The Surgical Management of Toxic Dilatation of the Colon:

A Report of 28 Cases and Review of the Literature

RICHARD J. STRAUSS, M.D., GEORGE W. FLINT, M.D., NORBERT PLATT, M.D.,
LEROY LEVIN, M.D., LESLIE WISE, M.D.

Experience with 28 patients with toxic dilatation of the colon is reviewed. The operative mortality in this series was 32% (9/28). Eight of the 9 patients who died were found to have colonic perforations at operation; in contrast, the group of patients with no perforations had a mortality rate of only 6%. Colonic perforation and sepsis were the most significant factors contributing to mortality and morbidity in this series. A review of the literature showed an overall operative mortality rate of 19.5% for patients with toxic megacolon; the mortality rate was 41% for patients with perforations and 8.8% for patients without perforations. It appears that the keystone to successful management is the avoidance of colonic perforation and sepsis; protracted medical management of toxic megacolon seems to have been at least partly responsible for these complications. Sixteen of the 18 survivors following subtotal colectomy required removal of the rectum within 9 months because of continued symptoms and disease in the rectal stump.

From the Departments of Surgery and Pathology, Long Island Jewish-Hillside Medical Center, New Hyde Park and The State University of New York at Stony Brook, New York

ONE OF THE most dramatic and ominous complications which may occur in the course of non-specific inflammatory disease of the colon is the development of massive colonic dilatation. This complication, which carries with it an extremely high morbidity and mortality, has been reported to have an incidence which ranges between 1.6% and 5.8% of all patients admitted to hospitals with a diagnosis of ulcerative colitis.[1,5,7,20,26] Prohaska[27] reported a 12.5% incidence and Flatmark[9] a 28% incidence of toxic dilatation of the colon in their series of surgically treated patients with non-specific inflammatory disease of the colon.

In this paper we present the results of a retrospective analysis of the hospital course of 28 patients who underwent surgical treatment for toxic dilatation of the colon.

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Reprint requests: Richard Strauss, M.D., Department of Surgery, Long Island Jewish-Hillside Medical Center, New Hyde Parke, N.Y. 11004.

Material

Between 1957 and 1975, there were one hundred and fifty-seven patients who had surgery performed for non-specific colitis at the Long Island Jewish-Hillside Medical Center. In 28 of these patients (18%), a diagnosis of toxic megacolon was made on the basis of a markedly dilated colon on plain films of the abdomen during a severe, fulminating attack of inflammatory bowel disease.

Nine of the 28 patients were transferred from other hospitals, usually after being treated for several days or weeks. They were critically ill upon arrival and 6 of these 9 patients died postoperatively. The 28 patients were divided into two groups. Group I consisted of the 9 patients (4 females and 5 males) who died; group II included the 19 patients (11 females and 8 males) who survived.

A summary of the clinical features is presented in Table 1. Fever and diarrhea were almost constant features, the temperatures often spiking up to 40°. There were two patients without fever; both of them were receiving high doses of steroids and antibiotics. Crampy

abdominal pain, bloody diarrhea, anemia, abdominal distension and abdominal tenderness were also very frequent findings in these patients.

The age distribution of the patients at the time of operation is shown in Fig. 1. The youngest patient was 15 years old and the oldest was 70.

Table 2 summarizes the pertinent clinical history and several aspects of the hospital course related to the 28 patients in this study. The duration of the disease prior to the occurrence of toxic dilatation of the colon was extremely variable, ranging from three days to 29 years (Table 2). The average length of known disease in patients in Group I was 35 months, and in Group II it was 54 months. The present episode was the first attack of inflammatory bowel disease in 7 of the 28 patients; 3 of these 7 patients died.

Initial treatment of these patients included intravenous fluids, antibiotics, nasogastric intubation, and transfusions were given as indicated. In addition, 6 patients in Group I received steroids and 8 received ACTH during their hospitalization. Sixteen of the patients in Group II received steroids and 10 received ACTH. Four (44%) of the patients in Group I had barium enemas and 5 patients (26%) in Group II had barium enemas during the present admission.

Although some degree of leukocytosis was frequently noted, only 8 patients had white blood cell counts of over 14,000 per mm^3. Hypoalbuminemia appeared to be a fairly constant finding. The mean serum albumin in Group I was 2.1 ± 0.3 and in Group II it was 2.8 ± 0.1 ($P < 0.05$).

