A STUDY OF CHLOROFORM ANAESTHESIA IN A PRECISION SYSTEM:

II. THE EFFECT ON CIRCULATORY DYNAMICS AND ANAESTHETIC MORBIDITY¹

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SINCE THE GENERAL ACCEPTANCE of halothane, several authors have speculated about the possibility of reintroducing chloroform as a clinical agent, because the chemical and physical properties of these two are akin. It was felt that any new clinical studies of chloroform must be done with the same care and attention to detail as have been found necessary with halothane since the physiological and pathological effects on vital organs appear to be similar. This report deals with the effect of chloroform on circulatory dynamics and post-anaesthetic morbidity when it was used clinically in a precision system.

MATERIALS AND METHODS

Data were collected from 154 patients who underwent major operations. Anaesthetic management was the same as described in the previous report (1).

Circulatory dynamics were observed closely in each operation. Pulse rate and arterial blood pressure were recorded at 5 min. intervals by palpation and auscultation respectively (2). The electrocardiogram (lead 2) was monitored in those patients with evidence of heart disease. The mean blood pressures and pulse rates during maintenance were obtained by averaging the individual readings, starting 10 min. after induction of anaesthesia, and ending when chloroform administration was discontinued at the end of the operation. The recordings noted during the 10 min. period immediately following induction of anaesthesia were tabulated separately, as were the pre-induction recordings, and those at the end of the operation. Stroke volume and cardiac output were estimated from recordings of the vital signs (3).

Post-anaesthetic morbidity and mortality were recorded and reviewed in each case.

RESULTS

Circulatory Dynamics

Analyses of the variation in blood pressure, pulse rate, estimated stroke volume, and cardiac output are shown in Table I. Figure 1 shows the mean variation and standard deviation for two groups of patients who underwent

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TABLE I

**CIRCULATORY DYNAMICS DURING CHLOROFORM-NITROUS-OXIDE ANAESTHESIA IN A NON-REBREATHING SYSTEM AND ARTIFICIAL RESPIRATION
A. GASTRECTOMY AND INTRATHORACIC OPERATIONS IN SUPINE HORIZONTAL AND LATERAL FLEXED POSITION (41 PATIENTS)**

Age	Sex	Build	Physical state	Anaesthesia time (min.)	Muscle relaxant	Before anaesthesia				Induction				Maintenance				End of anaesthesia							
						Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)
30	M	M	2	85	G	124	72	120	66	7.9	126	72	122	69	8.2	120	78	140	57	8.0	126	86	140	52	7.2
35	M	O	3	150	dMC	104	76	100	50	5.0	124	82	104	52	5.4	112	82	106	47	4.7	112	82	106	47	4.9
37	M	M	1	150	G	108	60	62	67	4.1	110	68	86	60	5.1	122	80	98	53	5.2	140	88	80	53	4.2
39	F	M	1	145	G	114	70	80	60	4.8	124	74	90	60	5.4	120	80	108	51	5.5	122	80	90	52	4.7
39	M	O	2	280	G	110	74	60	52	3.1	112	76	74	50	3.7	132	96	116	38	4.0	128	92	120	41	4.9
41	F	L	2	125	dMC	90	70	100	45	4.5	110	76	104	48	5.0	116	80	82	47	3.8	130	86	80	47	3.8
41	M	O	2	240	G	100	70	70	50	3.5	100	70	84	50	4.2	90	70	86	45	3.9	100	74	80	46	3.7
44	M	L	2	215	dMC	106	70	80	51	4.1	106	74	80	47	3.7	146	100	80	39	3.2	160	100	86	45	4.0
45	M	O	2	145	G	120	80	100	47	4.7	120	80	100	47	4.7	116	84	120	40	4.8	120	90	110	35	3.9
45	M	M	1	145	G	120	76	90	51	4.7	124	80	92	47	4.3	126	90	102	40	4.2	128	90	110	40	4.4
45	M	O	2	138	dMC	138	100	80	35	2.8	140	100	100	35	3.5	144	110	130	26	3.3	152	108	132	32	4.2
45	M	M	3	125	G	106	70	90	51	4.6	106	76	102	44	4.5	90	60	96	53	5.1	96	70	90	46	4.1
48	F	L	3	135	G	110	64	90	52	5.1	106	64	92	55	5.0	120	80	92	45	4.0	116	78	90	53	4.2
49	F	O	2	150	dMC	90	60	70	51	3.6	90	60	80	51	4.7	102	66	64	51	3.2	120	80	84	45	3.8
49	M	O	3	120	dMC	116	90	70	31	2.1	130	100	94	27	1.8	134	94	64	36	2.2	150	100	68	37	2.5
54	M	O	2	155	G	140	90	82	40	3.2	132	88	98	38	3.7	152	102	90	34	3.0	162	110	80	30	2.3
54	M	O	2	290	dMC	160	104	106	35	3.7	156	100	110	37	4.1	150	100	130	35	4.5	140	90	150	40	6.1
55	M	O	2	250	G	114	65	90	50	4.5	106	65	90	49	4.4	94	66	110	43	4.7	106	70	100	44	4.4
55	M	M	3	135	G	200	120	100	37	3.7	200	120	100	37	3.7	190	120	108	32	3.5	220	140	100	28	2.8
56	F	L	2	230	dMC	130	80	110	45	4.9	140	90	120	39	4.7	150	106	92	25	2.0	150	100	100	32	3.2
57	F	M	3	255	G	136	90	80	37	3.0	130	90	84	34	2.7	118	82	102	36	3.7	120	84	100	35	3.5
58	F	O	2	180	G	120	90	80	21	2.1	110	76	92	38	3.5	124	80	100	41	4.1	130	76	100	48	4.8
58	M	L	3	80	G	110	80	88	32	2.8	118	86	106	31	3.2	130	92	116	30	3.4	132	90	120	34	4.0
58	M	M	1	190	G	120	80	70	38	2.7	130	90	90	32	2.8	130	84	90	40	3.7	140	92	100	35	3.5
58	F	M	3	165	dMC	160	94	96	43	4.1	200	110	84	46	3.9	250	120	80	60	4.8	260	120	90	59	5.5
59	M	L	2	75	G	112	64	80	51	4.1	118	74	94	44	4.1	130	93	108	29	3.1	130	94	102	28	2.7
59	M	O	2	150	G	130	80	100	43	4.3	124	80	94	40	3.7	128	88	108	32	3.5	130	90	100	33	3.3
59	M	O	2	185	G	130	80	82	43	3.5	126	88	90	32	2.9	120	86	98	31	3.0	132	90	100	33	3.3
60	M	M	2	310	G	130	70	90	53	4.8	114	74	96	41	3.4	100	74	120	35	4.2	96	70	124	36	4.4
61	M	L	3	290	G	104	60	80	37	3.0	96	64	96	42	4.1	104	78	100	31	3.1	102	78	100	30	2.9
61	M	M	1	140	G	106	80	70	29	2.0	100	70	90	38	3.5	140	100	80	27	2.0	150	92	80	37	3.5
63	M	M	3	180	dMC	130	90	70	29	2.0	122	80	82	36	2.9	138	92	84	36	3.0	140	88	90	37	3.3
64	M	M	3	180	dMC	100	60	64	47	3.0	138	84	72	42	3.0	154	96	80	33	2.6	140	92	82	32	2.5
67	M	L	1	190	dMC	140	80	82	43	3.5	140	80	90	44	4.0	116	74	76	38	3.0	140	66	80	44	3.5
68	F	M	2	75	G	150	90	80	36	2.8	135	88	82	31	2.5	140	92	98	29	2.8	148	100	88	24	2.0
70	M	O	3	135	G	180	90	100	50	5.0	140	82	108	41	4.2	144	90	112	32	3.6	160	100	110	24	2.5
71	M	L	2	125	G	140	80	100	41	4.1	126	74	82	40	3.2	130	92	118	22	2.5	140	86	120	35	4.1
73	F	O	3	270	G	140	70	80	51	4.1	130	70	110	46	5.1	120	76	110	34	3.8	110	70	120	36	4.2
75	M	L	3	175	dMC	70	60	80	35	2.8	100	70	90	29	2.6	122	72	70	38	2.6	140	90	74	27	1.3
75	M	M	2	155	G	140	60	90	61	5.5	130	60	100	55	5.5	176	96	116	38	4.4	174	100	120	34	4.1
79	F	L	3	120	dMC	180	90	160	45	7.3	155	84	120	39	4.7	160	90	124	35	4.2	180	92	118	42	5.0
Mean				174		126	78	87	45	4.0	125	79	94	43	4.0	132	88	100	39	3.8	137	90	100	39	3.8
S.D.						25	14	20	10	1.3	23	13	12	9	1.1	29	14	18	9	1.1	30	14	19	9	1.1
S.E.						4	2	3	2	0.2	4	2	2	1	0.2	5	2	3	1	0.2	5	2	3	1	0.2

