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Effect of Brain-Stem Lesion on Colonic and Anorectal Motility
Study of Three Patients

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The supraspinal control of colonic motility and anorectal motility is poorly documented. We had the opportunity to study colonic function, esophageal function, and urinary bladder function in three patients who presented with vascular lesions limited to the anterior (case 1) or the posterior area (cases 2 and 3) of the pons. Esophageal manometry, urodynamic examination, whole and segmental transit time measurements (using radiopaque markers) and anorectal motility were systematically performed. The results were the following: (1) in the first case esophageal motility was not altered, whereas abnormal micturition, right colonic inertia, and absence of rectoanal inhibitor reflex were observed; (2) in cases 2 and 3, there was a poor esophageal coordination, the micturition and rectoanal inhibitor reflex were normal, and the transit time of the left colon was increased. Our observations are consistent with the previously described localization of neurological areas controlling swallowing and micturition; they also favor the pons as the possible level of supraspinal control of colonic and anorectal motility.

The extrinsic control of colonic and rectoanal motility is exerted by the parasympathetic and sympathetic vegetative nervous system. The dorsolumbar and sacral centers of the spinal cord are known, and the existence of supraspinal control of colonic and rectoanal motility has been suggested (1–7). It is known that other visceral organs are under the neurovegetative control of supraspinal centers. This is the case for the esophagus, where swallowing control is located in the posterior part of the pons (8, 9). This is also the case for the urinary bladder system, where micturition is controlled in the anterior part of the pons (10, 11). We have formulated a hypothesis according to which supraspinal control of colonic and rectoanal motility could also be located in the pons. In this context, we have examined three patients with vascular lesions limited to the pons in order to confirm the consequences of this condition on esophageal and urinary bladder function, and employ the transit of markers and rectoanal manometry to determine if a colonic and/or rectoanal dysfunction also exists, which could suggest the existence of a pontine zone which participates in the supraspinal control of colonic and rectoanal function.

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MATERIALS AND METHODS

Patients

Case 1. A 51-year-old male presented in October 1981 with a stroke, including quadriplegia with mutism and neurovegetative and respiratory disorders requiring sev-

eral days of assisted breathing. Once respiratory autonomy was recovered, a quadripyramidal syndrome with mutism was observed, but consciousness was normal. The patient responded "yes" or "no" to questions by blinking, and the electroencephalogram was one of an alert subject. Sensitivity to all modes was normal, as was oculomotricity. The gag reflex was present. The CT scan of the brain was normal as was the ultrasonic flow-rate study of the large vessels of the neck. It was concluded that there was an infarction of the anterior part of the pons, resulting in a "locked-in syndrome" (12, 13). The anatomical extent of the lesions, deduced by the clinical signs, is schematically shown in Figure 1. Voluntary swallowing was possible when food was placed in the posterior part of the mouth; micturition was automatic. The patient had experienced no prior constipation before the stroke. Since then, he was constipated, with spontaneous emission of a stool every 8–10 days. He experienced repeated fecaliths, requiring numerous enemas. The abdomen was constantly swollen and evacuation of gas was possible only by installing a rectal probe. Thirteen months after the stroke, a pseudoocclusive episode occurred with fecaloid vomiting, which required the installation of gastric aspiration for 4 days. This episode was resolved by repeated enemas.

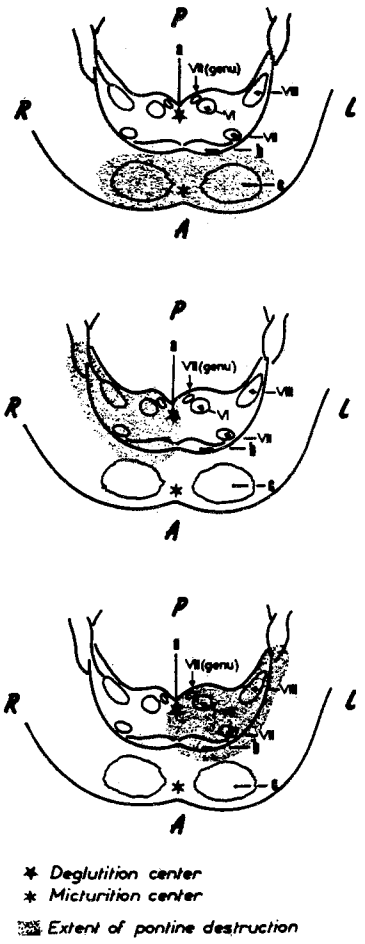

Fig 1. Diagram of pons at the level of destruction. Upper, case 1; middle, case 2; lower, case 3. a = medial longitudinal fasciculus, b = medial lemniscus, c = corticopontine tract. A = anterior, P = posterior, R = right, L = left.