In order to evaluate the influence of duration of dilatation of the colon upon mortality, we compared the length of time dilatation was present in the two groups before surgery. The patients in Group I had toxic dilatation an average of 15 ± 3.7 days, and the patients in Group II had a mean of 7.4 ± 1.5 days. This was a statistically significant difference ($P < 0.05$).

Failure of satisfactory clinical response to nasogastric intubation, intravenous fluids, antibiotics, steroids and/or ACTH accompanied by increasing size of the gas-filled, distended colon were the indications for surgical intervention.

Results

The operative mortality in this series was 32% (9/28). At operation, colonic perforation was found in 12 (43%) of these 28 patients. Among the patients in Group I, 8 of the 9 patients (88%) had either free or walled-off perforations with a loculated abscess. One of the patients who had a walled-off perforation died on the operating room table of a cardiac arrest secondary to cardiac arrhythmia. The other patients died an average of 20 days postoperatively.

TABLE 1. *Clinical Findings on Admission (28 Patients)*

Clinical Finding	Non-survivors (9)	Survivors (19)	Total (28)
Fever	9	17	26
Diarrhea	8	15	23
Blody diarrhea	6	11	17
Anemia*	7	11	18
Abdominal distension	7	11	18
Crampy abdominal pain	7	13	19
Abdominal tenderness	6	11	17

* Anemia was defined as a Hb value of less than 10 gm %.

Four of the 19 patients (21%) in Group II had perforations and two of these 4 patients had septic and very complicated postoperative courses. Thus, in our series, 12 patients (8 from Group I and 4 from Group II) had perforations and fecal contamination of the abdominal cavity at the time of surgery. Among these 12 patients there was a 66% mortality. In sixteen patients (15 from Group II and one from Group I), the colon was not perforated at the time of surgery and the mortality of these patients was 6.25%.

Twenty-five patients had subtotal colectomy, ileostomy, and sigmoid mucous fistula as their initial operative treatment. Eighteen of these patients survived; 15 (83%) required resection of the rectal segment because of persistent symptoms of bleeding, discharge, and pain during a mean followup period of 9 months. All these resections were done on an elective basis. One patient (R.C.) had an ileoproctostomy 18 months after the STC and has been doing well for 11 years. Another patient will soon have an abdomino-perineal resection because of persistent disease. It should be noted that two of the patients in Group II had their ileostomies converted to a continent ileostomy and both are doing well (R.V. and R.S.)

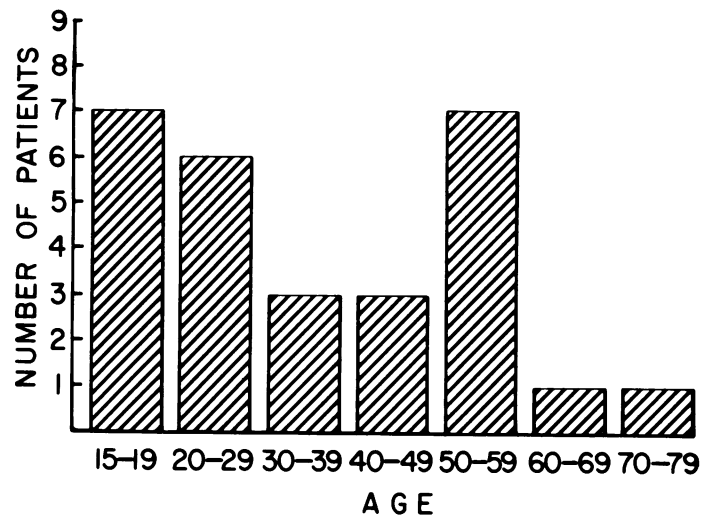

FIG. 1. Age incidence (in years) in the 28 patients with toxic megacolon.

