THE EFFECT OF ANAESTHESIA ON THE INTRAOCULAR VOLUME OF THE C_3F_8 GAS BUBBLE

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SUMMARY

Long-acting intraocular gas bubbles are frequently used during vitrectomy to tamponade retinal breaks. The aim of this study was to determine the effect of nitrous oxide anaesthesia on the size and effectiveness of the post-vitrectomy gas bubble. Twenty vitrectomy procedures with injection of 12% perfluoropropane (C_3F_8) gas were performed. For 10 of the cases routine anaesthesia with nitrous oxide was used and for 10 cases non-nitrous anaesthesia with propofol was used. The volume of the intraocular gas bubble was estimated 24 hours post-operatively using A-scan biometry. At 24 hours the gas bubble occupied a mean of 65.1% of the eye in anaesthesia with nitrous oxide and a mean of 66.1% in anaesthesia with intravenous propofol. The wide range of values of gas-fill recorded at 24 hours makes comparison of the two groups inappropriate. Several factors may account for this spread of values, but in our opinion it is the uncontrolled leakage from the sclerostomies which is the most likely. This study suggests that anaesthesia using nitrous oxide does not adversely affect the size of the C_3F_8 gas bubble at 24 hours post-vitrectomy when compared with anaesthesia without nitrous oxide.

Long-acting intraocular gas bubbles are frequently used during vitrectomy to tamponade retinal breaks until a chorioretinal adhesion has formed.[1] The kinetics of gases within the vitreous cavity have previously been studied, including their interaction with nitrous oxide anaesthesia.[1–4] The use of nitrous oxide anaesthesia produces an increase in the size of the intraocular gas bubble, with a subsequent rise in intraocular pressure. In addition the nitrous oxide displaces other gaseous constituents from the bubble including the tamponading gas, e.g. perfluoropropane (C_3F_8). Following cessation of anaesthesia the nitrous oxide leaves the eye quickly.[5] The consequence of this is hypotony in the immediate post-operative period and a reduction in the size of the gas bubble. This has led to the practice of discontinuing nitrous oxide at least 15 minutes before gas–fluid exchange in vitreous surgery to allow clearance of nitrous oxide from the anaesthetic system as well as the body. This may prevent the possibility of a smaller, less effective gas bubble and an uncompensated drop in intraocular pressure. It has, however, never been clinically determined whether this practice is of any value. Mostafa *et al.*[6] demonstrated that if continuous 65% nitrous oxide in oxygen anaesthesia was used for 20 minutes after gas–fluid exchange, the percentage nitrous oxide in the gas bubble varied between 4% and 21% (mean 9%), and they concluded that the nitrous oxide gas should be turned off during vitrectomy to reduce displacement of the tamponade gas and to avoid hypotony.

Our aim was to investigate the effect of nitrous versus non-nitrous anaesthesia on the post-operative C_3F_8 gas bubble and on the intraocular pressure following gas–fluid exchange. Anaesthesia maintained by the induction agent propofol, without nitrous oxide, is employed routinely by one of the anaesthetists in our unit. This gave us the opportunity to compare the post-operative gas bubble size and post-operative intraocular pressure in patients operated upon using nitrous oxide and in those in whom non-nitrous anaesthesia was used.

PATIENTS AND METHODS

Approval was obtained from the hospital ethics committee and written informed consent obtained from 19 ASA class I–III patients[7] undergoing vitrectomy with injection of C_3F_8 gas recruited to the study. Ten vitrectomies were performed in 10 patients (group 1) using nitrous anaesthesia administered by J.K. Ten vitrectomies were performed in 9 patients (group 2; one patient had vitrectomy of each eye on separate occasions) using non-nitrous

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Table I. Details of 20 eyes studied