Case 2. A 67-year-old male presented on Feburary 20, 1983, with a stroke. Consciousness was retained, and there existed a pyramidal-type motor deficit of the left half of the body leaving the face intact, a peripheral-type right facial paralysis, a hypoesthesia at all the modes of the left half of the body, and a kinetic cerebellar syndrome of the right arm. Oculomotor disorders existed: paralysis of laterality of gaze to the right and a left anterior internuclear ophthalmoplegia. The gag reflex was absent, but the 11th and 12th cranial nerves were normal. The CT scan was normal. It was concluded that there was an infarction of the right posterior pons (the anatomical spread of the lesion deduced from the clinical signs is shown schematically on Figure 1). The patient had swallowing disorders with false passages. Micturition was voluntary and occurred upon demand during the day, but with nocturnal enuresis. Although the patient had never had transit troubles, he was constipated since the stroke with emission of one stool per week, abdominal swelling, and pain in the left flank. A pseudoocclusive syndrome occurred in the hospital two months after the accident which was resolved by installing gastric aspiration and repeated enemas. No digestive obstacle was demonstrated by radiological and endoscopic examinations.

Case 3. A 63-year-old male presented on September 29, 1983, with a stroke with degradation of consciousness lasting 24 hr. There was a slight pyramidal-type motor deficit of the right half of the body, leaving the face intact, a peripheral-type left facial paralysis, a sensory deficit at all modes of the right half of the body including the face, and a kinetic cerebellar syndrome of the left half of the body. There were oculomotor disorders including bilateral paralysis of laterality of gaze and bobbing of the left eye. Certain pairs of cranial nerves were affected: deficit of the left motor trigeminal, abolition of the gag reflex, deficit of left vocal cord mobility, while the 11th and 12th pairs were normal. The diagnosis of a hematoma in the posterior pons was confirmed by a CT scan (the anatomical spread of the lesion deduced from the clinical and radiological signs is shown schematically in Figure 1.). The pyramidal motor deficit regressed during the following weeks, but the sensory and cerebellar disorders persisted, as did the involvement of cranial nerves. The patient experienced swallowing difficulties with frequent false passages. Micturition was not spontaneous, but it could be triggered by suprapubic percussion. The patient had previously had daily stools, but since the stroke complained of one stool every 3–4 days with abdominal swelling.

Methods

The patients were examined 23 (case 1), 11 (case 2) and 5 months (case 3) after the stroke, after having obtained the consent of the patients and their families.

Esophageal Manometry. Three perfused polyvinyl catheters (OD 1.5 mm, ID 0.8 mm) were used to transmit intraluminal esophageal pressure changes to electromanometers (Statham P23 ID). Each catheter was pierced

with a 1-mm-diameter orifice arranged in such a way that pressures were simultaneously recorded at three points 5 cm from each other. A hydraulic perfuser was used for perfusion. Recorder chart speed was 2.5 mm/sec for the lower sphincter and the esophageal body and 10 mm/sec for the upper sphincter and the larynx. The probe was introduced so that the three orifices were inside the stomach. It was then removed from the stomach, centimeter by centimeter, up to the pharynx.

The resting pressure of the lower esophageal sphincter is the pressure difference between that measured at the stomach and that measured at the sphincter. The value considered was the mean of pressures measured in each channel at the level of the point of respiratory inversion. Contractions of the esophageal body and relaxations of the upper and lower sphincters in response to swallowing saliva were examined. Each swallow was separated from the next by at least 1 min. The analysis of contractions of the esophageal body was carried out with published criteria (14). Coordination of the upper and lower sphincters was judged normal if sphincter pressure decreased by at least 90% in comparison to resting pressure during relaxation and if this relaxation persisted during the entire length of the contraction observed 5 cm above the sphincter. At the level of the body, we examined the propagation of contractions in response to swallowing. The number of relaxations of the sphincter and contractions of the esophageal body could not be standardized because of the limited possibilities of cooperation by our patients.

