epidemic if they experience some increase in per caput income which, in turn, will be greatly influenced by slowing of the rate of population growth.[7]

Conclusions

Rapid population growth is no longer a problem looking for a solution; it is a solution looking for resources. Nearly all the work to be done in the 1990s will depend on contraceptive methods and channels of distribution that are already in use and well understood.

Sir Dugald Baird's achievement was apparent in the Scottish city of Aberdeen, where he showed that when family planning services were improved and access to surgical contraception and abortion were added, people of all social and economic groups both wanted and could achieve similar family size. Baird's genius was to extrapolate this model to a world level; the past two decades have shown that access to realistic family planning services can also help close the gap in fertility between rich and poor countries. 50–80% of women in developing countries now want to space or limit future childbearing.[8]

The world has a choice. People want to restrict family size and, by making realistic and accessible family planning services universally available, we can probably achieve a stable global population of about twice our present numbers. If we achieve Baird's vision and try to eliminate unintended pregnancies, global population could stabilise at under 8 billion (fig 2). To do this we would have to include virtually universal access to safe, cheap abortion as well as to contraception and VSC. If we continue to give family planning low priority, global population could drift upwards towards 15 billion before it stabilises. The health and progress of hundreds of millions of people will be influenced by how we respond to this choice. How we answer the challenge of global family planning will also help decide whether we achieve an ecologically sustainable global economy. That, in turn, may well determine the future of our species on this planet.

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CLINICAL PRACTICE

Acute hypervolaemic haemodilution to avoid blood transfusion during major surgery

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16 patients underwent acute hypervolaemic haemodilution with dextran 40 and Ringers lactate, to see whether this procedure could avoid preoperative blood transfusion. Packed cell volume (PCV) and oxygen extraction decreased, and cardiac index and pulmonary wedge pressure increased, although end-systolic area was unchanged. PCV was not significantly different between patients who lost less than or greater than 20% of their initial blood volume. This preoperative manoeuvre, which reduces loss of red blood cells, allowed major surgery to be completed safely without blood transfusion.

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Introduction

The risk of alloimmunisation and transmission of viral infection from homologous blood transfusion is well known. In addition, it is suggested that transfusion may promote tumour growth.[1] Some patients may refuse blood transfusions on religious grounds.[2] Transfusion with donor blood may be diminished by predeposited autologous blood,[3] intraoperative autotransfusion with a cell-saver,[4] and haemodilution techniques. With haemodilution, fewer red cells are lost because of the non-linear decrease in packed cell volume after the procedure.[5] Preoperative acute haemodilution can be achieved in two ways. First, by withdrawal of blood and simultaneous infusion of plasma substitutes (normovolaemic haemodilution).[6] Second, by rapid infusion of fluid without blood withdrawal (hypervolaemic haemodilution). We have evaluated the effects of hypervolaemic haemodilution on haemodynamics, left ventricular size, systemic oxygenation, and packed cell volume to establish whether this technique avoids blood transfusion peroperatively.

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Patients and methods

16 consecutive Jehovah's Witness patients (3 males, 13 females; mean age [SD], 51 [14] years) were admitted for major surgery and refused both homologous blood products and autologous transfusion. All patients gave informed consent to the study protocol, which was approved by an ethical committee at the Erasmus University Hospital, Rotterdam.

On the day of surgery two intravenous cannulae were inserted. The radial artery was cannulated and a thermodilution catheter ('Swan Ganz', AEL, USA) introduced into a pulmonary artery via the internal jugular vein. 2·5 mg intravenous midazolam was given before catheter placement. Heart rate, arterial blood pressure, pulmonary artery pressures, and right atrial pressure were monitored continuously ('Horizon 2000', Mennen Medical, Israel). After 30 min stabilisation, baseline values of mean arterial pressure (MAP), mean pulmonary artery pressure ($\overline{PAP}$), pulmonary wedge pressure (PWP) and cardiac output were obtained. In addition, arterial and mixed venous blood samples were taken for measurements of haemoglobin (Hb), Hb oxygen saturation ('Spectrophotometer OSM3', Radiometer, Copenhagen), packed cell volume (PCV), and pO_2, pCO_2, and pH ('ABL 330', Radiometer, Copenhagen). Systemic vascular resistance, pulmonary vascular resistance, oxygen flux, and oxygen uptake were calculated from these data. Oxygen flux is the product of arterial oxygen content and cardiac output. Oxygen uptake is the product of cardiac output and the arteriovenous oxygen content difference. The oxygen extraction ratio is calculated by dividing oxygen uptake by oxygen flux.

