VENTILATION IN PATIENTS ANAESTHETIZED FOR LAPAROSCOPY

JOHN DESMOND, M.B., B.S., F.R.C.P.(C), AND
R. A. GORDON, M.D., F.R.C.P.(C), F.F.A.R.C.S.*

INTRAPERITONEAL ENDOSCOPY, now referred to as laparoscopy, has been performed intermittently since the beginning of this century. Renon, a surgeon from Paris, published a paper on the technique of and indications for laparoscopy as long ago as 1913. Its use as a means of visualizing the abdominal organs was never popular until Steptoe in 1964¹⁻² published a large series of cases showing that the technique was a valuable addition to the armamentarium of the gynaecologist and general surgeon. Eighteen months ago, Steptoe demonstrated the use of this instrument at the Toronto General Hospital. At the time, one of us who was fortunate enough to be present showed some concern about what was happening to the large quantity of carbon dioxide which was introduced into the peritoneal cavity, particularly as the patient was breathing spontaneously on nitrous oxide and halothane anaesthesia and was maintained in a steep Trendelenburg position. On being questioned, Steptoe stated that this was the technique used by his anaesthetists in England and that they had not had any problems. Six months later, laparoscopy became a routine procedure in our hospital, and reports of bigeminal rhythm in several cases and a cardiac arrest (successfully resuscitated) prompted us to set up a study to determine, firstly, whether spontaneous ventilation might be dangerous for these patients and, secondly, if so, what the anaesthetic technique of choice should be for this procedure.

BRIEF DESCRIPTION OF THE PROCEDURE

Following the induction of anaesthesia, the surgeon introduces 3 to 5 litres of carbon dioxide into the peritoneal cavity, making sure that the pressure of the gas within the cavity at no time exceeds 25 mm Hg. A large trocar is then introduced through the abdominal wall, and through this the laparoscope is passed. The patient is placed in the Trendelenburg position (15–20°) to move the abdominal viscera up towards the diaphragm, thus leaving the pelvic organs free to be visualized by the surgeon. The procedure lasts from one-half to one hour, depending on whether or not the surgeon decides to do one of the several additional operations now possible with the use of the laparoscope. A small amount of carbon dioxide is permitted to flow into the peritoneal cavity throughout the procedure, to compensate for any leakage that might occur in and around the laparoscope. At the end of the procedure all the carbon dioxide is evacuated through the scope.

*From the Department of Anaesthesia, University of Toronto and the Toronto General Hospital.

METHOD AND MATERIALS

A total of 14 patients selected randomly from the gynaecological service were studied in this series. Ten of the patients (groups I and II) were permitted to breathe spontaneously throughout the study. Induction of anaesthesia was with thiopentone, and maintenance of anaesthesia in this group of patients was carried out with nitrous oxide, halothane ($\frac{1}{2}$ -1 per cent) and oxygen (33 per cent). All the patients were intubated and a non-rebreathing system (Sierra valve) was used in each case. A Wright spirometer on the expiratory side of the valve was used to measure tidal and minute volumes.

Arterial samples were taken for blood gas analysis at induction of anaesthesia and at 5, 15, and 25 minutes following the insufflation of the carbon dioxide into the peritoneal cavity. In five patients (group I) all expired gases were collected in a Douglas bag, both before and after the insufflation of the carbon dioxide. Electrocardiographic monitoring was instituted in all cases. Two patients (group III) were curarized and ventilated by the Radford nomogram and another two patients (group IV) were hyperventilated.

ANALYSIS OF RESULTS

Group I

1. The pre- and post-insufflation expired gas collections showed that four of the patients produced 66-133 ml of carbon dioxide per minute more following the insufflation than before.

2. Minute volumes increased from 50 to 100 per cent during the post-insufflation period.

3. A significant change in respiratory rate following the insufflation was also noted.

4. One patient developed bigeminal rhythm and multi-focal ventricular contractions shortly after the insufflation of the carbon dioxide. The arrhythmia lasted for eight minutes even though the patient was immediately given 100 per cent oxygen.

5. Analysis of the blood gases showed a rise in arterial Pa_{CO_2} to 59 mm Hg in case no. 3.

6. There appeared to be some respiratory and metabolic acidosis present during the post-insufflation period.

7. Low Pa_{O_2} (46-64 mm Hg) was noted in case no. 4 of this group.

Group II

In this group, minute volumes and respiratory rates were recorded frequently with a Wright spirometer, which was attached to the expiratory side of the Sierra valve. Arterial blood gases were taken at the times specified earlier.