TABLE 2. *Clinical Features and Postoperative Followup*

Patient, Age and Sex	Duration of UC	Duration of Acute Dilation (days)	Duration Preop Medical Rx (days)	Operative Procedure	Free (P) or Walled-off (A) Perforation	Followup	
						Duration	Other Operations
1. MB, 55M	3 wk	34	5	TC*	No	Patient expired 56 days postop	
2. EH, 50F	4 yr	2	1	STC†	A§	Patient expired immediately postop	
3. GR, 41M	1.3 yr	8	10	STC	A	Patient expired 35 days postop	
4. JG, 19F	9 mon	29	29	STC	A	Patient expired 6 days postop	
5. AM, 26M	7 yr	2	1	STC	P‖	Patient expired 33 days postop	
6. ET, 70F	10 yr	20	7	TC	A	Patient expired 1 day postop	
7. MN, 50M	9 mon	13	24	STC	A	Patient expired 6 days postop	
8. WS, 50M	5 wk	10	0	STC	A	Patient expired 14 days post colectomy; one day post gastrectomy	
9. CL, 56F	3 wk	13	13	STC	A	Patient expired 7 days postop	
10. RV, 21F	5 yr	3	12	STC	No	10 mon postop had Kock procedure and APR	
11. EF, 31F	5 yr	1	8	STC	No	8 mon postop had APR	
12. FK, 15M	3 dy	3	3	STC	No	1 mon postop had laparotomy for small bowel obstruction; 21 mon postop had APR	
13. RK, 22M	4.7 yr	16	0	STC	No	6 mon postop had APR	
14. MK, 28F	18 yr	5	6	STC	No	5 mon postop had APR	
15. RS, 20M	2 yr	18	18	STC	P	3 mon postop had APR with Kock procedure	
16. WR, 16M	2 yr	3	3	STC	No	3 mon postop had laparotomy for subhepatic abscess; 9 mon postop had APR	
17. BM, 49M	3 yr	11	1	STC	No	6 mon postop had APR	
18. RSL, 51F	1 mon	17	21	BH‡	No	5 mon postop had APR	
19. RC, 50F	20 yr	7	7	STC	No	1 mon postop had revision of ileostomy; 18 mon postop had ileo-rectal anastomosis	
20. HM, 19M	5 yr	3	6	STC	No	5 years postop had APR	
21. SB, 17F	3 yr	5	1	STC	No	2 years postop had APR and 2½ years postop had laparotomy for small bowel obstruction	
22. MB, 64F	2 mon	3	23	STC	No	1 mon postop had laparotomy for removal of foreign body from abdomen; 7 mon postop had APR	
23. HP, 18M	3 dy	18	19	STC	P	13 mon postop had APR	
24. AK, 38F	3 wk	2	2	STC	A	Doing well 6 mon postop; then lost to followup	
25. MB, 47F	2.3 yr	20	21	STC	A	To have APR in the near future	
26. ML, 16M	10 mon	1	10	STC	No	1 mon postop had laparotomy for small bowel obstruction	
27. SL, 20F	1 yr	4	6	STC	No	5 mon postop had APR; 14 mon postop had excision of perineal sinuses	
28. RM, 37F	3 yr	2	10	STC	No	4 mon postop had APR	

* Total proctocolectomy.
† Subtotal colectomy.
‡ Blow-hole.
§ Abscess.
‖ Free perforation.

One of the patients had a "less than colectomy," or blowhole procedure, as her primary procedure. A total proctocolectomy was done on this patient 5 months later and she is still doing well. Two of the patients had total proctocolectomies as their initial operation but they both expired postoperatively.

Table 3 lists the postoperative complications. Sepsis was the main complication which contributed to the death of most of the patients. Complications necessitating re-operation also occurred (Table 2). Three patients were re-explored for small bowel obstruction. One patient had a revision of ileostomy and one patient had

TABLE 3. *Postoperative Complications (28 Patients)*

Complications	Group I Patients	Group II Patients
Pneumonia	5	2
Septicemia	5	2
Wound infection	4	2
Intra-abdominal abscess	3	1
Renal failure	3	—
Dehiscence	1	2
Intestinal obstruction	—	5
Upper GI bleeding	2	1
Prolonged postop fever	—	3
Pleural effusion	—	2
Gangrene of ileostomy	—	1
Retraction of colostomy	—	1

a subtotal gastrectomy for upper GI bleeding. Other reoperations included tracheostomy, laparotomy for subhepatic abscess, laparotomy for removal of foreign body from abdomen, and excision of perineal sinuses.

All colonic specimens showed both large and small punctiform ulcerations. Large areas of mucosal denudation resulted in exposure of the circular muscular layer of the colon in 30% of the specimens. In 6 specimens this mucosal loss extended over 50% of the mucosa. The remaining mucosa was seen as isolated islands or interconnecting tracts with undermined borders. Mucosal bridges were often seen, however, pseudo-polyps had not yet formed. Only one patient (M.B.) showed this pattern of involvement in the rectum as well as the colon.