TABLE I (continued)
B. CHOLECYSTECTOMY AND RELATED OPERATIONS IN SUPINE HORIZONTAL POSITION (29 PATIENTS)

Age	Sex	Build	Physical state	Anaesthesia time (min.)	Muscle relaxant	Before anaesthesia				Induction				Maintenance				End of anaesthesia				
						Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)		
18	F	O	1	90	dMC	100	70	62	64	3.9	104	70	90	66	5.9	98	68	82	65	5.2	74	5.2
23	F	M	2	75	dMC	118	80	70	59	4.1	104	72	72	61	4.2	96	64	80	76	6.1	82	5.6
33	F	M	1	170	dMC	102	64	78	63	4.9	106	68	82	60	4.9	110	64	74	62	4.6	72	4.7
34	M	M	1	130	dMC	120	78	90	55	5.0	120	80	90	53	5.2	124	94	90	41	3.7	100	3.7
34	F	M	1	105	G	120	70	80	65	5.2	108	64	120	65	7.2	110	70	130	59	7.6	118	6.5
35	F	O	2	110	G	110	60	70	69	4.8	108	60	96	69	6.6	122	84	120	50	6.0	108	4.9
38	F	M	1	145	G	108	68	88	63	5.6	108	72	96	54	5.2	124	90	100	42	4.2	132	4.6
40	F	M	1	90	dMC	100	70	60	50	3.0	96	62	72	57	4.1	104	70	68	53	3.6	78	3.7
46	F	O	2	155	G	124	72	80	57	4.6	122	78	100	49	4.9	126	80	122	49	6.0	134	5.7
51	M	O	2	155	dMC	140	78	84	55	4.6	116	72	96	50	4.8	130	90	94	36	3.3	134	5.1
53	F	O	2	60	G	128	70	80	57	4.6	120	74	94	48	4.5	118	80	104	41	4.2	118	4.0
54	F	M	1	115	G	110	66	90	58	4.7	104	62	84	53	4.4	98	66	110	46	5.1	108	4.9
56	F	M	1	205	G	126	80	90	43	3.9	130	84	96	41	3.9	118	78	82	41	3.3	130	3.7
56	F	O	2	110	G	136	80	80	48	3.9	128	80	90	44	4.0	126	80	92	43	4.0	140	2.8
57	F	O	2	230	G	110	70	72	46	3.4	106	70	78	43	3.4	104	72	80	38	3.0	102	3.2
57	M	O	3	150	G	120	60	72	62	4.4	124	74	74	48	3.5	130	80	126	45	5.6	120	5.9
59	M	O	2	90	G	130	70	60	54	3.3	122	74	92	46	4.2	112	76	120	39	4.6	110	5.3
60	M	M	1	120	G	122	74	68	45	3.0	120	78	84	40	3.3	126	90	128	29	3.7	136	3.6
63	M	O	3	225	G	128	70	80	51	4.1	128	68	90	52	4.7	124	80	98	37	3.6	138	3.4
63	F	O	2	215	G	130	70	72	51	3.7	124	72	86	46	4.0	126	86	94	32	2.9	136	2.6
65	M	O	3	105	G	170	90	80	48	3.9	176	94	96	47	4.5	164	92	92	43	4.0	170	3.1
68	F	M	2	100	G	102	80	70	24	1.6	104	76	72	29	2.0	104	68	80	37	3.0	100	3.1
68	F	M	3	95	G	120	84	80	29	2.2	110	76	88	32	2.8	112	70	82	39	3.1	122	3.0
71	M	L	3	170	G	118	68	70	43	3.0	110	60	70	48	3.4	102	72	108	30	3.1	110	3.6
71	M	L	3	80	G	90	60	62	38	2.1	90	60	86	37	3.2	106	72	100	32	3.2	112	2.5
72	F	M	2	105	G	140	80	100	40	4.0	138	78	98	42	4.1	124	76	106	36	3.8	140	4.1
73	M	O	4	170	dMC	138	80	90	38	3.4	144	80	90	42	3.8	144	78	86	44	3.8	130	3.2
78	M	O	3	125	G	160	90	100	36	3.6	146	88	102	30	3.0	150	94	100	26	2.6	160	2.1
83	F	M	3	145	G	124	68	120	38	4.6	124	70	120	36	4.4	110	60	120	40	4.8	122	4.1
Mean				132		122	73	79	50	3.9	119	73	90	48	4.3	119	77	99	43	4.2	124	4.0
S.D.						17	8	13	11	.9	17	8	12	10	1.0	16	10	17	11	1.2	16	1.1
S.E.						3	2	3	2	.2	3	2	2	2	0.2	3	2	3	2	0.2	3	0.2

TABLE 1 (continued)