Patient no.	Age (years)	Sex	Phakic status	Disease	Anaesthetic technique	Surgeon
1	33	M	Aphakic	RRD	Nitrous	D.W.
2	66	M	Pseudophakic	RRD	Nitrous	D.W.
3	77	M	Pseudophakic	RRD	Nitrous	D.W.
4	42	M	Aphakic	RRD	Nitrous	D.W.
5	80	F	Phakic	Macular hole	Nitrous	D.W.
6	22	F	Phakic	RRD	Nitrous	D.W.
7	28	F	Phakic	RRD	Nitrous	D.W.
8	57	F	Aphakic	RRD	Nitrous	D.W.
9	54	M	Phakic	Trauma	Nitrous	D.W.
10	62	M	Phakic	RRD	Nitrous	D.W.
11	64	F	Phakic	RRD	Non-nitrous	J.McG
12	74	F	Phakic	RRD	Non-nitrous	D.W.
13	31	M	Phakic	RRD	Non-nitrous	D.W.
14L	45	F	Phakic	RRD	Non-nitrous	J.McG
14R	45	F	Phakic	RRD	Non-nitrous	J.McG
15	70	M	Phakic	RRD	Non-nitrous	J.McG.
16	61	F	Phakic	RRD	Non-nitrous	D.W.
17	79	F	Aphakic	RRD	Non-nitrous	D.W.
18	70	M	Phakic	RRD	Non-nitrous	J.McG
19	72	M	Phakic	RRD	Non-nitrous	J.McG

RRD, rhegmatogenous retinal detachment.

anaesthesia. Table I shows the patient details, indications for vitrectomy and details of the procedure for each of the 20 vitrectomies, including the surgeon: D.W. or J.Mc.G.

Pre-operative evaluation consisted of measurement of axial length by A-scan (Echorule Ultrasonic Biometer, Vision Care-3M), measurement of intraocular pressure by Tonopen (Mentor) and estimation of the number of quadrants of retinal detachment. A standard three-port 20 gauge vitrectomy was performed in all patients. At the end of the procedure fluid–air exchange was performed, removing all preretinal fluid and subretinal fluid via retinotomy. The air-filled eyes were then flushed through with 50 ml of 12% C_3F_8 (a non-expansile concentration of the gas[8]). This was prepared immediately prior to use as follows: 6 ml of neat gas was drawn up into a 50 ml syringe followed by 44 ml of filtered air. Care was taken to ensure that the system was well flushed through with C_3F_8 to remove air from the dead space in the plastic tubing. Sclerotomies were closed with 8-0 Vicryl. Explants were used where clinically indicated (see Table I).

In the patients in group 1 anaesthesia was induced with intravenous thiopentone and neuromuscular blockage with atracurium. Anaesthesia was maintained by 65% nitrous oxide in oxygen and 1–2% enflurane with supplements of fentanyl and droperidol as required. Ventilation was controlled to end-tidal carbon dioxide concentration at 4–4.5%. Residual neuromuscular blockage was reversed with glycopyrrolate and neostigmine. The nitrous oxide was not discontinued until the end of the procedure.

For the patients in group 2 anaesthesia was induced with intravenous propofol and neuromuscular blockage with atracurium. Anaesthesia was maintained by 35% oxygen in air, propofol infusion (3–8 mg/kg per hour) and 0.5–1% enflurane with supplements of fentanyl and droperidol as required. Ventilation was controlled to end-tidal carbon dioxide concentration at 4–4.5%. Residual neuromuscular blockage was reversed with glycopyrrolate and neostigmine.

Post-operative evaluation included measurement of intraocular pressure by Tonopen 20 minutes and 24 hours following the procedure. Estimation of the