Anaesthesia was induced with fentanyl 5 μg/kg, thiopentone 5 mg/kg, and pancuronium 0·1 mg/kg. After tracheal intubation, the lungs were ventilated with 70% nitrous oxide in oxygen, and tidal volume was adjusted to achieve normocapnia. Anaesthesia was maintained with fentanyl and enflurane (end-tidal 0·4 vol %) and muscle relaxation was achieved with pancuronium. The bladder was catheterised.

A transoesophageal ultrasound transducer (5 MHz 'Toshiba', connected to a Toshiba 'SSH 160' machine) was placed for continuous and real-time visualisation of the heart. The transducer was positioned to obtain a two-dimensional short-axis view of the left ventricle at the level of the papillary muscles. After the optimum view had been obtained, the steering mechanisms of the transducer were locked to maintain the identical cross-sectional view throughout the period of volume-loading. Simultaneously with the haemodynamic recordings, the images were recorded on 'VHS' videotape for subsequent analysis. The end-diastolic and end-systolic enclosed areas of the left ventricle were measured after tracing the endocardial borders. Three consecutive beats were averaged.

After 15 min stabilisation, haemodynamic and echocardiographic measurements together with an analysis of blood gases were made. We undertook hypervolaemic haemodilution in three equal steps. 500 ml dextran 40 and 500 ml Ringers lactate were infused over 10 min. All measurements were repeated after each step. After surgery began, the same variables, except for echocardiographic values, were recorded after the 500 ml blood loss. Peroperatively, Ringers lactate was infused in a volume equal to the urine output plus 8 ml/kg per h (to compensate for fluid loss from the wound). Blood loss was replaced by an equal volume of gelatin solution.

Immediately before surgery was completed, the forced infusion was stopped. Measurements were repeated at the end of surgery, and at 20 min, 2 h, and 4 h thereafter.

The reported baseline blood volumes are calculated values.[7] The accepted probability for a statistical difference between means was $p < 0.05$. Statistical analysis of results was by Student's *t*-test and the Wilcoxon signed-rank test.

Results

11 patients underwent a laparotomy and 1 a nephrectomy; 3 patients had a hip replacement; and 1 patient received a facial bone reconstruction. Haemodynamic and echocardiographic results are summarised in table I. Stepwise acute hypervolaemic haemodilution, with a change in PCV from 36·9 (3·1) to 26·3 (2·4), resulted in a 29·3% decrease in the mean systemic vascular resistance index. The increase in the mean cardiac index of 27·5% (2·45 [0·65] *vs* 3·10 [0·68]) correlated with a 27·6% increase in the mean end-diastolic area of the left ventricle. Increases in mean PWP and $\overline{PAP}$ from 5·3 mm Hg (3·2) to 20·8 mm Hg (4·2) and from 12·3 mm Hg (3·9) to 31·0 mm Hg (5·2) respectively, were recorded. Other haemodynamic variables did not change, except for a slight increase in MAP and a slight decrease in heart rate. 2 hours postoperatively all haemodynamic variables had returned to pre-anaesthetic