C. LOWER ABDOMINAL AND ABDOMINAL PERINEAL OPERATIONS IN TRENDLENBERG AND HEAD LOW—LITHOTOMY POSITION (44 PATIENTS)

Age	Sex	Build	Physical state	Anaesthesia time (min.)	Muscle relaxant	Before anaesthesia				Induction				Maintenance				End of anaesthesia							
						Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)					
20	M	O	2	60	dMC	100	62	86	72	6.1	108	70	96	67	6.4	110	68	90	70	6.3	112	62	76	78	5.8
26	F	M	1	145	G	100	68	98	62	6.0	120	88	102	50	8.2	112	84	112	50	5.7	120	78	110	61	6.8
28	F	M	1	50	G	108	60	118	72	8.5	104	60	122	71	8.7	108	68	122	64	7.8	120	70	110	68	7.5
32	F	O	2	135	dMC	90	60	70	61	4.3	110	76	72	54	3.9	110	76	74	54	4.0	110	72	82	58	4.7
36	F	M	1	90	dMC	110	70	100	58	5.8	100	70	110	53	5.8	108	72	90	55	5.0	112	80	74	48	3.5
37	F	M	1	45	dMC	138	90	100	50	5.0	126	92	102	41	4.2	112	90	86	37	3.1	110	84	84	41	3.4
40	F	M	1	155	dMC	126	78	92	51	4.7	104	70	96	52	5.0	110	76	80	46	3.7	110	76	80	49	4.0
41	F	M	1	155	dMC	100	60	72	61	4.3	106	64	86	59	5.1	100	60	72	61	4.3	100	60	90	61	5.5
41	F	M	1	165	dMC	120	90	78	38	2.9	126	90	74	41	3.0	108	74	96	49	4.6	126	80	92	51	4.7
43	F	L	1	95	G	138	82	96	53	5.1	128	82	98	48	4.7	104	72	96	47	4.6	138	90	102	45	4.6
43	F	M	1	100	G	110	80	100	42	4.2	108	78	100	44	4.4	102	76	110	43	4.7	100	70	110	49	5.3
44	F	M	1	95	G	106	74	72	47	3.3	116	76	96	50	4.8	106	70	108	52	5.6	110	60	100	64	6.4
45	F	M	2	75	G	120	80	86	48	4.1	132	94	96	38	3.7	130	98	110	35	3.9	136	100	110	35	3.9
45	F	O	2	145	G	124	86	90	42	3.7	120	82	92	44	4.1	122	90	92	36	3.3	122	86	78	42	3.2
46	F	M	1	105	dMC	128	80	70	50	3.5	130	82	72	49	3.5	142	88	68	49	3.3	168	100	72	48	3.5
47	F	O	2	100	G	100	68	72	55	3.9	110	70	84	58	4.3	122	82	100	44	4.4	128	84	100	45	4.5
47	F	M	1	95	G	108	80	94	40	3.7	116	80	108	44	4.7	124	86	120	44	4.4	130	90	100	40	4.0
50	F	M	2	125	dMC	130	70	98	59	5.8	136	84	98	47	4.6	142	98	80	35	2.7	140	92	76	41	3.1
50	F	M	1	130	G	92	60	48	41	4.1	108	82	96	37	3.6	100	72	102	40	4.1	102	80	100	32	3.2
54	M	M	3	165	dMC	148	80	140	55	7.7	146	88	124	45	5.6	140	86	100	45	4.5	130	82	100	44	4.4
54	F	M	2	205	G	150	86	82	49	4.0	154	88	80	49	3.9	130	86	82	40	3.2	122	76	90	47	4.2
54	M	O	2	75	G	130	80	82	47	3.9	142	84	84	49	4.1	124	68	104	57	6.0	130	64	90	63	5.7
55	F	O	2	105	G	138	70	92	61	5.5	148	102	100	31	3.1	136	100	90	27	2.4	132	102	92	23	2.0
56	F	L	2	60	G	160	100	90	38	3.5	148	100	90	32	3.0	130	90	84	35	3.0	130	90	100	35	3.5
56	F	M	2	190	G	120	60	80	62	5.0	120	62	100	60	6.0	114	64	100	54	5.4	124	70	80	53	4.2
57	F	O	3	250	G	118	70	80	49	4.0	106	60	90	55	5.0	104	70	90	43	4.0	106	70	80	45	3.6
57	M	O	2	225	G	116	78	82	41	3.5	116	78	86	41	3.6	108	92	92	20	1.8	140	90	100	39	3.9
59	M	M	3	110	G	148	80	82	58	4.7	124	72	110	48	5.3	120	80	108	38	4.0	138	90	94	36	3.3
61	F	O	3	195	G	84	60	92	41	3.7	94	66	92	40	3.7	122	90	94	27	2.4	128	92	100	27	2.7
62	F	O	3	290	G	146	72	110	57	6.3	150	78	110	55	6.0	136	82	114	41	4.7	122	72	106	45	4.8
64	F	O	2	75	dMC	170	100	90	38	3.4	184	106	94	39	3.6	206	112	82	43	3.3	216	112	82	47	3.9
66	F	O	2	230	G	120	80	100	25	2.5	108	74	100	35	3.5	110	70	92	40	3.7	130	84	90	35	3.1
67	F	O	2	145	G	120	80	100	34	3.4	100	70	106	35	3.7	160	96	106	36	3.8	164	100	104	33	3.3
71	M	M	3	250	dMC	190	90	80	55	4.4	162	84	76	47	3.6	144	88	76	34	2.5	170	110	86	23	1.7
71	M	O	3	150	dMC	128	90	78	49	3.9	100	76	74	25	1.7	116	76	76	33	2.5	108	70	82	36	2.9
72	F	O	4	195	G	130	90	120	24	2.8	120	70	110	41	4.5	160	90	100	40	4.0	170	110	100	23	2.3
73	M	M	2	170	G	120	86	100	24	2.4	126	82	82	30	2.2	124	84	114	27	2.9	138	90	118	27	3.1
73	F	M	3	330	G	120	70	90	40	3.6	120	68	84	43	3.6	106	64	80	39	3.0	110	70	84	36	2.9
74	F	M	4	130	dMC	140	80	110	39	4.3	118	68	94	41	3.8	106	68	88	35	3.0	112	70	90	36	3.1
75	F	M	3	80	G	150	90	82	33	2.5	170	98	94	34	3.1	164	106	96	22	2.0	180	108	100	28	2.8
76	F	M	1	135	dMC	128	70	80	43	3.4	116	66	78	41	3.1	110	68	78	36	2.7	116	66	80	41	3.3
76	F	M	3	60	dMC	180	92	80	44	3.5	164	92	82	37	3.0	184	108	82	29	2.4	198	110	84	34	2.7
81	F	M	3	80	dMC	104	70	78	28	2.0	90	50	82	42	3.4	100	62	90	34	3.0	122	80	92	26	2.1
91	M	M	2	170	dMC	160	70	76	50	3.8	126	72	70	30	2.1	148	82	66	39	2.1	168	82	68	40	2.6
Mean				140		128	77	90	47	4.2	124	78	94	45	4.3	125	81	93	41	3.8	132	84	92	43	3.9
S.D.						25	11	15	12	1.3	21	12	17	10	1.4	23	13	14	11	1.3	26	15	12	13	1.3
S.E.						4	2	2	2	0.2	3	2	3	2	0.2	4	2	2	2	0.2	4	2	2	2	0.2