Urodynamic Examination. Posturination residues were determined in the three patients, and a search was made for a prostatic obstacle by rectal palpation, pre-, per-, and posturination cystography and urethrocystoscopy. The urodynamic examination was not possible in case 1 because of sequellae, prohibiting the prolonged gynecological position necessary to perform this examination in our technical conditions. In the other two cases, the bladder was perfused with a double polyvinyl catheter, 2.4 mm OD, with 0.15 M NaCl. One of the two catheters had two 1-mm-diameter orifices at the extremity of the probe and was connected to an electromanometer (Statham P23 ID) for the transmission of bladder pressure changes. The other catheter had four lateral 1-mm-diameter orifices around the circumference, mutally separated by 2 mm and 6 cm away from the orifices in the first catheter. This second catheter was used to fill the bladder when all the orifices were inside and was used to measure urethral pressure by withdrawing the probe at constant speed. The bladder was filled at a rate of 20 ml/min, and the possible occurrence of bladder contractions with an amplitude greater than 15 cm H_2O was noted during filling, as were the various patient sensations of need to evacuate. In case 2 the electromyographic activity of the perineum was recorded with skin electrodes connected to an electromyograph under oscilloscopic and audiographic control. The electromyographic activity of the external sphincter of the urethra of patient 3 was recorded directly with a needle electrode.

Three criteria were used to define detrusor external sphincter synergy. Type I: during the contraction of the detrusor in the course of a voluntary micturition there existed a decrease (skin electrodes) or an arrest (needle) or external sphincter activity, corresponding to the absence of dyssynergia. Type II: in spite of a contraction of the detrusor during micturition, the electromyographic activity of the perineum (skin electrodes) or of the external sphincter (needle) was unchanged. Type III: during urination, electromyographic activity increased, in spite of contraction of the detrusor (15, 16).

Transit Times of Radiopaque Markers. At the time of this examination, the patients received no medication which could modify smooth digestive muscle function. Case 1 was placed in a chair several times a day. Up to the day before administration of the marker, he received an anthroquinone laxative and had abundant stools daily after an enema. The other two patients has assumed a normal social life for the previous several months, their movement being limited only by the necessity to use forearm crutches because of the cerebellar syndrome. No patient received alimentary fibers during the examination, which was carried out according to the technique described by Arhan et al (17). The three subjects ingested the 20 markers on day 1 and an abdominal film was taken without preparation every 24 hr for at least 5 days. The number of markers in the right colon, left colon, and the rectosigmoid were counted on each film, and total and segmentary transit times were calculated. When markers persisted on the last film, it was admitted that they had completely disappeared 24 hr later and in this case we indicated that transit time was greater than or equal to the result thus obtained.

The results were compared to those obtained with 21 volunteers of both sexes examined at Rouen, and also to those of Arhan et al (17). Our control subjects, 17–35 years of age were on a normal uncontrolled diet, especially in terms of alimentary fibers. In order to take the possible role of decreased physical activity of our three patients into account, we also studied the transit times of six patients (five males and one female, 30–80 years old, median 57 years) bedridden as a result of orthopedic surgery (three cases) or an operation on soft parts (three cases) without laparotomy, during the period immediately after the anesthesia. None of these six patients had had transit disorders as antecedents.

Rectoanal manometry was performed with a balloon probe. Ahead of the distending balloon were two 1-cm-diameter recording balloons, 1.5 cm apart. The probe was placed in such a way that the most external balloon was in the lower part of the anal canal. In these conditions, the second balloon was in the upper part of the anal canal and the distending balloon in the lower part of the rectal ampulla. Pressures were recorded with electromanometers (Statham P23 ID). There were two successive steps in the rectal manometry examination: (1) Pressures in the upper and lower part of the anal canal were recorded for 15–30 min and the presence or absence of abnormal phenomena was noted, eg, ultra slow waves with an amplitude greater than 20 cm of H_2O, or spontaneous rectal sphincter antagonisms. (2) The rectal ampulla was then distended with increasing volumes of 10, 20, 30, 40, and 50 ml of air for 3-sec periods, enabling us to determine the threshold of conscious perception, to study the components of the rectoanal inhibitor reflex (RAIR), and the existence of an overshoot greater than 20 cm of

TABLE 1. RELAXATION OF LOWER ESOPHAGEAL SPHINCTER (LES) AND PERISTALTIC WAVES IN DISTAL 2/3 ESOPHAGEAL BODY

	Case 1	*Case 2*	*Case 3*
LES relaxation			
Number swallows	6	32	5
Normal	6	9	1
Incomplete or absent	0	23	4
Esophageal body			
Number swallows	13	42	11
Normal amplitude	13	42	11
Normal duration	13	22	11
Normal propagation	13	22	11

H_2O. (3) Finally, the patient was asked for a voluntary anal contraction of maximal amplitude and duration. The results of these manometric examinations were compared to normal values obtained in the same conditions with 24 volunteers of both sexes.