TABLE I—SYSTEMIC HAEMODYNAMIC MEASUREMENTS BEFORE AND AFTER INDUCTION OF ANAESTHESIA

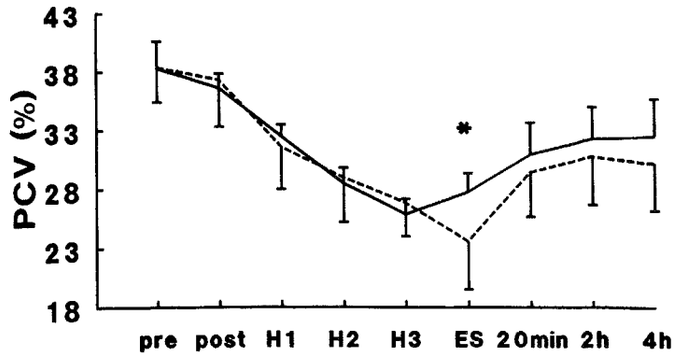
—	HR (bpm)	MAP (mmHg)	$\overline{PAP}$ (mmHg)	PWP (mmHg)	CI (l/min/m²)	EDA (cm²)	ESA (cm²)	PCV (%)
Pre-induction	85 *(15)*	104 *(28)*	14·3 *(3·1)*	4·9 *(2·9)*	3·06 *(0·85)*	..	.	38·3 *(2·8)*
Post-induction	95† *(16)*	81† *(15)*	12·3† *(3·9)*	5·3 *(3·2)*	2·43† *(0·65)*	12·3 *(3·7)*	6·0 *(2·7)*	36·9 *(3·1)*
H1	84* *(15)*	87† *(15)*	19·4†* *(4·3)*	10·8†* *(4·4)*	2·94* *(0·78)*	14·4* *(3·9)*	6·6 *(2·9)*	32·1†* *(2·8)*
H2	83* *(14)*	87† *(15)*	24·9†* *(3·6)*	16·6†* *(4·1)*	3·07* *(0·74)*	15·7* *(3·5)*	6·2 *(2·5)*	28·8†* *(2·9)*
H3	85* *(15)*	91†* *(15)*	31·0†* *(5·2)*	20·8†* *(4·2)*	3·10* *(0·68)*	15·7* *(3·9)*	5·8 *(2·3)*	26·3†* *(2·4)*
ES	89 *(20)*	83† *(15)*	19·8†* *(7·8)*	11·4†* *(6·6)*	3·19* *(0·89)*	..	..	26·6† *(3·2)*
20 min post-op	89 *(17)*	92* *(15)*	17·0†* *(4·0)*	6·5 *(4·6)*	3·53 *(1·19)*	..	..	30·6†* *(3·4)*
2 h post-op	87* *(13)*	86† *(16)*	14·9 *(3·1)*	4·3 *(2·8)*	3·43 *(1·24)*	..	..	31·6†* *(3·6)*
4 h post-op	88* *(15)*	84† *(15)*	15·0 *(3·2)*	4·4 *(3·3)*	3·51 *(0·91)*	..	..	31·6†* *(3·8)*

H1, H2, and H3 show data for each step of hypervolaemic haemodilution up to the end of surgery (ES)
HR = heart rate; CI = cardiac index, ESA = end systolic area.
*$p < 0.05$ compared with postinduction values †$p < 0.05$ compared with pre-anaesthetic values.

TABLE II—SYSTEMIC OXYGENATION BEFORE AND AFTER INDUCTION OF ANAESTHESIA

—	PaO_2 (mmHg)	$PaCO_2$ (mmHg)	PvO_2 (mmHg)	$PvCO_2$ (mmHg)	O_2 flux l/min/m²	VO_2 ml/min/m²	ER (%)	FiO_2
Pre-induction	87 *(15)*	36 *(3)*	41 *(4)*	40 *(3)*	505 *(144)*	102 *(21)*	0·21 *(0·04)*	0·21
Post-induction	168† *(29)*	32 *(3)*	44† *(4)*	37* *(4)*	418† *(127)*	91† *(21)*	0·23† *(0·05)*	0·32
H1	166† *(33)*	33 *(3)*	46† *(4)*	36* *(3)*	431† *(122)*	84† *(17)*	0·20* *(0·04)*	0·32
H2	167† *(29)*	33 *(3)*	46† *(4)*	36* *(3)*	404† *(103)*	80†* *(16)*	0·21* *(0·05)*	0·32
H3	170† *(27)*	32 *(4)*	47† *(4)*	35* *(3)*	378† *(97)*	73†* *(15)*	0·20* *(0·06)*	0·32
ES	135†* *(38)*	36* *(4)*	45† *(6)*	39 *(4)*	399† *(123)*	82†* *(21)*	0·22 *(0·07)*	0·32
20 min post-op	88* *(19)*	42†* *(4)*	43 *(6)*	45†* *(4)*	477 *(151)*	112* *(35)*	0·24† *(0·05)*	0·21
2 h post-op	102* *(24)*	39 *(5)*	42 *(6)*	44†* *(6)*	492 *(164)*	119†* *(33)*	0·26† *(0·07)*	0·21
4 h post-op	96* *(24)*	39* *(4)*	42 *(6)*	43* *(4)*	500 *(138)*	123* *(30)*	0·25 *(0·06)*	0·21

*$p < 0.05$ compared with postinduction values †$p < 0.05$ compared with pre-anaesthetic values



Change in PCV pre and post anaesthetic induction.

