths have even been recorded. It is true that if one lives in a temperate climate and is not likely to have further infections the worms eventually die after a period, which can be as short as five years or as long as 20 years. The only other microfilaricidal drug that has been used and has slightly fewer side reactions than Banocide is metriphonate or bilarcil, which has been used successfully against schistosomiasis. The dose is 10 mg/kg, given daily for three days, and this might be worth trying in the hope that the patient may not react as badly to it as she did to Banocide. There is no danger whatsoever of the patient infecting any other member of the family by contaminating the food. The only way the infection can be transmitted is by the vector *Simulium mosquito*, and in a temperate climate this is highly unlikely.