considerations apply when reviewing the choice of analgesic agent for use with a relaxant in lower abdominal surgery.

The use of diathermy precludes the employment of any inflammable agent, but once again the introduction of pethidine has reduced the indications for trichlorethylene in this sphere.

The use of trichlorethylene as an analgesic vaporized by air in the apparatus commonly used to date is obviously not without risk. Whether these dangers have been completely overcome by the introduction of thermostatically controlled vaporizers remains to be seen.

Conclusions

Pharmacologically, trichlorethylene bears a strong resemblance to chloroform, and while it is not suggested that the incidence of cardiac arrest is at all comparable it may well be that the occurrence is not so rare. Reported cases are indeed few, but it is possible that this is due to a natural reluctance to publicize mishaps. When cardiac arrest occurs during anaesthesia many possible factors may be implicated. As this is an infrequent occurrence in the clinical experience of any one anaesthetist, and as trichlorethylene is reputedly a safe anaesthetic, it is not surprising that the blame in these cases is apportioned elsewhere and the incident passes unrecorded. This is well seen in Case 6 of our series, where if death had occurred the exclusion of air embolism or coronary thrombosis would have been virtually impossible.

We feel that the place of trichlorethylene in modern anaesthesia is very limited, and in the absence of strong indications for its use other, and safer, agents should be employed. When it is used, however, the possibility of cardiac arrest should be borne in mind and the risk balanced against the advantages to be gained. There is still a place for a safe non-inflammable volatile anaesthetic agent should such be discovered.

Summary

A critical survey of the literature pertaining to cardiac arrest during trichlorethylene anaesthesia is presented. Seven previously unpublished cases are reported. The aetiology of the cardiac arrest in these cases is discussed and the possible role of trichlorethylene is emphasized. The scope of trichlorethylene in modern anaesthetic practice is reassessed and the employment of alternative agents whenever possible is recommended.

We are indebted to Dr. R. W. Cope for making available to us further details of the cases already reported by him. We are grateful to our colleagues whose cases are included in this report, and no less to those whose cases have for various reasons been omitted. Finally, we thank Dr. A. C. Forrester and Dr. D. M. Armstrong for their encouragement in the preparation of this paper.

ADDENDUM.—Since this paper was prepared the Committee upon Deaths Associated with Anaesthesia has produced a further report (*Anaesthesia*, 1956, **11**, 194). In this report nine cases of primary cardiac failure are mentioned and a typical case history is given. This type of death accounts for 1.5% of the cases where the anaesthetic is considered to have been responsible for the fatality.

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AN OBSCURE CASE OF RUPTURED DIAPHRAGM

BY

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The following case of herniation of the stomach into the left pleural space may be considered rare enough to be placed on record.

Case Report

A man aged 54 was admitted to hospital as an emergency case on April 26, 1956. He was too distressed to give a history; he was gasping for breath, cold sweat was running down his face, and his extremities were blue and cold. It was noted that the heart pulsations were over to the right of the sternum, the trachea was also displaced to the right, the left chest was moving poorly and air entry could not be heard.

The history relevant to this episode is that he had been admitted to hospital as an acute abdominal emergency on September 9, 1954, and a diagnosis of perforation of a diverticulum of the pelvic colon was made. At operation the peritoneum was found to be full of foul-smelling fluid and a large, immovable inflammatory mass was felt in the pelvic region. Left iliac colostomy was performed and the abdomen drained. After a stormy convalescence, complicated by bilateral bronchopneumonia, he left hospital a month later.

On September 5, 1955, he was again admitted and a diagnosis of right-sided subphrenic abscess was made. At operation, after resecting the 12th right rib, foul-smelling pus was evacuated from the anterior superior space beneath the diaphragm and the space was drained for 15 days. He was discharged 21 days after his operation, and he remained at home, reporting to hospital occasionally, until the catastrophe here reported.

Immediately after the present admission a skiagram of his chest showed almost complete collapse of the left lung, with displacement of the mediastinum to the right, apparently due to an extensive hydropneumothorax (Fig. 1). On viewing the skiagram, the Senior Medical Registrar (Dr. D. C. Watson) passed a needle into the chest posteriorly,

FIG. 1.—Extensive hydropneumothorax with collapse of the lung.

and drew out with great difficulty a small quantity of inspissated pus, but could get no more. When I saw the patient a short time later the picture had not changed except that he was more restless and was grunting with distress. I passed in a long wide-bored needle posteriorly for 2 in. (5 cm.), but no fluid was encountered here.

I then passed a needle anteriorly into the third interspace, above and external to the nipple line, and drew out very easily a syringe of fluid resembling dark beef-tea; this looked like stomach contents, and indeed it was strongly acid to litmus paper. A rubber catheter was inserted in this situation, by means of a trocar and cannula, and attached to a suction apparatus, which aspirated 50 oz. (1,420 ml.) of fluid. At once his condition improved strikingly; a skiagram now showed that the drainage tube was in a large cavity and that the left upper lung had begun to re-expand.