TABLE I (continued)
D. SPINAL OPERATIONS IN PRONE POSITION (24 PATIENTS)

Age	Sex	Build	Physical state	Anaesthesia time (min.)	Muscle relaxant	Before anaesthesia				Induction				Maintenance				End of anaesthesia							
						Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)					
14	M	M	2	180	dMC	110	80	110	61	6.7	116	78	106	66	7.0	112	84	76	57	4.4	112	88	80	53	4.2
14	M	M	2	300	G	130	90	110	59	6.5	118	90	140	54	7.6	120	90	120	55	6.6	110	80	118	61	7.7
21	M	L	2	115	dMC	110	70	80	68	5.4	110	70	80	67	5.3	114	82	82	56	4.6	120	90	92	50	4.1
22	F	M	1	185	G	120	74	82	68	5.5	116	72	110	68	7.5	108	80	96	50	4.8	110	80	98	56	5.5
23	M	M	1	190	G	110	70	72	66	4.7	100	70	78	61	4.7	104	78	94	55	5.2	114	90	82	46	3.6
28	F	O	2	225	dMC	88	60	68	63	4.3	92	62	68	62	4.3	96	74	72	51	3.5	102	64	80	66	5.2
28	M	M	1	240	dMC	118	80	60	56	3.4	120	82	60	54	3.2	118	94	76	40	3.0	116	86	74	48	3.5
30	M	O	2	370	G	100	60	80	67	5.3	100	62	80	65	5.2	104	72	94	56	5.3	92	66	84	57	4.8
35	M	M	1	350	G	108	78	74	49	3.6	112	80	124	49	6.0	116	82	122	48	5.8	112	82	128	47	6.0
39	F	O	2	105	dMC	128	78	82	56	4.4	128	78	84	57	4.8	130	90	77	45	3.4	140	100	82	39	3.1
39	M	M	2	330	dMC	90	60	64	57	3.3	92	62	68	56	3.9	98	66	74	54	4.0	96	60	72	60	4.3
42	M	M	2	175	dMC	130	90	80	44	3.6	90	76	70	38	2.6	122	96	78	35	2.6	118	92	78	34	2.6
42	M	M	1	225	G	128	80	90	53	4.8	130	80	90	54	4.9	118	90	84	36	2.6	110	82	92	41	3.8
45	M	M	1	340	dMC	140	80	90	56	5.2	140	90	96	46	4.4	118	82	64	44	2.7	116	80	58	45	2.6
45	M	M	1	320	dMC	102	68	60	52	3.2	102	68	60	51	3.1	114	91	78	32	2.4	110	80	76	42	3.1
47	M	O	2	85	dMC	116	80	72	44	3.0	116	84	72	39	2.8	118	70	76	56	4.2	120	64	68	63	4.3
47	F	L	1	90	dMC	130	80	104	51	5.3	130	80	104	51	5.3	112	74	82	48	3.9	110	78	68	43	2.9
50	M	O	2	305	dMC	84	54	60	53	3.2	84	52	60	57	3.4	90	54	88	57	5.0	90	50	100	61	6.1
50	M	O	3	410	dMC	110	68	80	52	4.1	98	66	80	48	3.8	90	68	76	41	3.1	92	64	78	46	3.6
51	M	M	1	180	dMC	100	70	60	43	2.6	100	72	72	42	3.0	90	86	104	31	3.2	108	87	118	28	3.2
53	F	O	3	435	G	110	78	82	38	2.9	102	80	78	32	2.4	110	74	82	37	3.0	90	50	80	55	4.4
60	M	O	2	275	G	120	70	90	48	4.3	120	70	100	48	4.8	108	94	82	27	2.5	138	96	108	27	2.8
62	F	M	1	135	dMC	120	92	80	23	1.8	120	90	100	25	2.5	132	94	95	27	2.5	138	96	108	27	2.8
62	F	O	3	320	dMC	150	96	100	33	3.3	134	90	80	32	2.5	116	90	86	23	1.9	112	84	92	27	2.5
40				245		115	75	80	53	4.1	111	75	86	51	4.4	112	81	85	45	3.8	110	78	86	47	4.0
Mean						115	75	80	53	4.1	111	75	86	51	4.4	112	81	85	45	3.8	110	78	86	47	4.0
S.D.						16	11	15	11	1.5	15	10	21	12	1.6	11	10	15	11	1.2	13	14	18	11	1.4
S.E.						3	2	3	2	0.3	3	2	4	2	0.3	2	2	3	2	0.3	3	3	4	2	0.3

TABLE I (continued)
E. MISCELLANEOUS SUPERFICIAL OPERATIONS IN SUPINE HORIZONTAL POSITION (16 PATIENTS)

Age	Sex	Build	Physical state	Anaesthesia time (min.)	Before anaesthesia				Induction				Maintenance				End of anaesthesia								
					Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)	Systolic BP (mm. Hg)	Diastolic BP (mm. Hg)	Pulse rate (min.)	Stroke volume (ml.)	Cardiac output (L.)						
16	F	M	1	80	G	120	80	118	65	7.7	116	76	120	67	8.0	124	86	126	59	6.3	130	84	120	65	7.8
17	M	M	1	170	dMC	106	60	60	81	4.7	94	58	72	76	5.4	92	60	70	72	5.1	96	64	70	70	4.9
18	M	M	2	280	dMC	112	60	74	80	6.1	110	64	78	76	5.9	100	58	98	79	7.7	100	60	100	75	7.5
22	F	M	1	175	dMC	110	70	80	67	5.3	110	68	78	70	5.5	112	76	80	61	4.9	120	84	88	56	4.9
41	F	M	1	195	G	110	88	92	36	3.1	110	90	96	33	3.1	116	90	108	37	3.8	116	92	110	33	3.7
46	M	O	2	70	dMC	128	90	70	41	2.8	128	90	78	39	3.0	130	86	78	45	3.5	136	88	82	46	3.8
48	F	M	2	110	dMC	140	80	72	55	3.9	130	82	82	47	3.7	132	90	80	39	3.1	140	92	88	40	3.6
49	F	M	1	170	dMC	100	60	96	57	5.4	118	80	90	49	4.4	124	90	80	35	2.7	106	76	80	42	3.4
50	M	O	2	110	dMC	128	94	88	30	2.8	140	96	108	35	3.7	142	94	72	39	2.8	148	96	70	40	2.8
54	M	M	1	40	dMC	110	80	70	37	2.6	110	80	70	36	2.5	118	84	90	36	3.2	120	86	80	35	2.8
62	M	M	1	155	dMC	128	70	80	50	4.2	120	74	84	43	3.2	124	70	78	49	3.9	140	86	84	39	3.2
64	F	M	2	115	G	170	102	68	35	2.0	154	90	70	41	2.9	166	114	90	32	2.8	152	110	92	20	1.7
64	F	O	2	110	G	128	82	78	35	2.6	128	80	74	39	2.5	170	104	112	33	3.7	180	110	92	33	3.0
71	M	M	3	95	G	100	80	90	21	2.0	106	88	102	16	1.6	108	86	120	23	2.7	106	82	130	21	2.7
71	M	M	4	155	dMC	158	110	94	16	1.4	120	60	100	53	5.3	130	80	112	36	4.0	140	80	118	41	4.8
74	F	O	4	150	dMC	160	100	80	27	2.1	140	90	86	28	2.4	140	98	86	19	1.6	150	100	90	23	2.0
Mean				136		126	82	82	46	3.7	121	79	87	47	4.0	113	85	93	43	3.9	130	87	94	43	3.9
S.D.						22	15	14	20	1.8	15	12	15	18	1.7	21	15	18	17	1.5	23	14	18	17	1.7
S.E.						5	4	3	5	0.1	4	3	4	4	0.1	5	4	5	4	0.1	6	3	4	4	0.1