The inflammatory process of the colonic specimens was studied and graded using the system applied by Norland and Kirsner.[24] The microscopic findings were similar to those obtained by these authors; colonic specimens bearing areas of perforation showed transmural inflammation with peritonitis and destruction of the muscle. Twenty-six of our patients were diagnosed as having ulcerative colitis, and two patients (R.C. and R.V.) were diagnosed as having Crohn's disease on a histological basis.

The cellular reaction consisted of a mixture of lymphocytes, plasma cells and polymorphonuclear leukocytes, with frequent loss of the muscularis propria at the base of the ulcers. Various degrees of involvement of neural plexus were evident as the inflammatory process extended into the deeper layers of the colon. However, there was no evidence of destruction of the neural plexuses beyond the areas of muscle involvement.

Thirteen of the 16 patients who had subsequent removal of their rectums showed striking microscopic inflammatory changes. These consisted of dense lymphocytic infiltrates of the mucosa, with the formation of numerous lymph follicles exhibiting active germinal centers. The colonic crypts were markedly reduced in number and often displaced by the lymphocytic infiltrates. Similar changes were also seen in the rectal biopsy of one of the surviving patients (M.L.). These changes are usually seen in retained defunctionalized rectums and they closely mimic the picture of Crohn's disease or lymphoma of the rectum.

Discussion

Both the etiology and pathogenesis of toxic megacolon are obscure. Bockus and associates[3] suggested that paralysis and destruction of the myenteric plexus might be the cause of the colonic paralysis and dilatation. McInerney[20] reported distortion and edema of the nerve cells in patients with toxic megacolon, but others have failed to show consistent changes in the neural mechanism.[28] The pathogenesis of this morbid dilatation of the colon has been recently ascribed to transmural extension of the inflammatory process to all coats of the colonic wall.[13,17,20,26] The muscularis is often completely replaced by vascular granulation tissue in some areas, and degeneration and necrosis of muscle fibers are widely observed. Lumb et al.[17] and Spiro[35] emphasized that this penetration of the ulcerative in-

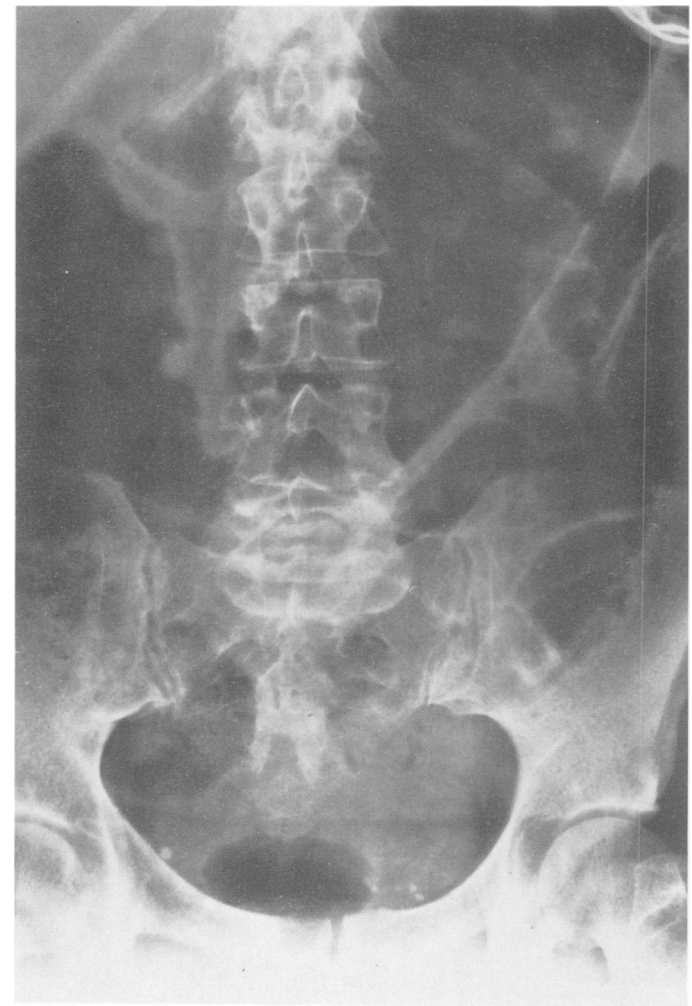

FIG. 2. Toxic dilatation of the colon showing multiple pseudopolyps in a patient receiving codeine.

flammatory process into the muscular coats of the colon with necrosis and destruction of the muscle fibers was the most important factor in the genesis of acute toxic dilatation and was the most important factor in subsequent perforation of the colon.