1. Two patients in this group had tidal volumes (V_T) of 90 cc.

2. Respiratory rates increased during the post-insufflation period by 75 to 100 per cent, probably accounting in part for the high minute volumes noted in three of the five cases.

GROUP I

	Patient no.				
	1	2	3	4	5
Age	19	30	15	30	23
Premedication (mg)	meperidine 50	diazepam 10	atropine .6	pantopon 20	diazepam 15
Postinduction blood gases	atropine .4	atropine .4		hyosine .6	atropine .4
Thiopentone (mg)	200	250	225	200	225
pH	7.301	7.402	7.3	7.3	7.391
Paco ₂	41.5	34	49	32	30
CO ₂	20.5	21.5	24	22	18
Pao ₂	147	319	141	50	185
BE	-5	-2	-3	-5	-5
f (min)	20	20	22	24	18
Douglas bag pre-insufflation collection (srp)					
V _E (L)	35	66.9	49.6	25.3	38.16
time (min)	12	12	12	10	12
total CO ₂ (ml)	1332	1850	1209	1041	1861
V _{CO₂} (ml)	111	154	100	104	155
Amount of CO ₂ (L)	3	3	3.5	3	5
Pressure of gas in peritoneum (mm Hg)	10-15	15	15	15	15-20
Post-insufflation, 5 min blood gases					
pH	7.265	7.414	7.219	7.31	7.335
Paco ₂	46	33.1	51	51	33
CO ₂	20	21	19	26	17.5
Pao ₂	129	165	96	46	136
BE	-7	-3	-9	-2	-7
f (min)	32	34	28	28	28
Post-insufflation, 15 min blood gases					
pH	7.264	7.403	7.222	7.29	7.364
Paco ₂	45	38.9	55.3	49	34
CO ₂	19	24	22	25	137

GROUP I (concluded)

	Patient no.				
	1	2	3	4	5
Pao ₂	115	168	114	55	137
BE	-7.5	0	-6.5	-4	-5
f (min)	30	48	34	31	28
Post-insufflation, 25 min					
blood gases					
pH	7.255	7.407	7.22	7.29	7.361
Paco ₂	50	35.5	59	51	32
CO ₂					
Pao ₂	21.5	21.5	23	25	18
BE	113	160	105	54	144
f (min)	-6	-3	-6	-3	-6
Douglas bag post-insufflation	30	48	36	29	33
collection (STP)					
VE (L)	57	75.9	69.6	57.2	70.02
time (min)	12	12	12	15	12
total CO ₂ (ml)	2208	2076	2381	2560	3466
Vco ₂ (ml)	184	173	198	170	288
Electrocardiographic					
changes	nil	nil	nil	nil	bigemini and multifocal ventricular contracture for 8 mins
Comparison of amount of CO ₂ expired pre- and post-insufflation (ml/min)	111-184	154-173	100-198	104-170	155-288
Comments	respiratory and metabolic acidosis noted in all cases	marked increase in respiratory rate	marked increase in respiratory rate Paco ₂ 59 mm Hg	very low Pao ₂ (46), respiratory acidosis	serious ventricular arrhythmia for 8 mins; ventilatory rate markedly increased

GROUP II

	Patient no.				
	1	2	3	4	5
Age	25	34	30	27	25
Premedication (mg)	pantopon 15	meperidine 50	atropine 3 i.v.	atropine 4 i.v.	pantopon 20
Postinduction	atropine 6	atropine 6			atropine 6
blood gases	250	250	250	300	250
pH	7.306	7.34	7.421	7.405	7.38
Paco ₂	43	40	33.5	30	42
CO ₂	22	22	22	18	26
Pao ₂	65	149	144	165	280
BE	-4	3.5	-2	-5	-1
ventilation					
mv(L)	6	10	9.1	10.6	4.8
V _T (ml)	250	420	450	350	350
f (min)	24	24	20	28	14
Amount of gas insufflated (L)	3	3	3	4	3
Pressure of gas in peritoneum (mm Hg)	15	15	15	10-15	15
Post-insufflation 5 min					
blood gases					
pH	7.335	7.25	7.35	7.27	7.26
Paco ₂	36	57	40	50	58
CO ₂	19	26	22.5	24	27
Pao ₂	66	146	113	145	90
BE	-6	-4	-3	-5	-2.5
ventilation					
mv (L)	3.6	10	10.8	11.2	3
V _T (ml)	100	380	540	250	200
f (min)	36	26	20	45	15

GROUP II (concluded)