Rectal parietal tensions were studied with a second probe and according to the methodology described elsewhere (18). Pressures were initially measured in the rectum during distensions with 10, 20, 30, 40, and 50 ml in a random sequence. Distensions were maintained for 60 sec and were 60 sec apart. Immediately after measurements in the rectum, pressures were measured in ambient air. Air pressures were subtracted from rectal pressures point by point, taking the initial pressure peak as the origin of time. Pressures were measured at the beginning of the distension (P_0) and at the 60th second (P_∞). The inflated balloon was spherical, so the rectal parietal tension (T) and intrarectal pressure (P) were related by Laplace's law: $T = kP\ r/2$, where r is the radius of the distension balloon for the distension volume in question. Using this relationship, tensions T_0 and T_∞ were calculated for each of the five distension volumes with P_0 and P_∞.

RESULTS

Esophageal Manometry. The pharynx and upper sphincter were normal in cases 1 and 2 and could not be studied in case 3. In the three cases, there were no tertiary waves in the esophageal body and the resting pressure of the lower esophageal sphincter (LES) was normal. The result of LES relaxation and peristaltic contractions are shown in Table 1.

Urodynamic Examination. In case 1, micturition was incomplete, with residues between 100 and 400 ml which were not explained by a prostatic obstruction. In case 2, posturination residues were always less than 100 ml, in spite of prostatic hypertrophy. Bladder pressure was normal up to a filling volume of 260 ml, at which point there was a bladder contraction of 25 cm H_2O, contemporaneous with an immediate and urgent need. Micturition was triggered upon demand and detrusor–external sphincter synergy was type I. In case 3, posturination residues were between 200 and 300 ml. Intravenous urography and urethrocystoscopy showed a prostatic hypertrophy. Bladder pressure during filling was high, at 48 cm of H_2O for 360 ml, contemporaneous with an immediate and painful need. Micturition was triggered by suprapubic percussion, and detrusor–external sphincter synergy during micturition was type I.

Transit Times of Radiopaque Markers. The results (Tables 2 and 3) showed that whole transit times were augmented in the three patients. The transit times in the right colon and the rectosigmoid were augmented in case 1, as was left colon transit time in cases 2 and 3.

Rectoanal Manometry and Rectal Parietal Tension. The resting pressure of the anal canal was normal in the three patients. There were ultra slow waves in case 1. The RAIR was absent in this same case, in spite of normal rectal parietal tensions (T_0 and T_∞) during distensions. There was no voluntary anal contraction, but the threshold of conscious perception of rectal distension was normal. In case

TABLE 2. TRANSIT OF RADIOPAQUE MARKERS THROUGH LARGE BOWEL IN THREE PATIENTS

		Days after ingestion								*Transit time*
Subjects	*Site*	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*24*	*(hr)*
Case 1	Right colon	19	19	18	12	12	—	—	1	≥225
	Left colon	1	1	1	0	0	—	—	0	not calculated
	Rectosigmoid	0	0	0	7	7	—	—	11	≥191
	Total	20	20	19	19	19	—	—	12	≥320
Case 2	Right colon	11	6	4	1	0	—	—	—	27
	Left colon	9	13	15	18	5	—	—	—	≥66
	Rectosigmoid	0	0	0	0	12	—	—	—	not calculated
	Total	20	19	19	19	17	—	—	—	≥92
Case 3	Right colon	6	2	0	0	0	0	0	—	8
	Left colon	14	18	8	3	1	0	0	—	55
	Rectosigmoid	0	0	10	7	9	10	0	—	45
	Total	20	20	18	10	10	10	0	—	108