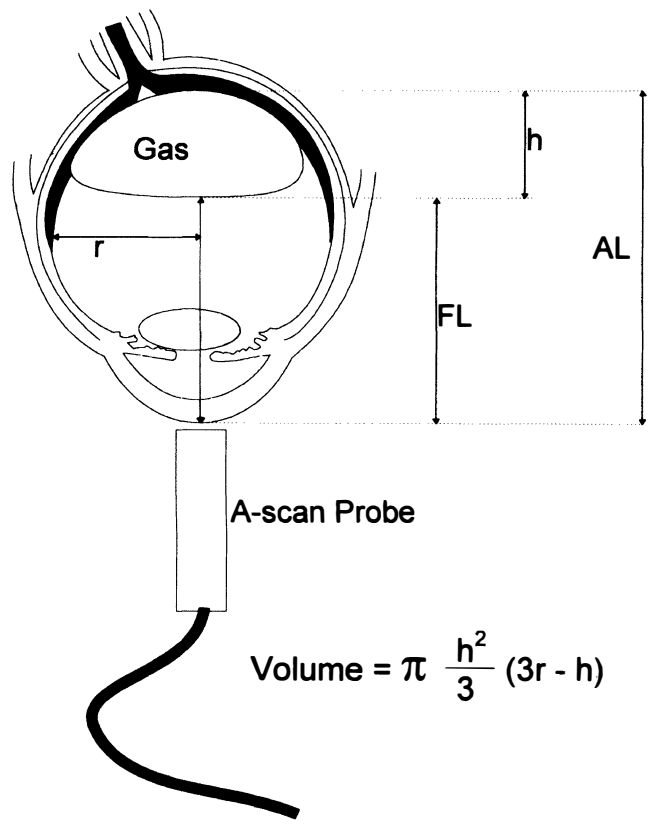

Fig. 1. *Diagram showing measurement of the distance from cornea to gas–fluid interface and the formula for calculation of the volume of the gas bubble.*

size of the intraocular gas bubble at 24 hours used the method described by Jacobs.[9] The patient was positioned with the optical axis perpendicular to the floor and the A-scan probe placed on the cornea (Fig. 1). The gas–fluid interface then lay perpendicular to the A-scan probe and the ultrasound beam was reflected from this interface giving a peak (Fig. 2) which represented the distance from anterior cornea to the gas–fluid interface (FL). A 'ringing echo' was usually demonstrated, indicating accurate alignment. Three such measurements were taken at 24 hours post-operatively. As scleral buckling may alter the dimensions of the globe, measurement of axial length was repeated after the gas bubble had disappeared and this value was used in conjunction with the mean of the three FL measurements to calculate a value for the bubble height (h) (Fig. 1). An estimate of the internal radius (r) was then used in conjunction with h to calculate the absolute volume of the gas bubble in each eye (see Jacobs[9] for a full description of the method).

For the purposes of this study we wished to compare the size of the bubble in different patients. The absolute volumes of gas for each eye are not comparable as the eyes are of different volumes. A value for the percentage of the eye occupied by gas was therefore calculated using the volume of the gas bubble as shown above and the total volume of the eye. The volume of the eye was calculated with the same formula but using the axial length alone (measured post-operatively) in place of the difference between FL and the axial length. Scleral buckling is a source of error for both the volume of the gas bubble and the total ocular volume. We compensated for this by subtracting an error factor from each of the two volumes as follows: For a radial or circumferential sponge we subtracted 0.15 ml, for a 40 band 0.5 ml and for a 40 band combined with a 287 tyre 1.5 ml.[10]

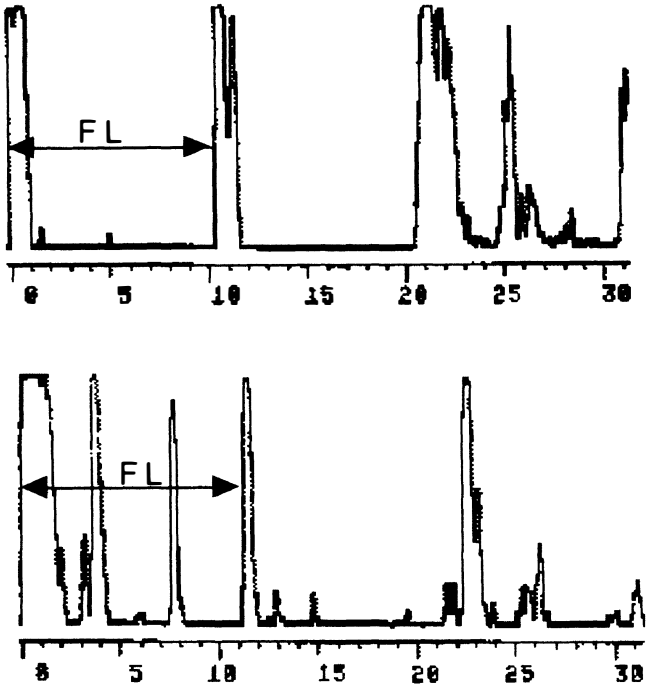

Fig. 2. *Two examples of A-scan ultrasonography showing the spike indicating the position of the gas–fluid interface, the first (above) in a phakic and the second (below) in an aphakic patient.*

Table II. Intraocular pressures

		Post-operative IOP	
Patient no.	Pre-operative IOP	20 minutes	24 hours
1	15	33	23
2	8	40	16
3	14	11	17
4	17	19	15
5	18	34	10
6	19	*a*	16
7	18	8	12
8	17	5	27
9	21	23	27
10	8	0	8
11	7	19	35
12	12	20	10
13	16	27	23
14L	14	37	27
14R	18	10	20
15	11	10	21
16	13	24	31
17	17	48	40
18	14	25	30
19	19	21	18

[a]Patient refused IOP measurement.