	Patient no.				
	1	2	3	4	5
Post-insufflation 15 min					
blood gases					
pH	7.329	7.31	7.319	7.27	7.29
Paco ₂	37	46	41.7	74	47
CO ₂	19.5	24.5	21.5	34	23.5
Paco ₂	61.5	189	105	135	90
BE	-6	-3	-5.5	+4	-4.5
ventilation					
mv (L)	3.7	14	10.5	12.6	3.9
V _T (ml)	90	540	270	300	260
f (min)	40	26	38	40	15
Post-insufflation, 25 min					
blood gases					
pH	7.343	7.31	7.328		
Paco ₂	38	46	40		
CO ₂	21	24.5	21.5		
Pao ₂	64	149	107		
BE	-4.5	-2.5	-4.5		
ventilation					
mv (L)	3.3	10	10.4		
V _T (ml)	90	450	220		
f (min)	36	22	42		
Electrocardiographic changes	nil	nil	nil	severe sinus bradycardia, rate 40/min	nil
Comment	marked respiratory depression			blood pressure fell to 80 mm Hg and pulse to 40/min; Paco ₂ 74 mm Hg	

3. One patient in this group showed a rise in arterial Pa_{CO_2} (74 mm Hg). The blood pressure fell to 80 mm Hg systolic and sinus bradycardia also occurred (40/min). Fortunately, the procedure terminated soon after this serious state of affairs developed. The blood pressure and pulse rate returned to normal soon after the carbon dioxide had been evacuated from the peritoneal cavity.

4. Some degree of respiratory and metabolic acidosis was also noted in this group.

Group III

The two patients in this group were paralysed, and ventilatory volumes were set by the Radford nomogram. Only slight post-insufflatory increases in arterial Pa_{CO_2} were noted in these patients. The reason for including this group in the series will be elaborated later in the discussion.

Group IV

Hyperventilation was used for both patients in this group.

1. The falls in arterial Pa_{CO_2} were predictable.
2. Respiratory alkalosis was seen in both patients.
3. No changes in blood pressure or pulse rate were noted.

DISCUSSION

Probably the most significant finding to come out of this study is that a large amount of the insufflated carbon dioxide is eventually excreted through the lungs. This being so, a patient who is under-ventilating because of the steep Trendelenburg position and splinted diaphragm might be unable to get rid of the carbon dioxide. The resulting changes in the arterial pH and Pa_{CO_2} must be considered dangerous when halothane is being used in these cases.

The observations in groups I and II indicate that it may be extremely hazardous to permit these patients to ventilate spontaneously. Controlled ventilation is also unsatisfactory if the volumes used are too low, as was the case with the patients in group III. Although we saw no untoward changes in this group, one must suspect that the arterial Pa_{CO_2} could rise to critical levels. We feel that the patients in the hyperventilated group probably received the best anaesthetic management.

Several other problems and questions must be considered:

1. To what extent is the insufflated gas in the peritoneal cavity (15–25 mm Hg) interfering with venous return?
2. Hyperventilation, which we are suggesting as necessary to eliminate the excess carbon dioxide in these patients, will also depress the venous return and the cardiac output, and must therefore be carried out with caution.³
3. The anaesthetist must be constantly aware that carbon dioxide embolism, visceral perforation, and haemorrhage might occur and lead to severe cardiovascular collapse. Perforation of the bowel occurred in one case not included in our series. This patient had had several previous operations, resulting in extensive intra-abdominal adhesions. At the laparotomy which had to be carried out in this case, four perforations were found in the intestine. The rapid onset of

GROUP III

	Patient no.	
	1	2
Age	23	15
Premedication (mg)	diazepam 15 atropine .4	pantopon 15 atropine .6
Thiopentone (mg)	300	200
Postinduction blood gases		
pH	7.46	7.423
Paco ₂	35	34.7
CO ₂	25	21.5
PaO ₂	158	167
BE	+1	-2
ventilation		
mv	6000	4500
V _T (ml)	500	450
f (min)	12	10
Amount of insufflation (L)	3	4
Pressure of gas in peritoneum (mm Hg)	15	10-15
Post-insufflation, 5 min		
blood gases		
pH	7.4	7.321
Paco ₂	38	45.9
CO ₂	23	22
PaO ₂	148	154.5
BE	-1	-4
ventilation		
mv (ml)	6000	4500
V _T (ml)	500	450
f (min)	12	10
Post-insufflation 15 min		
blood gases		
pH	7.399	7.319
Paco ₂	39	48.4
CO ₂	24	23.5
PaO ₂	143	152
BE	0	-3
ventilation		
mv (ml)	6000	4500
V _T (ml)	500	450
f (min)	12	10
Post-insufflation, 25 min		
blood gases		
pH	7.40	7.314
Paco ₂	40	48.4
CO ₂	24	23
PaO ₂	140	152
BE	0	-3
ventilation		
mv (ml)	6000	4500
V _T (ml)	500	450
f (min)	12	10
Electrocardiographic changes	nil	nil
Comment	no significant changes in Paco ₂	slight elevation in Paco ₂ with time