TABLE 3. TRANSIT OF RADIOPAQUE MARKERS THROUGH LARGE BOWEL IN CONTROL SUBJECTS

Subjects	*Site*	*Days after ingestion*								*Transit time* (hr)*
		1	*2*	*3*	*4*	*5*	*6*	*7*	*24*	
Personal data										
Ambulatory subjects† (N = 20)	Right colon	15	2	1	0	0	—	—	—	22
	Left colon	11	5	1	0	0	—	—	—	21
	Rectosigmoid	20	10	6	0	0	—	—	—	27
	Total	20	16	6	0	0	—	—	—	45
Postoperative‡ (N = 6)	Right colon	20	20	12	3	1	0	—	—	46
	Left colon	18	13	11	1	0	—	—	—	31
	Rectosigmoid	0	0	14	4	3	0	—	—	25
	Total	20	20	19	9	8	0	—	—	89
Arhan et al (17)†	Right colon	20	10	4	1	0	0	0	—	38
	Left colon	15	11	8	4	3	1	0	—	37
	Rectosigmoid	9	12	10	7	3	1	0	—	34
	Total	20	20	18	10	5	3	0	—	93

*Transit times have been calculated according to Arhan et al (17).
†Control values = upper limit of the normal range.
‡Upper value observed.

2, the RAIR, T_0, and T_∞ were normal; the amplitude of voluntary anal contraction was diminished; and rectal distension was perceived only with a volume of 60 ml. The amplitude of the RAIR in case 3 was constant, regardless of the rectal distension volume. T_0 did not increase with increasing distension volumes, and T_∞ decreased with increasing volumes. Rectal distension was not perceived, regardless of the distension volume applied. Voluntary anal contraction was normal.

All the results of the examinations in the three patients are summarized in Table 4.

DISCUSSION

In spite of the absence of anatomic documents, the good correlation between the neurological signs and anatomical topography of the lesions enables us to localize the site of the neurological lesions in the brain stem (Figure 1). The pons can be separated into an anterior part, whose damage leads to a uni- or bilateral pyramidal-type motor deficit, with retention of sensitivity and consciousness. This is the picture of the "locked-in syndrome" (12, 13). The lesion in case 1 corresponds to this anatomical territory. Lesions in the posterior part of the pons are reflected by a uni- or bilateral decrease of sensitivity of the extremities. The rostral extent of these lesions can be evaluated by studying eye motricity and awareness. A peduncular lesion causes a paralysis of the 3rd oculomotor nerve or of the verticality of gaze. In order for awareness to be retained, a part of the ascending activating reticular formation must be spared, ie, the lesion must not go beyond the motor nucleus of the 5th pair of cranial nerves in its rostral extent (19). The lesions in cases 2 and 3 did not go beyond this level, since they responded to these clinical criteria. The caudal extent of the lesion can also be evaluated by studying the function of bulbar cranial nerves. The lesions of patients 2 and 3 reached the middle part of the medulla oblongata, since the gag reflex was abolished, but they did not reach the lower part of the medulla, since the 11th and 12th cranial nerves were normal in case 2. The lesion in case 1 probably stopped at the medullopontine junction, since the

TABLE 4. SUMMARY OF ANOMALIES OBSERVED IN THREE PATIENTS*

Pontine destruction	*Marker studies*		*Esophagus*		*RAIR*	*Detrusor–sphincter synergia*
	Right colon	*Left colon*	*LES Relaxations*	*Peristaltic waves*		
Anterior (case 1)	A	N	N	N	A	A†
Posterior (case 2)	N	A	A	A	N	N
Posterior (case 3)	N	A	A	N	N	N

*A: abnormal; N: normal; RAIR: rectoanal inhibitor reflex.
†See methods case 1.

gag reflex was present and there were no swallowing disorders.

In spite of the limited number of deglutitions by our patients during the esophageal manometry examination and the lack of the urodynamic examination in case 1, the disorders in esophageal and urinary bladder function observed in our three patients are consistent with the localization of zones controlling swallowing (8, 9) and micturition (10, 11). The central control of swallowing is assured by medullar hemicenters, one facilitating, the other inhibiting, and connected to a pontine coordinating center located in the posterior part of the pons in the reticular formation between the posterior pole of the facial nerve nucleus and the rostral part of the inferior olive 1.3 mm behind these structures (8, 9). This center was damaged by the lesion in cases 2 and 3, which could explain the clinically observed difficulties in swallowing and the manometric anomalies. Inversely, in case 1, swallowing and esophageal manometry were normal, since the most anterior lesion left the swallowing center intact. This is confirmed by the similar observation of a locked-in syndrome reported by Feldman (20), where swallowing was clinically normal. This swallowing center participates above all in esophagosphincter coordination, since each hemicenter of the medulla oblongata is capable of triggering esophageal contractions (8, 9), which would explain why the observed manometric anomalies include primarily coordination troubles.

