Mechanism of Gouty Inflammation

SIR,—In your leading article on this subject (20 October, p. 125) you emphasize the occurrence of leucotaxis after ingestion of sodium urate crystals by leucocytes. You might also have mentioned the elegant work of Naff and his colleagues,^{1,2} which showed that sodium urate crystals (but not calcium pyrophosphate) are capable of activating complement with release of chemotactic factors directly, without ingestion by cells. This mechanism may operate in acute gouty arthropathy as well, and contribute to the exudate. Complement depletion in animals inhibits uric acid crystal-induced inflammation.³—I am, etc.,

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- 2 Byers, P. H., Ward, P. A., Kellermeyer, R. W., and Naff, G. B., *Journal of Laboratory and Clinical Medicine*, 1973, 81, 761.
- 3 Webster, M. E., Maling, H. M., Zweig, M. H., Williams, M. A., and Anderson, W., *Immunological Communications*, 1972, 1, 185.

Intratracheal Anaesthesia

SIR,—In defence of the allusion in your leading article on "Post-intubation Granuloma" (10 November, p. 313) to endotracheal anaesthesia, it is nowadays perhaps necessary to comment on the considerable distinction between the insufflation method adopted by Mr. R. E. Kelly in Liverpool before the first World War and later developments. The former was by custom always referred to by the hybrid term "intratracheal" intubation, and entailed the insufflation of a relatively small flow of oxygen and ether vapour through a narrow gum-elastic catheter passed down to the region of the tracheal bifurcation, leaving most of the tracheal lumen available for normal to-and-fro breathing of atmospheric air. Like Dr. C. Langron Hewer (1 December, p. 551), I also (in Liverpool) had the fortune to be present at many operations in which this method was used in the 1920s. The improvement it constituted on previous "open" methods had to be seen to be believed. The narrow tube caused no pressure, friction, or distension on surrounding structures, and so far as I know no granulomas were then caused.

It was the "endotracheal" intubation with a wide-bore tube designed to carry the whole respiratory exchange which was widely adopted in the 1930s, and it was then that granulomas started to be reported.¹ The wider-bore tube was quite capable of causing laryngeal trauma, and one would assume that it is this practice which was alluded to in your article.—I am, etc.,

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- 1 Moulden, G. A., and Wynne, R. L., *British Journal of Anaesthesia*, 1951, 23, 92.

SIR,—Sir Ivan Macgill (1 December, p. 551) asks whether the old art of anaesthesia has been lost entirely. This must be a purely rhetorical question because he well knows that the "old art" depended mainly on the skilful administration of toxic volatile vapours.

The majority of the modern generation

of anaesthetists have never used chloroform and an increasing number are becoming unfamiliar with ether. But the "old art" has been replaced by a new art in the handling and administration of intravenous drugs for sleep, tranquillity, analgesia, and muscular relaxation against a background of non-toxic or mildly toxic gases delivered by a ventilating apparatus through "the tube" which "Maggie" showed us how to use.

My generation, and I hope the younger generations also, know that it was Sir Ivan's pioneering and inspiring skill which started raising anaesthesia from a minor specialty to its present high status level.—I am, etc.,

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Forms of Colitis

SIR,—I was interested in your leading article on this subject (17 November, p. 370) and would like to comment on two aspects mentioned therein.

Firstly, I was surprised that you stated that "Crohn's disease has had a more precise historical passage" than ulcerative colitis. The disease process may have been first described by Morgagni as long ago as 1769.¹ Descriptions of the disease affecting the small intestine appeared in England and in Scotland in 1813 and 1828³ respectively. Moynihan described six patients with large-bowel disease in 1907⁴ and Robson three patients (one large-bowel and two small-bowel disease) in 1908,⁵ though neither author appeared aware of the significance of his contribution. Heinrich Braun in 1909⁶ showed a deeper histological understanding of the disease.