Prior to 1950, no mention of toxic dilatation of the colon was made in the English literature until Madison and Bargen[21] published a report of "unusual segmental dilatation" of the transverse colon without distal obstruction in association with a fulminating relapse of ulcerative colitis.

Some of the therapeutic agents used in the treatment of ulcerative colitis have been incriminated as possible precipitating factors leading to toxic dilatation.[11,13,17,24,34] Notable among these are opiates and anticholinergic drugs. These drugs have a strong inhibitory effect on colon motility, and since patients with acute ulcerative colitis have already diminished propulsive peristalsis, they could act to compound the problem. Most of the patients in this series were receiving one or both of these agents when toxic dilatation occurred (Fig. 2).

The onset of dilatation soon after barium enema has also frequently been noted[20,24,25,34]; 9 of the patients in this series had a barium enema during the present admission. Other authors, such as Cohn[4], have indicated that hypokalemia may be involved in the pathogenesis of this condition. However, most current reports fail to confirm this observation and Marshak[22] reported no striking response to potassium therapy. Most authors agree[20,28,34] that steroids have no role in the production of toxic megacolon.

Toxic dilatation frequently appears during the first attack of ulcerative colitis. Five of the 12 patients in Roth's series,[28] 15 of the 21 patients in Koudahl's[16] series, and almost half of the patients in Foley's[10] series developed toxic megacolon during their initial episode of symptoms. Many authors have recognized that there is a large variation in duration of illness preceding the attack of toxic megacolon.[26,34,24,31] It thus appears that acute dilatation can occur at any time in the course of the disease, from the initial attack to an acute exacerbation of chronic disease of many years' duration.

There has been a growing acceptance of early colec-

TABLE 4. *Review of the Literature on Toxic Megacolon*

Author	Year	Total Cases	Total Number of Pts. with Perforation	Medical Mortality	Surgical Mortality			Overall Surgical Mortality %	Overall Mortality %
					Overall	With perforation	Without perforation		
Lumb[17]	1955	7	6	0/1	4/6	4/6	0	66	57
McConnell[18]	1958	26	4	5/21	1/5	0/2	1/3	20	23
Roth[28]	1959	12	4	1/6	2/6	1/2	1/4	33	25
Klein[14]	1960	3	0		0/3	0	0/3	0	0
Korelitz[15]	1960	16	4	2/5	1/11	1/4	0/7	9	19
Peskin[26]	1960	9	7	0	2/9	2/7	0/2	22	22
Sampson[30]	1961	14	4		4/14	4/6	0/8	28	28
McInerney[20]	1962	36	10	5/28	5/8	4/5	1/3	63	28
Smith[34]	1962	11	4	2/3	0/8	0/3	0/5	0	18
Rowe[29]	1963	10	5	0/1	1/9	1/5	0/4	11	10
Edwards[7]	1964	10	1	3/9	0/1	0	0/1	0	30
Prohaska[27]	1964	16	9		5/16	5/9	0/7	31	31
Silverberg[33]	1964	4	2	1/1	0/3	0/1	0/2	0	25
Diethelm[6]	1964	6	0		0/6	0	0/6	0	0
McElwain[19]	1965	9	1	0/4	0/5	0/1	0/4	0	0
Ferrante[8]	1965	6	1	0/1	0/5	0/1	0/4	0	0
Neschis[23]	1968	12	?	0/4	2/8	?	?	25	17
Jalan[13]	1969	55	16	10/17	15/38	8/12	7/26	39	45
Odyniec[25]	1969	37	9	1/14	3/23	?/9	?/14	13	11
Thomford[36]	1969	23	6	1/4	5/19	?/5	?/14	26	26
Norland[24]	1969	42	10	3/10	7/32	5/9	2/23	22	19
Goligher[12]	1970	28	11	0/1	5/27	?	?	19	18
Foley[10]	1970	28	12		9/28	7/9	2/19	32	32
Turnbull[37]	1971	42	?		2/42	0	2/42	5	5
Collier[5]	1971	35	?	9/24	1/11	?	?	37	29
Adams[1]	1973	16	5	0/4	3/12	3/5	0/7	25	19
Shipp[32]	1974	8	?		0/8	?	?	0	0
Scott[31]	1974	17	7		1/17	1/7	0/10	6	6
Binder[2]	1974	18	0	0/1	1/17	0	1/17	6	6
Koudahl[16]	1975	21	6	0/1	6/20	1/6	5/14	30	29
Flatmark[9]	1975	27	?		2/27	?	?	7	7
Total		604	144 (24%)	43/160 (27%)	87/444 (19.5%)	47/114 (41.2%)	22/249 (8.8%)	19.5%	19%