MEAN CIRCULATORY EFFECTS OF CHLOROFORM- N_2O ANAESTHESIA during Gastrectomy & Intrathoracic operations in 41 patients, mean age 55 yrs.) MEAN CIRCULATORY EFFECTS OF CHLOROFORM- N_2O ANAESTHESIA (during Lower Abdominal & Abdominal-perineal operations in 44 pts., mean age 55)

FIGURE 1

gastrectomy and/or intrathoracic operations, and those who had lower abdominal operations.

There was little alteration in the systolic and diastolic blood pressure. The pulse rate usually rose slightly. This rise was most often in response to either intravenous gallamine or atropine, which blocked the tendency toward bradycardia that is seen characteristically with chloroform. The only significant change was a moderate reduction in the estimated stroke volume, while the cardiac output decreased slightly in a few patients.

There was clinical and ECG evidence of moderate to severe heart disease in 24 patients, of whom 11 had a previous history of coronary thrombosis or cerebral vascular accidents, and three others who had a history of congestive heart failure. One of these patients had circulatory difficulties during induction of anaesthesia and again postoperatively. Another had circulatory difficulties five days after recovery from anaesthesia. These two patients died and will be mentioned in detail below. The other 22 patients had an uneventful course.

Cardiac arrhythmias were almost completely absent during this study. Two patients had auricular fibrillation and two patients had bundle branch block preoperatively. They had a smooth recovery from anaesthesia and surgery. Two

patients developed intermittent extrasystoles and two others had coupled beats during anaesthesia. These disappeared at the end of the operation.

Respiration and Muscle Relaxation

It became evident at the beginning of this study that chloroform was the most potent respiratory depressant of all the volatile agents studied heretofore, and that augmenting muscle relaxation was seldom required after the patient was intubated, and chloroform was started. This held true for most of the patients studied, and indicated two necessary precautions in long operations: muscle relaxants must be given with discretion, especially towards the end of an operation; and the chloroform should be turned off shortly before completion of an operation in order to allow sufficient time for "wash out." Use of the respirator was discontinued before closure of the skin incision was started in order to ensure that the patient would have adequate spontaneous respiration by the time dressings were applied and the tube removed. No difficulty with resumption of spontaneous breathing occurred when this routine was followed.

Post-anaesthetic Morbidity

Recovery from the hypnotic effect of chloroform was not as rapid as was seen after halothane. Most patients were awake when they left the operating room, but often went to sleep again in the recovery room, unless they were stimulated.

Cardiovascular. Three patients had serious cardiovascular difficulties postoperatively. The first was a 71-year-old man with a fractured hip. Besides his primary condition, he had myocardial ischaemia, hypertension, severe emphysema, chronic bronchitis, and diabetes. He was a chronic alcoholic. At the admission examination, he complained mostly of pain in the abdomen (which apparently began just before the hip injury), but this was not taken seriously since abdominal examination did not reveal any localized tenderness or rigidity. After induction of anaesthesia and movement to an orthopaedic table, his blood pressure fell sharply. Positioning and manipulation of the hip were delayed until his condition improved. The anaesthetic and operative course was uneventful thereafter. Postoperatively he had respiratory and circulatory difficulty. Raising secretions was a problem, and he continued to complain of abdominal discomfort. On the second postoperative day his condition rapidly deteriorated in spite of vigorous therapy, and death ensued. At post-mortem, mesenteric thrombosis of recent origin was found, and the lungs showed bilateral bronchopneumonia (Fig. 2).

The second patient was a 57-year-old, very obese male with fever and tachycardia, who had a cholecystectomy from which anaesthetic and surgical recovery appeared to be rapid and smooth. On the third postoperative day he developed severe substernal pain and died suddenly. No post-mortem was obtained, but it was assumed that this death was caused by an acute coronary occlusion or a pulmonary embolus (Fig. 3).

The third patient, a 78-year-old male who had a normal ECG preoperatively, and appeared fit for his age, developed dyspnoea, chest pain, and hypotension while walking in the hospital corridor five days after a cholecystectomy. He was

FIGURE 2

returned to bed, given morphine, digoxin, oxygen, and anticoagulant therapy. ECG some hours later showed evidence of acute myocardial infarction. After three weeks on bed rest, he recovered completely and was discharged from hospital. He has been well since (three months later) (Fig. 4).

Gastrointestinal. Nausea and/or vomiting occurred in 43 patients (28 per cent) in the postoperative period. Most of these responded well to perphenazine. In a few cases these symptoms recurred after the patient was returned to the ward from the recovery room. In eight of these 43 patients, the initial onset of nausea and vomiting occurred several days after the operation, and after food or water was first taken by mouth. Postoperative vomiting that might be attributed to the anaesthetic occurred therefore in 23 per cent of the patients.

Jaundice. Clinical jaundice was present in nine patients during the postoperative period. Five of these were admitted to hospital with jaundice. In four of them, the jaundice subsided after operation. The fifth patient, a female aged 73 years, was investigated for six days, and then had an abdominal exploration, liver biopsy, and common bile duct exploration. A clinical diagnosis of chronic cholangitis, acute pancreatitis, and multiple cysts of the liver, was made. She had an uneventful course during the first five days after the operation, and her jaundice subsided. She then developed acute abdominal distension and vomiting.

FIGURE 3

This was thought to be due to an ileus, or to a subhepatic abscess. Her condition degenerated rapidly, and she died the next day. At post-mortem a bile peritonitis was found, and was considered the primary cause of death.

The other four patients developed jaundice postoperatively. In one very obese male patient, aged 61 years, it appeared two days after a gastrectomy operation of three hours duration. No blood was given during this operation because his preoperative haemoglobin was 16.3 gm. per cent and blood loss was slight. The jaundice gradually increased for four days, and then disappeared by the ninth postoperative day. During the early postoperative period the patient developed tachycardia, and his haemoglobin fell from 15 gm. (immediately after surgery) to 11 gm. a few days later, when it was also observed that he passed a tarry stool. Blood transfusions were given to restore the blood volume. He was discharged from hospital on the fifteenth postoperative day in good condition. It is possible that the chloroform was a cause of his jaundice although jaundice is not an uncommon occurrence after a gastrectomy, especially if marked bleeding continues postoperatively, and if the patient is obese.