Underwater drainage was continued and a Ryle tube was passed down the oesophagus, being left in for two days while fluids were given by mouth. Nine days later (May 4) he was fit enough to have a barium meal, and this showed that the cavity in which the catheter was lying was the stomach, and that the stomach was within the thoracic cavity (Fig. 2).

On May 9, the catheter being still in the stomach, a barium meal showed that that organ was folded transversely on itself within the thorax, the pylorus and duodenum cap were pointing directly downward, and the stomach was emptying fairly freely into the small bowel.

The patient's general condition now being quite good, it was decided to open the thorax, return the stomach to the abdomen, and repair the diaphragm.

Operation—On May 11 the chest was opened through the bed of the eighth rib on the left side. The stomach was found to be lying in the pleural cavity, with the fundus and anterior wall pressed up against and adherent to the thoracic wall anteriorly. Posteriorly the stomach was firmly adherent to the lung and to a dense inflammatory mass at the back of the chest wall. Considerable difficulty was found in separating the stomach from this mass, and a little pus was encountered in doing so. The diaphragm consisted merely of a ledge of tissue adherent all round to the stomach. The cardia-oesophageal junction was in its normal position, being held by the crus of the diaphragm. After

FIG. 2.—Barium in stomach showing position of this organ in chest. Draining-tube still in place. Note position of cardio-oesophageal junction.

FIG. 3.—Chest condition five and a half weeks after operation. Anterior-posterior view.

adhesions had been separated, the stomach was pushed down through the large aperture in the diaphragm into the abdominal cavity. The aperture was then drawn together by stay sutures and repaired by means of a tongue of intercostal muscle and by sutures of stainless-steel wire. The chest wound was closed, leaving two drainage-tubes for underwater seal drainage, one anteriorly and one posteriorly.

His post-operative course was smooth and without untoward incidents. A skiagram of the chest taken on May 19 showed that the lung had expanded well but there was some residual inflammatory reaction in the left lower lobe and in the pleura at the base of the pleural cavity. A later skiagram June 27 (Fig. 3) showed satisfactory clearing of the chest with no residual pockets.

Five and a half weeks after repair of his diaphragm the patient left hospital in very good condition.

Discussion

Even with this patient's previous history before us, the true nature of the condition was not at first recognized. On admission he appeared to have a spontaneous pneumothorax.

The first pleural paracentesis passed into a mass of old inflammatory tissue and drew off a very small amount of inspissated pus, suggesting an empyema. The skiagram, however, showed a large space filled with gas and air, with an almost completely collapsed lung, and the clue to the diagnosis was given only after a needle in the third intercostal space drew off the dark fluid described. The surprising feature is that passing a large needle through the chest wall into the stomach did not produce leakage of stomach contents into the pleural cavity.

After drainage of the intrathoracic stomach in this way the lung rapidly expanded, the stomach descended towards the abdomen, and the patient was able to eat and drink without discomfort.

After operation there was very little discharge from the two drainage-tubes, and no loculations of fluid or air were left in the pleural cavity.

The sequence of events leading to rupture of his diaphragm is shown in his history. First, the peritoneal catastrophe of a ruptured diverticulitis, then a subphrenic abscess, which presented on the side opposite to the subsequently

ruptured diaphragm. It is probable that there was a left subphrenic abscess at about the same time and that infection from this site tracked up through the foramen of Blakdale into the chest. This would account for the inflammatory mass at the posterior aspect of the left chest at the site of the first paracentesis and for the subsequent softening, and finally the rupture, of the diaphragm and the sudden passage of the stomach into the chest.

EFFECT OF CHLORPROMAZINE ON RENAL HAEMODYNAMICS AND FUNCTION IN CONGESTIVE HEART FAILURE

PRELIMINARY REPORT

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On the basis of an earlier observation of ours that chlorpromazine ("largactil") has a marked diuretic action in cases of congestive heart failure we investigated 12 cardiac patients and, for comparison, 6 normal persons for the effect of chlorpromazine (25 mg. intravenously) on diuresis, sodium excretion, glomerular filtration (endogenous creatinine clearance), and renal plasma flow (para-aminohippuric acid clearance).

Results

After the intravenous injection of the above dose an immediate increase in diuresis was seen in oedematous cardiac patients, reaching its maximum within about half an hour. The rise averaged two or three times the initial value, and in our 12 cases the average of the maximal diuresis rose to four times as much. Even the lowest values due to chlorpromazine were much higher than those observed in the control periods (Tables I and II).

Sodium excretion varied together with the diuresis, rising upon administration of chlorpromazine in general to more than twice that observed in the control periods. The peak values in some of the experiments reached three times those of the control periods. In the chlorpromazine-treated cases even the lowest sodium excretion was significantly higher than the control values.