The coordinating center of micturition is located in the anterior part of the pons in the reticular formation (10). It receives activating and inhibiting information from the superior situated mesencephalic area and cortex (10, 21). It is thus not surprising to have observed a normal detrusor–external sphincter synergy during urination in the urodynamic examination in cases 2 and 3, who had posterior pontine involvement. The existence of disinhibited bladder contractions in case 2 during filling is a reflection of poor corticosubcortical control of parasympathetic inhibition during the continence phase (10), probably explaining the enuresis of this patient. In case 1, the urodynamic examination was not possible for technical reasons. The posturination residues observable in the absence of a clinically detectable mechanical obstruction in this patient could be explained by a detrusor–external sphincter dyssynergia secondary to the anterior localization of stroke (Figure 1).

Table 3 lists the colonic and rectoanal functional anomalies observed in our three patients. These anomalies are undoubtedly secondary to the involvement of the brain stem, since questioning of the patients and their entourage revealed no transit disorder before the stroke. The stroke itself cannot be responsible for the observed disorders because of the interval separating the accident and the examinations. The decreased physical activity of the patients could certainly participate in the decreased transit, but is not sufficient to explain it, since the transits of our patients were higher than those of the six patients having received general anesthesia and examined while bedridden. Moreover, it is logical to imagine that a supramedullary control of this visceral functioning exists (5, 6, 22), and in the context of our results it is tempting to speculate that this control could be exerted at the level of the pons, as are those of swallowing and micturition.

Rectoanal coordination could be under anterior pontine control as is the case for detrusor–external sphincter synergy. The RAIR, which is a reflection of the coordination between a rectal stimulus and the anal response, was absent in case 1 and present in the other two patients. The amplitude of the RAIR in case 3 did not increase as a function of distension volume as a result of the abnormal viscoelastic properties of the rectal wall. The initial tension peak (T_0) was, in fact, constant regardless of distension volume, and the adaptation tension of the rectum to the distension (T_∞) decreased with increasing distension volumes. We thus considered that the anal response of case 3 was neurologically normal, since it corresponded to a constant rectal stimulation. The colonic segmentary transit times of the radiopaque markers were abnormal in all three cases, suggesting that colonic motility is under pontine control. Future observations should confirm that chance is not responsible for slowed left colonic transit in the case of involvement of the posterior pons (cases 2 and 3), and of the right colon when the anterior pons is involved (case 1).

In light of our results, it thus appears that the pons could participate in the control of colorectal function. Our observations do not allow us to determine whether this control is assured by pontine areas which were destroyed in our patients, or by suspontine centers whose efferent pathways were interrupted. By analogy with known facts on swallowing and micturition, it is tempting to speculate in favor of the existence of a pontine center. Although our observations would seem to furnish arguments

favoring pontine control of colonic and rectoanal functions, it is clear that the central nervous control of the colon and the rectoanal system are incompletely known. Thus, the level of integration of the RAIR is still a subject of discussion (6, 7). It could be purely a spinal reflex, but its retention in cases of lesions of the conus medullaris or of the cauda equina may suggest a more peripheral integration, at the level of the perivisceral ganglial plexus, analogous to suggestions at the detrusor–urethral level (11). There is a different anatomical innervation of the right and left segments, but the frontier is not well limited (2). Spinal section in the cervical or thoracic part does not change segmentary colonic motility, while a low lumbosacral section leads to an increase of this activity (2).

It is obvious that our results await confirmation on a larger series, but we nevertheless believe that (1) neurological diseases may represent an unrecognized means of studying the extrinsic control of colonic and rectoanal function; (2) our results could explain the constipation associated with urinary bladder disorders in the Shy-Drager syndrome, which is a diffuse involvement of the brain stem (23); (3) similarly, and if our hypothesis is correct, involvement of the brain stem could be one of the causes of the systemic disease combining colonic inertia and extradigestive manifestations (24), so indicating a neurological examination in these patients.

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