The second patient was a male, 59 years of age, who had a history of intermittent jaundice. At a previous operation a few years earlier, attempted chole-

FIGURE 4

cystectomy was abandoned because of technical problems and the gall bladder was drained. He had a prolonged and difficult recovery. In the present instance, cholecystectomy and common bile duct exploration were done during an intricate 4-hour operation. The measured blood loss at this operation was a little in excess of 500 ml. No blood was replaced because his preoperative haemoglobin was 16.2 gm. Later in the recovery room it was felt that he needed blood, and a transfusion was administered. He had a stormy postoperative course again, owing to development of a subhepatic abscess and duodenal fistulae, and several blood transfusions were given as supportive therapy. On the tenth postoperative day he developed slight jaundice. After the abscess was drained and the fistulae were closed at subsequent operations, complete recovery of this patient was slow. The jaundice that developed was undoubtedly due to the surgical complications, and multiple blood transfusions.

The third patient was an obese 60-year-old male with carcinoma of the stomach. He had lost 20 lb. in a short period, and was receiving cortisone for treatment of arthritis. During a 6½-hour operation, a total gastrectomy, splenectomy, and Roux en-Y anastomosis was done. Bleeding was difficult to control during the operation. Measured blood loss was 1,664 ml. and the estimated additional loss

was in excess of 1,000 ml. Blood transfusions were started one hour after the beginning of the operation when it was observed that bleeding was excessive, even though his initial haemoglobin was 16.0 gm. A total of 2,850 ml. of blood was administered during the operation. The patient recovered promptly from the anaesthetic and his vital signs were stable. On the first postoperative day he had difficulty coughing up secretions. His upper airway had to be suctioned frequently to stimulate him to cough and to remove the secretions because he was not co-operative. On the second postoperative day his temperature rose to 101°F., and there were decreased breath sounds in the right chest. An endotracheal tube was passed and tracheobronchial suctioning produced a large amount of sticky mucus. This improved the breath sounds considerably. In spite of vigorous physiotherapy, bronchial breath sounds and rales returned and his temperature spiked to higher levels each day. On the sixth postoperative day his general condition deteriorated. On the seventh day jaundice appeared, and he died the following day. Post-mortem examination revealed that the patient had diffuse carcinoma of stomach and widespread secondary carcinoma. The abdominal cavity had generalized fibrinopurulent peritonitis. There was mucopurulent bronchitis and right bronchopneumonia. The liver and kidney showed moderate and slight fatty degeneration respectively. Generalized arteriosclerosis was also evident. The primary cause of death in this case was fibrinopurulent peritonitis and bronchopneumonia. The jaundice was terminal and probably unrelated to the anaesthetic.

The fourth patient was a 56-year-old female who had a recurrent leiomyosarcoma removed from the transverse mesocolon and a total colectomy during a 3-hour operation. Measured blood loss during the operation was only 238 ml., but on account of her anaemia (preoperative haemoglobin 11.5 gm.) she was given a blood transfusion during the operation. She recovered promptly from the anaesthetic. On the fifth postoperative day she became slightly jaundiced (total bilirubin 4.8 mg. per 100), but felt well. The jaundice disappeared after one week, and she was discharged from hospital in good condition 16 days after her operation. The slight jaundice in this case may have been due to the chloroform anaesthetic and/or to the blood transfusion.

Postoperative Mortality

In addition to the four deaths mentioned above, one other patient who received chloroform anaesthesia died postoperatively. This was a 59-year-old male with myocardial ischaemia (previous history of myocardial infarction), hypertensive heart disease, and asthma, who had an abdominal operation 2 hours in duration. He had carcinoma of the rectum with wide extension that could not be removed, and a palliative left colostomy was done. The patient recovered from anaesthesia promptly, and was apparently well for four days, then developed marked abdominal distension and bronchopneumonia. He died the following day. At post-mortem, widespread infarction of the colon, acute inflammation of the small bowel, and acute bronchopneumonia were found. This death appeared to be due to surgical complications (Fig. 5).

NAME JH		PHYSICAL STA 3	AGE 59	SEX M	HT 71"	WT 159#	ANESTHESIA NO. 4092-59
PROPOSED OPERATION Abdomino-perineal resection of rectum & rt. hemicolectomy							
PRE OP DIAG Carcinoma of rectum & ascending colon							
PRE ANES MED Perphenazine 5mg. + Scopolamine 0.4mg.							
IDENTITY ☒ CONSENT ☒ RESP ☒ MOUTH ☒ TREAT nil PUPILS = R L TIME 6.30 X U NO. 13.7							
POSITIVE FINDINGS Old myocardial infarct., emphysema, asthma, labile BP							
TIME 8am							
N ₂ O 12 liters							
ETHER 0.8%							
CYCLO G 120							
T C E 20							
CHCl ₃ 5% D/W							
MUSCLE RELAX BLOOD							
INTRAV THERAP 6 liters							
ANALG 10							
OXYGEN							
ECG ☒ EEG ☒ MVN 10 liters BLOOD LOSS 5% D/W 200ml. NOT HEAR ☒ J.E.S. ☒ ESTIM TAKAOKA @ 18/min. VENTILATOR BLOOD 500ml. TOTAL FLUIDS							
ABSORPTION ☒ TIME OF REMARK ☒							
INDUCTION		PRIMARY		SECONDARY		TERTIARY	
Thiopent. Gallamine 2.5% i.v. 400mg.		CHCl₃ chlorotoc non rebreathing 0.8% 65%		N₂O		EMERGENCE	
ADJUVANTS 10 NASOTRA		ALINO 1		CLIPP ☒ ROOM 9		TOPICAL ☒ AIRWAY ☒ NONE ☐ OTHER	
SURGEONS L-C Skinner		INSTR Dobkin		OPERATION & DIAGNOSIS Inoperable - palliative left colostomy			

UNIVERSITY HOSPITAL
UNIV. of SASKATCHAWAN
COLLEGE OF MEDICINE

REMARKS
Smooth induction with
Thiopental 400mg.
Gallamine 120mg.
Atropine 0.6mg. (prophylactic)

Uneventful anaesthetic course

FIGURE 5

DISCUSSION

Over ten years ago it was concluded from a detailed re-evaluation of chloroform that this agent does not deserve to be abandoned as a surgical anaesthetic (4, 5). Although only a few reports on the clinical use of chloroform have been published recently (6, 7, 8), the opinion expressed above is supported by several anaesthetists who have continued to use chloroform even after the introduction of halothane—whose advantage over chloroform still appears to be quite nebulous (9).

The bad press which chloroform has had may be said to have started with the report of Hannah Greener's fate (10). In spite of the Hyderabad Commissions (1888 and 1889) this culminated in the opinion expressed by the Committee on Anaesthesia of the American Medical Association, which stated in 1912 that the "use of chloroform for major operations is no longer justifiable . . ." (11). Since then, anaesthetists who have persisted in using chloroform have been on the defensive because postoperative morbidity is readily blamed on this ideal scapegoat. Despite this, its defenders continue to believe that it is a useful anaesthetic agent which has been inordinately maligned (12).