RESULTS

Pre-operative intraocular pressures in group 1 ranged from 8 to 21 mmHg (mean 16 mmHg, $n = 10$) and in group 2 from 7 to 19 mmHg (mean 14 mmHg, $n = 10$). There was no significant difference between groups in the intraocular pressures measured at 20 minutes post-operatively ($p = 0.21$, Student's unpaired t-test): in group 1 values ranged from 0 to 40 mmHg (mean 19 mmHg, $n = 9$ – one patient would not tolerate intraocular pressure measurement) and in group 2 corresponding values were from 10 to 48 mmHg (mean 24 mmHg, $n = 10$). At 24 hours post-operatively intraocular pressures were higher in group 2, with a p value of 0.014 (Student's unpaired t-test): values in group 1 ranged from 8 to 27 mmHg (mean 17 mmHg, $n = 10$) and in group 2 from 10 to 40 mmHg (mean 26 mmHg, $n = 10$). All intraocular pressures are shown in Table II and post-operative intraocular pressures in Fig. 3.

All eyes had a flat retina at 24 hours. Table III shows the details of the surgical and anaesthetic technique used for each case together with the volume of the gas bubble, the volume of the globe and the percentage gas fill for each eye calculated using the method described above. Percentage gas fill of eyes in group 1 ranged from 45.4% to 71.2% (mean 65.1%, SD 10.0). Gas fill in eyes in group 2 ranged from 43.4% to 76.2% (mean 66.1%, SD 9.0).

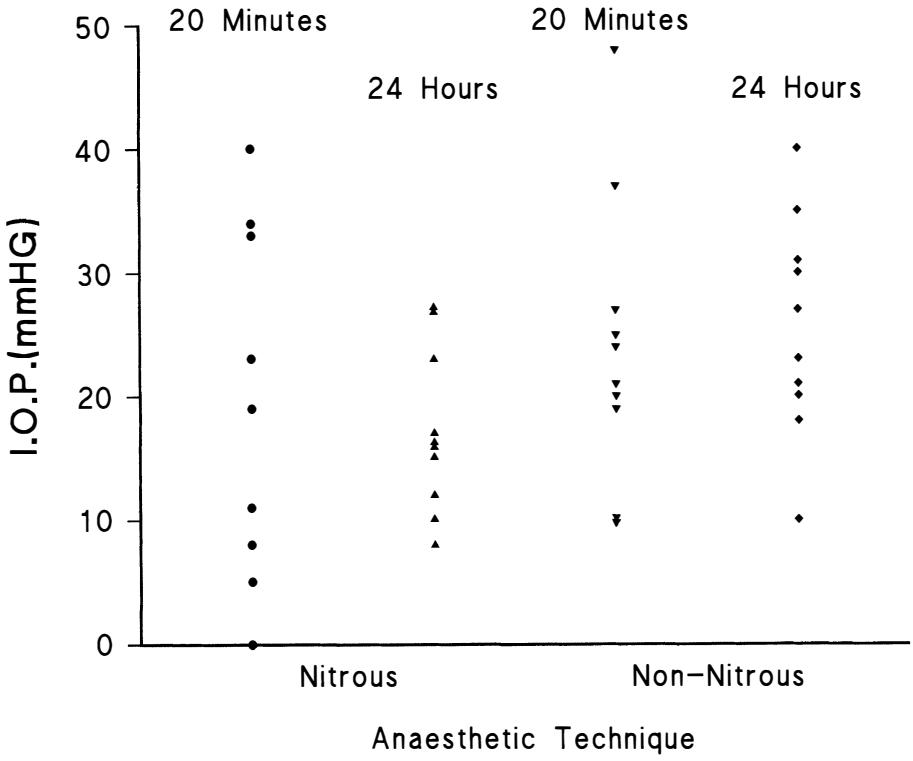

Fig. 3. *Scattergram of post-operative intraocular pressures.*

These results are illustrated in Fig. 4. There was no significant difference in the mean gas fill for the two groups ($p = 0.41$, Student's unpaired t-test).