——, <20% blood loss, – – –, >20% blood loss. *p<0·05.

values except for the systemic vascular resistance index. The increase in cardiac output was associated with an increase in end-diastolic area. With further volume loading, end-diastolic area no longer increased, and the rise in cardiac output was associated with a decrease in end-systolic area. In no patient did an increase in end-diastolic area lead to a significant decrease in cardiac output.

The results of systemic oxygenation are shown in table II. Stepwise hypervolaemic haemodilution did not lead to changes in arterial or mixed venous blood gases. Oxygen flux was constant over the study period because cardiac output compensated for decreased oxygen transport capacity that took place with haemodilution and blood loss. The constant oxygen flux and decreased consumption resulted in a decreased extraction ratio.

The changes in PCV in patients with a blood loss 20% of their calculated initial blood volume (14·2 [4·1]%; n = 8) and in patients with a blood loss >20% of the baseline blood volume (42·2 [17·7]%; n = 8) are shown in the figure. 2 h postoperatively the difference in PCV was not statistically significant (32·3 [2·7] *vs* 30·8 [4·1]). Only at the end of surgery was a significant difference in PCV seen between the two groups (27.8 [1·6] *vs* 24·8 [3·9]). Blood loss in all patients was 28·2 [18·3]% of the calculated initial blood volume.

Discussion

Hypervolaemic haemodilution improves cerebral circulation[8] and may be a useful treatment for haemorrhagic disorders in pre-eclampsia.[9] In these reports, haemodilution was induced slowly over 24 h or more. Apart from one case-report of an anaemic Jehovah's Witness,[10] the effects of rapid volume loading have not been documented in man.

We found significant increases in PWP and $\overline{PAP}$. This result may be because of an increased venous return to the heart after a reduction of viscous resistance of blood by haemodilution.[11] Excessive volume loading is also likely to have contributed to an increased PWP, but in no patient was there any clinical evidence of pulmonary oedema. Intermittent positive pressure ventilation may have avoided such a complication. Extubation of the patients' lungs was possible directly after surgery because, when the forced infusion was stopped, PWP and $\overline{PAP}$ returned quickly to normal.

Although the end-diastolic diameter of the left ventricle increased, there was no reduction in left ventricular performance because end-systolic diameter remained the same.

A small but significant reduction of oxygen consumption and extraction ratio was observed. Postoperatively the extraction ratio increased to a value above that seen before induction of anaesthesia and was due to an increase in oxygen consumption that confirms the findings of others.[12]

Acute preoperative isovolaemic haemodilution gives a supply of the patient's blood and results in fewer red cells being lost in an episode of haemorrhage. This technique is time consuming and needs special arrangements—eg, collection and storage of patients' blood. The technique of hypervolaemic haemodilution allowed major surgery without blood transfusion and was tolerated safely by all patients. However, this study applies only to surgical patients with no associated medical illness and cannot be recommended in patients with a compromised cardiovascular system. Furthermore, it is uncertain whether hypervolaemic haemodilution can influence the total amount of blood loss during surgery.

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From The Lancet

The Congo cannibals

Mr Herbert Ward, one of the surviving members of the rear guard of Mr Stanley's expedition, and who spent no less than five years in Central Africa (on the banks of the Congo), related some of his experiences . . . Amongst topics of special interest from a medical point of view were the custom of the ordeal by poison among the Bateke of Stanley Pool, the culprit being selected by the "medicine man", and his acquittal depending upon his survival of the test. The extent to which rum drinking is carried was illustrated by an amusing anecdote in which a native praised the virtues of the spirit as giving pleasure to the partaker of it and profit in the subsequent disposal of the bottle. But, in reply to Mr. Ward's query as to the "next morning's headache", the native admitted that that at least could not be sold. A few details of the "sleeping sickness" were given. This curious affection is invariably fatal, and is characterised by pain in the back of the neck, followed by gradually increasing somnolence. Sometimes whole districts are ravaged by it.

(Oct 25, 1890)