GROUP IV

	Patient no.	
	1	2
Age	23	41
Premedication (mg)	meperidine 100 atropine .4	pantopon 15 atropine .4
Thiopentone (mg)	250	350
Postinduction		
blood gases		
pH	7.524	7.31
P_{aCO_2}	25.5	37
CO_2	21	21.5
P_{aO_2}	170	240
BE	0	-7
ventilation		
MV (L)	15	15
V_T (ml)	1000	1000
f (min)	15	15
Amount of insufflation (L)	3	5
Pressure of gas in peritoneum (mm Hg)	15	15
Post-insufflation 5 min		
blood gases		
pH	7.45	7.32
P_{aCO_2}	31.8	37
CO_2	22	20
P_{aO_2}	151	115
BE	-1	-6
ventilation		
MV (L)	15	15
V_T (ml)	1000	1000
f (min)	15	15
Post-insufflation, 15 min		
blood gases		
pH	7.45	7.38
P_{aCO_2}	28.4	32
CO_2	19.5	20
P_{aO_2}	149.5	130
BE	-3	-5
ventilation		
MV (L)	15	15
V_T (ml)	1000	1000
f (min)	15	15
Post-insufflation, 25 min		
blood gases		
pH	7.44	7.41
P_{aCO_2}	27.5	30
CO_2	22	20
P_{aO_2}	150	126
BE	-5	4.5
ventilation		
MV (L)	15	15
V_T (ml)	1000	1000
f (min)	15	15
Electrocardiographic changes	nil	nil
Comment	progressive respiratory alkalosis	progressive respiratory alkalosis

shock due to perforation in patients who have had previous surgery must be closely watched for at all times. Constant monitoring of these patients is therefore essential.

4. Brown *et al.*⁴ showed that there is severe respiratory impedance following the insufflation of the carbon dioxide even in the ventilated patient. In four cases where a chest X-ray was taken at the times of insufflation we found the diaphragm to be considerably elevated, and this probably accounted for the low tidal volumes seen in some of the patients who were breathing on their own throughout the procedure. We are not able to give any explanation for the metabolic acidosis seen in these patients. However, the venous stagnation which occurs in the upper portion of the body in this steep position could have contributed in part to metabolic acidosis.

SUMMARY

Spontaneous ventilation during laparoscopy is dangerous and must be condemned. Adequate controlled ventilation, sufficient to remove the carbon dioxide being excreted by the lungs, must be instituted in all cases. Some of the more serious complications peculiar to this procedure have been discussed, as these directly affect the anaesthetic management of these patients. These include reduction of cardiac output by impedance of venous return, carbon dioxide embolism, visceral perforation, and haemorrhage.

RÉSUMÉ

La ventilation spontanée durant la laparoscopie est dangereuse et condamnable. Il faut dans tous les cas pratiquer une ventilation contrôlée convenable, suffisante pour éliminer l'acide carbonique excrété par les poumons. On a étudié quelques unes des plus sérieuses complications particulières à cette intervention, et toutes concernent directement la conduite de l'anesthésiste envers ces malades. Parmi ces complications, notons la diminution du débit cardiaque, l'embolie à l'acide carbonique, la perforation viscérale et l'hémorragie.

REFERENCES

1. STEPTOE, PATRICK C. *Laparoscopy in Gynaecology*. E. S. Livingstone Ltd. (1967).
2. STEPTOE, PATRICK C. *Gynaecological Endoscopy: Laparoscopy and Culdoscopy*. J. Obst. & Gynaec. (British Commonwealth). 72: 535 (1965).
3. SEED, R. F.; SHAKESPEARE, T. F.; & MULDOON, M. J. Carbon Dioxide Homeostasis during Anaesthesia for Laparoscopy. *Anaesthesia*. 25: no. 2 (1970).
4. GAYLORD, D. ALEXANDER, FRANCES, E. NOE; & BROWN, ELI M. Pelvic Laparoscopy. *Anesth. & Analg.* 48 (1969).