DISCUSSION

The importance of gas dynamics in the post-vitrectomy eye relates to the very high diffusion coefficient of nitrous oxide as well as its high solubility coefficient of 0.4689. This causes nitrous oxide to diffuse down its concentration gradient much more quickly than the other gases involved – namely nitrogen, oxygen, carbon dioxide and C_3F_8. The net effect of these movements is an expansion of the gas bubble. In a closed eye this leads to a rise in the intraocular pressure; however, in the post-vitrectomy eye the sclerostomies leak, preventing catastrophic rises in the intraocular pressure. Leakage from the sclerostomies results in loss of all the constituents of the gas bubble including C_3F_8. Since it is the insoluble C_3F_8 molecules which cause the extended survival of the intraocular bubble, then any loss of this 'active' constituent of the bubble will

Table III. Plombage, gas bubble volume, globe volume and percentage gas-fill for each eye studied

Patient no.	Buckle	Anaesthetic technique	Gas volume (ml) at 24 hours	Globe volume (ml)	Gas-fill (%) (corrected)
1	Radial plomb	Nitrous	7.38	10.37	71.2
2	40 band + 287	Nitrous	3.74	5.5	68.0
3	Radial plomb/40 band	Nitrous	3.76	6.25	68.4
4	40 band + 287	Nitrous	2.56	5.64	45.4
5	None	Nitrous	3.98	5.59	71.4
6	None	Nitrous	4.11	5.60	73.5
7	None	Nitrous	3.08	6.00	51.3
8	40 band + 287	Nitrous	4.22	5.5	76.7
9	40 band + 287	Nitrous	3.66	6.12	59.8
10	Radial plomb/40 band	Nitrous	6.24	9.55	65.3
11	40 band + 287	Non-nitrous	8.70	12.17	71.5
12	40 band	Non-nitrous	3.03	4.36	69.5
13	None	Non-nitrous	4.63	7.36	62.9
14L	Circumferential plomb	Non-nitrous	4.38	6.26	70.0
14R	None	Non-nitrous	4.38	6.55	67.0
15	40 band + 287	Non-nitrous	4.76	6.91	68.9
16	40 band + 287	Non-nitrous	1.97	4.54	43.4
17	40 band	Non-nitrous	4.09	5.86	69.8
18	None	Non-nitrous	8.54	13.85	61.7
19	None	Non-nitrous	4.32	5.67	76.2