tomy in patients with toxic dilatation of the colon and many publications have advocated early operation in these patients.[1,2,5,10,12,16,19,27] While many surgeons have advocated emergency resection of the colon with ileostomy, Turnbull and his associates,[37] concerned with the high mortality rate of colectomy in patients with toxic megacolon, have recommended initial surgical treatment by emergency loop ileostomy and so-called "blow hole" transverse and/or sigmoid colostomy. They suggest that it is the operative disruption of walled-off perforations and the resulting fecal contamination that results in the fatal sepsis. In this same study, Turnbull et al.[37] demonstrated that 16 of the 42 patients in which they did an ileostomy and colostomy as the primary treatment for toxic megacolon had a histological diagnosis of Crohn's disease.

The classical description of the patient presenting with toxic megacolon is a septic patient who is acutely ill and who is typically dehydrated, febrile and anemic, and who exhibits diarrhea, vomiting and prostration with accompanying abdominal distention, pain, tenderness and decreased bowel sounds. This presentation was indeed true for many of the patients, however, in other patients the presenting signs and symptoms were much less dramatic and the patients were afebrile and ambulatory. However, on clinical and radiological examination, these patients had an ominously dilated colon. Thus, the term "toxic" dilatation does not always apply and this massive dilatation of the colon should be taken as the physician's warning of impending perforation of a severely inflammed colon.

The recognition of toxic dilatation of the colon as a complication of ulcerative colitis has now been reported in over 600 cases (Table 4) and many authors have emphasized and confirmed the high incidence of perforation with this dilatation.

A survey of much of the recent literature on toxic megacolon is presented in Table 4. There was a 27% overall medical mortality rate in this collection of cases. The overall surgical mortality rate was 19.5%. The operative mortality rate for patients with perforations was 41%, in contrast to an 8.8% mortality for patients without perforations.

Perforation occurred in 24% of the patients listed in Table 4. Since perforation is the most important factor in the high mortality associated with toxic megacolon, it is apparent that the keystone to successful management is the avoidance of colonic perforation. We believe that in most cases it is not the operation itself, but rather the protracted medical management, which is the predominant factor responsible for colonic perforation and fatal sepsis.

The development of dilatation of the colon in a patient with non-specific colitis should be regarded as a sign of impending perforation. Such a patient should be prepared for surgery and if the dilatation does not subside within 48 to 72 hours, the patient should be operated on. The surgeon has a choice of three operative procedures: 1) total proctocolectomy and ileostomy; 2) sub-total colectomy, ileostomy and sigmoid mucous fistula; 3) "Less than colectomy," i.e., loop ileostomy, transverse and/or sigmoid colostomy.

In our experience, most of these patients are too ill and in too depleted a state to be subjected to a total proctocolectomy. A sub-total colectomy, ileostomy and sigmoid mucous fistula should probably be done, leaving the rectum to be removed in a second stage when the patient's condition has improved. In the event that free perforation exists, or walled-off perforation is found at laparotomy, consideration should be given to a "blow-hole" procedure, with colectomy at a later date.

Earlier operation on patients with toxic dilatation of the colon should result in a considerable decrease in the incidence of perforation and in the morbidity and mortality.

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ERRATUM

In the article 'Manpower Goals in American Surgery' (Moore, F. D., Ann. Surg. 184:125–144, 1976) there are errors that should be corrected. On page 142, second column, last paragraph, the following:

> 'Although it has often been considered unlikely that voluntary National Health Agencies, colleges, academies and boards, can be traced back over 100 residency openings in hospital based specialties including surgery . . .'

should be changed to:

> 'Although it has often been considered unlikely that voluntary National Health Agencies, colleges, academies and boards, can be relied upon to reduce residency openings in hospital based specialties including surgery, the record of voluntary agency control in the United States is impressive'.

In Tables 24 and 25 the population cutoff for "cities and towns" should be 10,000–250,000 and for metro areas 250,000 or more. Appropriate changes in text wording are required in the first paragraph of the first column on page 137. And in the footnote on page 139.