The glomerular filtration rate also increased as the result of chlorpromazine, though by no means to the extent seen in diuresis and sodium excretion. In the periods after chlorpromazine administration the glomerular filtration rate was some 20 to 50% higher than the control values, the rise being statistically significant in only the first two periods after administration of the drug. The maximal glomerular filtration rate values in the periods after chlorpromazine were on the average 86% higher than in the controls. The minimal values were 9.5% lower than the control values, and are not statistically significant.

Chlorpromazine produced an average rise of 30% in the renal plasma flow, but this rise, except for the first period after administration, cannot be regarded as statistically significant. The average of maximal values due to chlorpromazine exceeded the controls by 69%, the difference being highly significant. The mean of the lowest values was actually 12% lower than the controls, but from the standpoint of statistical analysis was not substantially different.

Comment

From the above, therefore, it appears that in congestive heart failure chlorpromazine has a pronounced diuretic and

TABLE I.—Results in 12 Cases

Case No.	Diuresis (ml./min.)						Glomerular Filtration						P.A.H. Clearance						Sodium Excretion (mg./min.)					
	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6	1	2	3	4	5	6
1	0.5	0.6	2.3	1.0	1.6	1.2	85	90	152	48	74	51	300	390	620	200	227	180	2.5	3.4	12.0	5.4	8.5	6.1
2	0.9	1.4	1.8	2.7	2.6	3.2	77	86	83	93	74	87	190	280	200	310	250	300	0.8	1.3	1.6	2.2	1.6	2.2
3	0.3	0.4	0.3	0.3	1.0	1.6	74	102	74	44	105	91	—	260	250	173	380	350	0.3	0.3	0.2	0.3	1.5	1.4
4	0.5	0.6	0.5	1.0	0.9	0.9	73	86	51	102	81	88	—	230	149	323	240	180	3.1	3.9	3.3	6.6	6.7	7.5
5	0.4	0.5	0.6	5.9	2.2	3.8	46	49	65	177	55	100	—	—	—	—	—	—	2.3	3.3	1.5	13.3	5.5	9.5
6	0.7	0.8	1.5	1.6	0.9	0.7	34	35	50	28	23	28	103	162	204	133	118	—	2.9	2.6	3.6	2.5	2.0	2.7
7	1.3	1.3	2.6	2.2	1.6	—	60	60	130	89	72	—	218	219	390	331	199	—	4.4	5.3	9.2	7.7	5.1	—
8	0.8	0.9	1.5	1.1	1.1	—	60	63	112	74	82	—	—	434	560	462	590	—	3.5	4.8	7.8	7.1	5.1	—
9	0.5	0.4	1.6	1.9	1.4	1.0	60	57	160	179	140	—	138	156	493	530	510	264	1.6	1.6	6.1	7.4	5.7	3.8
10	0.9	1.3	6.5	4.7	4.2	—	113	109	142	85	77	—	272	—	280	178	166	—	1.3	1.6	8.8	6.6	6.6	—
11	0.5	0.4	1.1	0.9	1.1	0.6	68	78	185	148	171	102	183	171	405	352	381	233	0.4	0.4	0.9	0.9	1.1	0.6
12	1.5	1.4	1.0	2.1	6.0	6.0	108	91	48	90	91	91	318	341	112	254	320	286	6.8	6.4	3.6	6.4	7.3	5.5

1 and 2=Control periods. 3, 4, 5, and 6=Periods after administration of chlorpromazine. P.A.H.=Para-aminohippuric acid.

TABLE II.—Effect of Chlorpromazine on Glomerular Filtration Rate, Renal Plasma Flow, and Water and Salt Excretion, Expressed as Percentage of Mean of Control Periods

		1	2	3	4	5	6	Min.	Max.
Diuresis (ml./min.)	Mean	94	106	216	298	260	301	146	396
	S.D. t P	9.5	9.3	164 3.491 ≤0.1	95 10.225 ≤0.1	124 6.357 ≤0.1	207 4.738 0.1	82 2.726 1.0	298 4.905 ≤0.1
Glomerular filtration rate	Mean	97	103	151	145	122	116	90.5	186
	S.D. t P	6.2	6.2	73 3.511 0.1	100 2.224 3.0	58 1.870 7.0	38 1.611 10.0	43 1.073 30.0	100 4.971 ≤0.1
P.A.H. clearance	Mean	92	106	138	133	133	112	88	169
	S.D. t P	9.3	8.8	71 2.259 3.0	86 1.616 12.0	83 1.690 10.0	42 1.156 30.0	39 1.241 25.0	73 3.970 ≤0.1
Sodium excretion (mg./min.)	Mean	92	108	211	213	234	210	141	311
	S.D. t P	9.6	9.6	136 3.852 ≤0.1	133 4.014 ≤0.1	135 4.682 ≤0.1	129 4.067 ≤0.1	101 1.903 5.0	159 6.270 ≤0.1

1 and 2=Control periods. 3, 4, 5, and 6=Periods after chlorpromazine administration. Min.=Mean of individual minimal values after chlorpromazine administration. Max.=Mean of the individual maximal values after chlorpromazine administration.