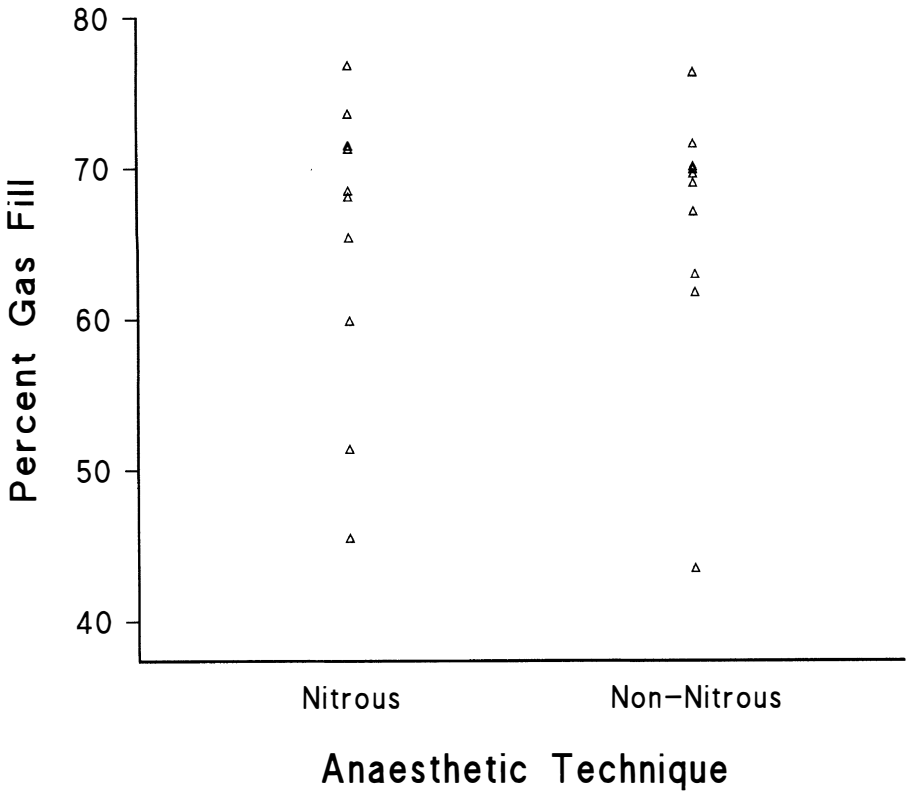

Fig. 4. *Scattergram of percentage gas-fill for the two groups of patients.*

reduce the size of the bubble post-operatively and ultimately the duration of the tamponade.

We used 12% C_3F_8 as this is the maximum non-expansile concentration.[11] In group 1, who received nitrous oxide anaesthesia, we would expect a rise in intraocular pressure as nitrous oxide passed into the eye followed by a fall as it left the eye at the end of the operation. No such pattern would be expected in the case of group 2 where no nitrous oxide was used. Our results showed a wide range of values in both groups, with an intraocular pressure at 20 minutes post-operatively of greater than 21 mmHg in 4 of 9 cases measured in group 1 and in 4 of 10 cases in group 2. There was a wide range of intraocular pressures recorded at 24 hours, with higher values in the non-nitrous group, although this was of relatively low significance ($p = 0.014$). We also noted a surprisingly low percentage of gas-fill in both groups 1 and 2 (mean gas bubble sizes of 65.1% and 66.09% respectively), with a wide range of values in both groups (SD of 10.0 and 9.0 respectively).

Other factors may, however, play a significant role in determining the size of the gas bubble. Variations in the concentration of the constituent gases may be a result of poor technique.[12] Clinical factors which may influence the gas bubble include lens status, previous vitrectomy, presence of peri-retinal membranes, retinectomy, scleral buckle, aqueous production, aqueous outflow, size of retinal breaks, extent of retinal detachment pre-operatively and extent of retinopexy. Some or all of these may account for both the underfill and the wide variation of values achieved. We acknowledge that none of these factors was controlled in our study. In addition the procedures were shared between two different surgeons.

The most striking observation was that there was no significant difference between the size of the gas bubbles in group 1 and group 2. This is an unexpected result from the gas theory discussed above. One clinical aspect that has not been emphasised previously is the leaking of gases from the sclerostomies when the posterior segment is totally gas-filled as is the case after gas–fluid exchange. Any rise in intraocular pressure cannot be compensated totally by an increase in outflow of aqueous.[13] Where the sclerostomy is incompetent it will result in leakage as shown by Mostafa *et al.*[6] We have observed a rise in intraocular pressure post-operatively in the non-nitrous group in 50% of cases. The low percentage gas-fill at 24 hours may simply be due to leakage of gas in the early post-operative phase. In the nitrous oxide group, where one would expect a fall in intraocular pressure, we have observed a high intraocular pressure in 44% of cases. In both groups the number of patients with a pressure rise may have been underestimated due to leakage normalising the intraocular pressure in the early post-operative phase. The amount of gas leak was totally unpredictable and in our view accounted

for the absence of an observable difference in gas-fill between the two groups. Although gas dynamics theoretically indicate that non-nitrous anaesthesia should be used, the kinetics only apply in the closed eye situation. In practice, with a three-port pars plana vitrectomy via 20 gauge sclerostomies, leakage as a result of high intraocular pressure is difficult to control. From the results of this study it would appear that this uncontrolled leakage is not significantly influenced by the use of nitrous oxide anaesthesia. We propose that it is the leakage from the sclerostomies which results in the underfill consistently noted at 24 hours post-operatively in both groups. To maximise the post-operative gas bubble size and therefore the duration of the tamponade, we may have to be content with gas top-up through repeated neat gas injections via the pars plana in phakic patients or through anterior segment fluid–gas exchange in aphakic patients.

Key words: Vitrectomy, Tamponade, C_3F_8, Intraocular gas, Anaesthesia, Nitrous oxide.

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