The best landmark in the history of the disease came, to my mind, in 1913, when Dr. T. K. Dalziel published his paper on "Chronic Intestinal Enteritis" in the *B.M.J.*⁷ He gave an accurate clinical, macroscopic, and histological account of the disease as it affected the small and/or the large intestine. He even suggested a mycobacterial (*M. johnei*) aetiology for the condition. In America Moschowitz and Wilensky (Mount Sinai Hospital, New York) reported the disease as it affected both the large (1923)⁸ and small (1927)⁹ intestine. This much abbreviated historical review should help to put your editorial landmarks into perspective.

I was also disappointed that the clinical differences between ulcerative colitis and Crohn's disease of the large intestine alone were mentioned so briefly. The history is often helpful. In ulcerative colitis the essence, by the time the patient sees the doctor, is the bloody nature of the diarrhoea; in Crohn's colitis the patient usually complains of diarrhoea which on questioning may or may not have been noticed to have contained blood. On examination finger clubbing favours a diagnosis of Crohn's colitis.^{10,11} Absence of perianal disease strongly favours a diagnosis of ulcerative colitis, whereas the presence of gross perianal disease equally strongly favours a diagnosis of Crohn's disease.¹²—I am, etc.,

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- 2 Combe, C., and Saunders, W., *Medical Transactions of the Royal College of Physicians of London*, 1813, 4, 16.

- 3 Abercrombie, J., *Pathological and Practical Researches on Diseases of the Stomach, the Intestinal Canal and Other Viscera of the Abdomen*, p. 238. Edinburgh, Waugh and Innes, 1828.
- 4 Moynihan, B. G. A., *Edinburgh Medical Journal*, 1907, 21, 223.
- 5 Robson, A. W. M., *British Medical Journal*, 1908, 1, 425.
- 6 Braun, H., *Deutsche Zeitschrift für Chirurgie*, 1909, 100, 1.
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- 10 Fielding, J. F., and Cooke, W. T., *Gut*, 1971, 12, 442.
- 11 Jalan, K. L., et al., *Gut*, 1970, 11, 748.
- 12 Fielding, J. F., *Journal of the Royal College of Surgeons, of Edinburgh*, 1972, 17, 32.

Exsanguinating Digital Tourniquet

SIR,—I have read with interest the recent correspondence on the dangers of using exsanguinating digital ring tourniquets (30 June, p. 779; 4 August, p. 293; and 20 October, p. 174).

These rings have been available for the past 10 years, since I first described the device.¹ The original rings were individually made from latex and were difficult to standardize in terms of thickness. Some surgeons also complained that the rings sometimes contained bubbles of air. About four years ago the manufacturers changed over to silicone rubber for these technical reasons and also because there was insufficient skilled labour to cope with large demand. The silicone rubber rings have the benefit of being of standard thickness and elasticity but are rather more difficult to apply and, as has been pointed out, are difficult to detect against skin. The distributors tell me that in 1972 almost 9,000 tourniquet rings were supplied and that the demand this year appears to have increased over last year.

There is, of course, a risk inherent in the use of any tourniquet and, as has been pointed out by your correspondents, digital tourniquets can very easily be left in place accidentally. For this reason I would not encourage the routine use of these rings in a casualty department where a doctor can so easily be called away just as he is about to take off the tourniquet ring. It seems to me that the tourniquet rings are more useful in formal operations on digits, where the surgeon is unhurried and has time to take off the tourniquet himself, before the dressing is applied.—I am, etc.,

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- 1 Smellie, G. P., *Lancet*, 1962, 2, 78.

Schönlein versus Henoch

SIR,—In articles on the nephritis associated with Henoch-Schönlein (or Schönlein-Henoch) purpura^{1,2} it is customary to make a double obeisance to the eponymous originators of the disease. It is usually stated that while Schönlein in 1837³ first described the association of purpura with joint manifestations, it was not until 1852 that Sir George Johnson⁴ noted the occurrence of kidney disease in purpura. Johnson certainly states: "Disease of the kidney, with albuminous urine is very common in connexion with purpura." However, the patient whom he describes (Charles Fox, p. 152⁴) was