In an erudite address entitled "The Natural History of Error," William Bean opened with the pertinent question: "Who has not wondered at man's marvellous talent for making mistakes and then perpetuating them? The capacity of the human mind to deceive itself knows hardly any limit." Citing numerous errors that have been perpetuated, he reminds us of Milton's words: "Error supports custom, custom countenances error: and these two between them, with the numerous and vulgar train of their followers, envy and cry down industry of free reasoning, under the terms of humour and innovation. Truth never comes into the world, but like the bastard, to the ignominy of him that brought her forth; till Time, the mid-wife, rather than the mother of truth, hath washed and salted the infant, and declared her legitimate" (13).

Several areas of anaesthetic teaching are suffering from the same type of prejudice as the attempts to re-evaluate chloroform—for instance, that of direct artificial respiration (oral resuscitation). The antagonists in both these controversies take the same bigoted line of reasoning: the agent or method was tried many years ago, and was superseded by something new, which is therefore better. With a few refinements in technique, direct artificial respiration is now proving to be an efficient and a widely applicable method of reviving the dying (14). With a little tolerance and some very careful clinical application, undoubtedly chloroform can also again become a useful anaesthetic agent with valid indications, which need not be replaced by modern agents but can be used along with them.

Middleton reported extensive experience with chloroform during World War II, and he feels that adequate atropinization of the patient is an important prerequisite for smooth chloroform anaesthesia (15). Armstrong Davison has observed that maintenance of chloroform anaesthesia is further facilitated if the patient is adequately premedicated with a phenothiazine derivative, in order to produce effective sedation and reduce the concentration of chloroform required for surgical anaesthesia (7). The general principles for maintaining a clear airway and avoiding hypoxia and hypercarbia certainly apply to the use of chloroform anaesthesia, as they would for any other potent anaesthetic agent (16, 17). In addition, much effort has been directed in the past few years to devising means for accurate regulation of the concentration of this agent in the anaesthetic circuit (18, 19).

In comparison with other halogenated anaesthetic vapours, the effect of chloroform on circulatory dynamics and on cardiac rhythm, even in the presence of epinephrine, is not more dangerous than that of the other agents in common and extensive use. Protection against cardiac arrhythmias afforded by a phenothiazine derivative is as reliable with chloroform as with cyclopropane, halothane, the halothane-ether azeotrope, and trichloroethylene. The most important precaution that must be emphasized with all these agents is the maintenance of adequate pulmonary ventilation throughout the time of their administration, and until almost complete washout from the patient's blood stream has been accomplished (20, 21). The reduction in cardiac arrhythmias and circulatory difficulties has been shown with these simple practices alone (22, 23).

In clinical use with identical methods of administration, the hypnotic effect of chloroform was only slightly less potent than Fluothane and slightly more

potent than the Fluothane-ether azeotrope (17, 24, 25, 26). Dogs, on the other hand, are more sensitive to this effect of chloroform (21), whereas Mørch and Jobgen have noticed that there is very little difference in the hypnotic potency of Fluothane and chloroform in small animals (27).

Muscle relaxation was better with chloroform than with either Fluothane or the Fluothane-ether azeotrope. Muscle relaxants did not appear to cause hypotension with chloroform, as was sometimes seen with Fluothane. The circulatory effect of chloroform resembled more the Fluothane-ether azeotrope than Fluothane—that is, chloroform was less likely to cause hypotension than Fluothane. There was a greater tendency to prolonged tachycardia when gallamine was administered for endotracheal intubation followed by chloroform than when followed by Fluothane or Fluothane-ether azeotrope. For this reason, it was considered preferable to use dimethyl-d'tubocurarine with chloroform instead of gallamine, and to use atropine to counteract bradycardia when it occurred.

Just as Goodman Levy's words made the cardiac effects of chloroform a hazard to be feared, so such reports as those of Whipple and Sperry, and, more recently, those of Sheehan have focused attention on the toxic effects of chloroform on the liver (28, 29, 30). Both these effects have been studied unrealistically. With regard to the liver, in most of the animal studies quoted, either no attempt was made to provide the usual supportive therapy during anaesthesia (intravenous fluids and augmented pulmonary ventilation) or no attempt was made to administer a regulated dose of chloroform that would be within the clinically useful range. In some of the studies the animals were starved for two to three days before an experiment, in order to augment the deleterious effects of chloroform on the liver. Such procedures would show diethyl ether and halothane in a bad light also. The dictum "chloroform poisoning is synonymous with chloroform anaesthesia" is logical only in the same sense as asphyxia and starvation may be synonymous with death (31, 32). The standards recommended presently for evaluating the effect of chloroform or any other halogenated anaesthetic agent on the liver of animals are of limited clinical application unless appropriate steps are taken to assure proper nutrition and to augment pulmonary ventilation—for these are essential prerequisites in the safe administration of any potent anaesthetic to the depth that is required for prolonged surgical anaesthesia (33, 34, 35).

Bunker has made the apt clinical observation that there are no differences in the effect on the liver that can be attributed to an anaesthetic agent or technique. Rather, the operative procedure and the degree of preoperative liver damage have the more marked effects on both the postoperative disturbances in liver function tests and the clinical result. As far as the liver is concerned, the choice of anaesthetic agent is of less importance than careful preoperative preparation, adequate oxygenation during the operation and postoperatively, and the use of fresh blood transfusions (36).

The administration of transfusions of stored bank blood is certainly to be implicated in a variety of metabolic and blood-clotting defects which harass both anaesthetist and surgeon during a major operation and during the postoperative recovery period. It is well known that disturbances such as jaundice

and uncontrollable capillary bleeding are caused by bank blood transfusions, especially when used for intrathoracic operations and those involving the portal circulation (37, 38, 39). This effect of administering stored blood undoubtedly occurs to a varying degree in most operations.

One further form of postoperative disease is beyond the control of the anaesthetist, and is often related to anaesthetic morbidity rather than to inherent metabolic derangements. An ever-growing proportion of major operations are performed on patients who are grossly obese. In the present study, 38 per cent of patients were definitely overweight, according to life insurance standards (40, 41, 42). A wide range of serious problems arise insidiously when anaesthesia is administered to an obese patient (43). Not the least of these is aggravation of the condition of the liver. Zelman has shown that the apparently healthy obese person has anatomical damage of the liver that is similar to that of the chronic alcoholic. Many have impaired glucose tolerance, and bromsulphalein retention is abnormal in all such individuals even when they are "well" (44). Any clinical reports on the effect of supposedly hepatotoxic anaesthetic agents must be analysed, therefore, in the proper perspective whenever the patient is obese.

