

Possible Prevention and Eradication of Cerebrospinal Fluid Shunt Infection with Povidone Iodine in Vitro

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Key words C.S.F. shunt infection – Povidone iodine

Since the first recognition of colonisation of cerebrospinal fluid (C.S.F.) shunts in 1961 (3), many attempts have been made in clinical studies to prevent this complication. These techniques have been reviewed by *Welch* (8) and *Bayston* (1). In 1982, *Forrest* described a technique using topical gentamicin which reduced the incidence to 1% (6). However, once infection is established exteriorisation or removal of the system has to be employed in most cases (4, 5). Few reports exist of apparent successful treatment with antibiotics alone (2, 4). A recently described reliable *in vitro* technique for colonising C.S.F. shunts (7) has allowed us to study the place of povidone iodine (P.V.I.) in prevention and eradication of infection. Our preliminary results are reported.

Material and Method

Prevention

Spitz Holter and *Holter Hausner* valves were primed with 1% (two valves) and 10% (four valves) P.V.I. twice and then filled with simulated C.S.F. containing 10^3 to 10^4 organisms per ml. The organism used was a *Staphylococcus epidermidis* isolated from a clinically infected shunt system. The valves were then incubated at 37°C in a CO₂ enriched atmosphere for 48 hours. Samples of the valve contents were taken at 24 and 48 hours for organism counts. At the same time four other valves were colonised in a like manner but without preliminary flushing with P.V.I.

Eradication

A series of 9 *Spitz Holter* and 5 *Hakim* valves were colonised (7) using a simulated C.S.F. containing approximately 10^4 orgs/ml. The valves were incubated for 3 hours at 37°C in a CO₂ enriched atmosphere before being perfused with sterile simulated C.S.F. at 5 ml/h for 48 hours. Confirmation of colonisation was obtained by culturing the perfusate at this point in time, though the results were not awaited before continuing with the experiments. Povidone iodine 10% was then injected through a 'T' piece between 5 and 10 cms proximal to the valve, while the tubing above this level was clamped, except for the *Hakim* valves. Two of the latter valves were injected with P.V.I. into the antechamber, and three via a T piece above the antechamber. The schedules for injecting P.V.I. were as follows:

1. 3 ml over 3 minutes – 1 valve
2. 10 ml over 10 minutes – 2 valves

3. 10 ml over 10 minutes, repeated at 24 hours – 2 valves
4. 10 ml over 10 minutes, repeated at 24 and 48 hours – 4 valves
5. as 4. but with 2 *Hakim* valves injected into antechamber
6. as 5. but with injection into T piece above antechamber

Cultures were taken from the perfusate, by allowing it to drip into a sterile container from the distal catheter, until growth of the organism was obtained or sterility was demonstrated at 96 hours.

Results

Prophylaxis

In all six valves treated initially with P.V.I. no organisms were recovered at 48 hours (Table I). In the controls, the organisms multiplied to 2×10^7 at 24 hours and 5×10^7 at 48 hours.

Eradication

All valves injected with P.V.I. on one or two occasions only were found to have organisms present between 24 and 96 hours after the first injection (Table II). In 3 valves of the four treated on three occa-

Table I PVI and prevention of infection

No. of valves	PVI soln	Colony count		
		0 hrs	24 hrs	48 hrs
2	1 %	6×10^3	0	0
4	10 %	6×10^3	0	0
4	Controls	6×10^3	2×10^7	5×10^7

Table II Colonisation of valves after injection of P. V. I. (10 %)

Time (hrs)	0	12	24	48	72	96
1.	+	0	0	+		
2.	+	0	+	+		
	+	0	0	+	+	+
3.	+	+	0*	0	0	+
	+	0	0*	+		
4.	+	0	0*	0*	0	0
	+	0	0*	0*	0	0
N.B.	+	0	+	0*	+	
	+	0	0*	0*	0	0
5.	+		+	+	+	
	+		0*	0*	0	+
6.	+		0*	0*	0	0
	+		0*	0*	0	0
	+		0*	0*	0	0

+ organisms isolated
N.B. Valve found to leak
0 no organisms isolated
* Time of injecting PVI

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sions with P. V.I., no organisms could be cultured at 96 hours. The fourth valve remained colonised but on inspection was found to be partly disrupted and leaking. Organisms were present both within and on the outside of the system.

When the *Hakim* valves were injected into the antechamber three times with P.V.I., no success was achieved (Table II). Three further valves were then treated in the like manner but the injection of P.V.I. made through a T piece above the chamber. In all 3 valves, eradication of the organisms was achieved.

Discussion

The results of preventing shunt infection *in vitro* by initial priming of the system with P.V.I. are encouraging. At present, we do not know how long the protection lasts against colonisation, nor do we know whether the same results will appertain to the clinical situation. Before this method can be tried in patients, the effects of small amounts of intrathecal and intravenous P.V.I. will have to be studied.

These studies suggest serial injections of P.V.I. may eradicate shunt infection. It is likely that when the organisms adhere to the silastic they may be able to survive more effectively, hence the need for repeated injections. Our results indicate that the method will only be applicable to systems where the infection can be placed above the level of infection i.e. distal catheter and some valve colonisations. However, it would be logical to employ a systemic antibiotic to eradicate organisms in the blood or peritoneum, depending on the position of the shunt. Physical considerations may also be important in the clinical situation, since it was noticed during this work that the position of the valve affected the rate of emptying of the P.V.I. in spite of continued perfusion. Longer contact of P.V.I. and bacteria may be achieved if the patient lies supine. Until more is known about intra-ventricular instillation of P.V.I., proximal occlusion of the system will be essential.

Throughout these experiments we have presumed that where organisms were retrieved from the perfusate they had established adherence to the silastic. This was based on results obtained in a previous study (7).

Results from the present experiments encourage us to pursue the possible role of P.V.I. in prophylaxis and eradication of infection in C.S.F. shunts. Using the *in vitro* model, further studies are now possible of other antiseptics, injected antibiotics, and antibiotics impregnated into the silastic.

Summary

The results of preliminary *in vitro* studies of the efficacy of povidone iodine (P.V.I.) in preventing and eradicating infection in cerebrospinal fluid shunts are reported. After preliminary flushing of the system with P.V.I., it was not found possible to colonise valves. To eradicate infection from a previously colonised shunt it was found necessary to inject P.V.I. three times at 24 hourly intervals at a point above the level of colonisation.

Acknowledgements

The authors are grateful to the following organisations for assisting in the funding of this work: ASBAH, The Richard Fund and The Chris Fund.

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