It is interesting to note some of the morbidity and mortality reports which are now appearing, that relate toxic changes in the liver to halothane. Barton gives no indication of other possible causes of the jaundice in his patients who received halothane (45, 46). Virtue and Paine, in a death report of a patient who had received halothane, imply that the acute yellow atrophy of the liver found at post-mortem could have been due to delayed halothane poisoning (47). These two reports contain some faulty reasoning as was noted above. The latter death was in an obese patient with an obvious history of liver disease. She had several episodes of severe hypotension during anaesthesia, which could have markedly reduced liver blood flow to a level that would cause acute prolonged hepatic anoxia. These factors alone might account for the post-mortem changes observed in the liver; so why imply that halothane alone was the culprit, in the way that chloroform was blamed in the past? With a little detective work and some logical reasoning, many factors could explain the liver damage without specifically implicating the particular anaesthetic agent that happened to be administered.

In carrying out the present study, every precaution was taken that was known to facilitate management of the anaesthetic. Nevertheless, there were many factors—the general condition of the patient, the skill and judgment of the surgeon, the availability of fresh blood, the care and attention of the anaesthetist, and the will to live by the patient, especially when he knew that he was suffering from a very serious illness—which were not all in the hands of the anaesthetist, and were mainly beyond his control. Even so, the morbidity in this group of 154 patients was not significantly different from that of other patients who were undergoing similar operations by the same surgeons, but with other anaesthetic agents.

The general surgical mortality during this study, in all major and minor procedures, except neurosurgical, paediatric, and cardiovascular operations, was 3 per cent (56 deaths in 1,839 patients). The mortality rate among patients who

received chloroform anaesthesia for major operations only was approximately the same (5 deaths in 154, or 3.2 per cent).

The postoperative mortality among the patients who underwent cholecystectomy, gastrectomy and bowel resection, and related operations during this study was reviewed in detail, and is shown in Table II. The mortality rate was 5.2 per cent for the chloroform group, and 15 per cent for the other group. This difference was too striking to have been due to chance alone; yet, in none of the cases that did not receive chloroform, did the surgeon question the anaesthetic selected, whereas the use of chloroform was criticized, in each of the deaths described. However, in none of these (with or without chloroform) was the anaesthetic agent finally considered as the primary or major contributing cause of death.

TABLE II
POSTOPERATIVE MORTALITY
(JUNE, 1959, TO FEBRUARY, 1960)

Operation	No. of patients	Anaesthetic		Deaths	
		Chloroform	Other	Chloroform	Other
Gastrectomy and related procedures	65	27	38	1	4
Cholecystectomy and related procedures	72	29	43	2	5
Bowel resection and related procedures	56	22	34	1	7
TOTALS	193	78	115	4	16

On the basis of the cases reported, this study supports the opinion that chloroform is a potent agent which must be vaporized in a precision system, and when administered with care and attention to the necessary prophylactic and therapeutic measures, it provides satisfactory operating conditions without increasing postoperative morbidity or mortality.

SUMMARY AND CONCLUSIONS

Chloroform with nitrous oxide and oxygen was administered to 154 patients during major operations, employing a precision non-rebreathing system with a calibrated vaporizer. Artificial respiration was provided with a pressure regulated ventilator designed by Takaoka, and pulmonary ventilation was set to the requirements of the individual patient. Smooth maintenance of anaesthesia was accomplished in this system with 0.5 to 1 per cent chloroform, 65 per cent nitrous oxide, and small supplements of muscle relaxant drugs. This method sufficed to produce satisfactory operating conditions without inducing cardiovascular depression. Dimethyl d'tubocurarine was preferred to gallamine as the muscle relaxant, because the latter sometimes caused prolonged tachycardia.

The only alteration in the vital signs that was frequently observed was a slowing of the pulse rate. This was effectively treated with atropine. A moderate reduction in the estimated stroke volume and cardiac output was also observed, but this was of the same degree as was seen with the Fluothane-diethyl ether azeotrope. Cardiac arrhythmias in these cases were significant by their absence even in those patients with serious cardiovascular disease.

Postoperative recovery from anaesthesia was not significantly slower than that observed after Fluothane. The incidence of postoperative nausea and vomiting was somewhat higher than average, but was similar to that seen with other anaesthetics for the particular operations in which it was used. Postoperatively serious cardiovascular depression attributable to the anaesthetic agent alone was not seen. The incidence of postoperative jaundice was regarded prominently because it was expected to occur more often after chloroform than after other anaesthetics, but this was not evident when the data of this study were reviewed. Surgical mortality was not higher after chloroform anaesthesia than after other anaesthetics for similar operations by the same surgeons.

This study shows that chloroform has, in fact, been inordinately maligned as an anaesthetic for major operations. When used with the same care and precision as are expected for other potent agents, it undoubtedly has a place in clinical practice along with the other halogenated anaesthetic vapours.

RÉSUMÉ

Au cours d'opération majeure, nous avons administré à 154 malades du chloroforme avec du protoxide d'azote et de l'oxygène; nous avons employé un système sans ré-inspiration et un vaporisateur calibré. A l'aide d'un ventilateur à pression réglable, conçu par Takaoka, nous avons pratiqué une respiration artificielle chez ces malades et le volume de la ventilation pulmonaire correspondait aux exigences préétablies de chacun de ces malades. Grâce à ce système, l'anesthésie a été doucement maintenue avec 0.5 à 1% de vapeurs de chloroforme, 65% de protoxide d'azote et de petites doses de myorésolutifs pour compléter. Cela s'est avéré suffisant pour fournir des conditions opératoires satisfaisantes sans provoquer de dépression cardiovasculaire. Comme agent myorésolutif, nous avons préféré la d'tubocurarine à la gallamine, parce que quelquefois cette dernière peut entraîner des tachycardies prolongées.

La seule modification des signes vitaux que nous avons observée a été un ralentissement du pouls. Cette bradycardie a été efficacement corrigée par de l'atropine. Nous avons également observé une légère diminution de l'ondée systolique et du débit cardiaque mais nous avons observé la même chose avec le mélange Fluothane-éther azeotrope. Nous avons été étonnés de n'avoir observé d'arythmie cardiaque chez ces malades même chez des porteurs de maladies cardiovasculaires.

Après l'opération, le réveil n'a pas été plus tardif qu'il n'avait été avec le Fluothane. La fréquence des nausées et des vomissements postopératoires a été un peu plus grande que la moyenne, mais elle différait peu de celle observée avec d'autres agents anesthésiques pour des opérations semblables à celles où il avait été employé. Nous n'avons pas observé de dépression cardiovasculaire sérieuse postopératoire attribuable exclusivement à l'agent anesthésique. Nous avons porté une attention spéciale à la jaunisse postopératoire parce que nous nous attendions d'en observer plus souvent qu'avec l'usage des autres agents anesthésiques mais, au moment de colliger les observations de cette étude, on ne nous a pas signalé cette complication. La mortalité opératoire après l'anes-

thésie au chloroforme n'a pas été plus élevée qu'avec les autres agents anesthésiques pour les mêmes opérations pratiquées par les mêmes chirurgiens.

Cette étude démontre que, en fait, le chloroforme s'est avéré particulièrement mauvais comme agent anesthésique pour des opérations majeures. Mais s'il est employé avec le même soin et la même précision qui doivent présider à l'usage d'agents puissants, il lui revient sans doute une place en clinique comme les autres vapeurs anesthésiques halogénées d'ailleurs.

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