LETTERS TO THE EDITOR

Residual shunt rate—It was stated that the buttoned device has the highest residual shunt rate compared with all previous devices. A careful comparison of residual shunts for all devices\textsuperscript{1–11} revealed similar residual shunt rates. Furthermore, the effective occlusion rate with non-manualized buttoned shunt is excellent acutely, and has further improved with follow up.\textsuperscript{7,11} The Nitinol mesh of the Amplatz device has better acute full occlusion rates, but the buttoned device has achieved good long term results because of better endothelialisation of the polyurethane foam. Elimination of residual shunts is possible with better centring and the use of inverted counter-occluders, with the newer modifications of the buttoned device. We centre and centring on demand buttoned devices.\textsuperscript{12}

The reason for these misconceptions is not clear, but is probably related to referring to an abstract\textsuperscript{10} from our group while ignoring many full papers in a variety of journals\textsuperscript{4–10,13–18} including the full paper\textsuperscript{11} of the referenced abstract.\textsuperscript{11,12} Or it may be related to relying on personal communications in preference to objective, peer reviewed, published data. Another misunderstanding is wire problems and chronic wire toxicity. Rigby has only superficially touched on the wire problems of the different devices, both acutely and on follow up. Acute problems, including arterial perforation and interference with heart valves, have been shown with all devices. Wire fractures up to 80% have been shown with the Clamshell device\textsuperscript{19} and with its successor the CardioSeal device (10% for the first year only). Among the metal used with the different devices, Nitinol has the most acute and chronic complications, despite its very attractive functional characteristics. Unfortunately, chronic nickel toxicity is not simply theoretical, as Rigby mentions, but rather a well established fact. Hundreds of people have died from lung cancer in the nickel mines, coronary spasm has been shown in experimental animals, and allergic reactions and tissue necrosis are well known. We believe that in 30–50 years time many wires in all devices will be fragmented and some others will have demonstrated some toxicity.

Although the 10 year follow up of the buttoned device used less than 1% wire related problems, we believe that there is a need for wireless non-toxic devices for heart defect occlusion. We believe that all current disc devices have some limitations and very similar application and we do not believe that the Amplatz, CardioSeal or the buttoned device can correct more defects than any other. Perhaps a significant factor for the proper device selection is the cost and availability. Wire price conscious than USA and the UK the fact that the Amplatz device is four times more expensive than the buttoned device can be important. The availability worthful long term results with one device\textsuperscript{17,18} and the absence of long term follow up with the others is another factor. However, we agree with Rigby in his assessment that “none of the devices is perfect, each has its own strengths and weaknesses” and “the modifications of the existing devices and the introduction of new systems will result in current practice changing rapidly in the near future.”

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Coronary pressure measurements: catheter induced errors

Editor.—Coronary pressure derived fractional flow reserve (FFR), as reviewed by Pijls and Bruyne,\textsuperscript{19} provides an excellent and reproducible technique to estimate the severity of a coronary lesion, and is a significant advance over coronary flow reserve. Three points need to be raised.

First, the arterial pressure measurement should be taken during diastole as most coronary flow is during diastole (not strictly true for the right ventricle). Using mean arterial pressure will induce significant errors.

Second, calculations of FFR without full assessment of the central venous pressure (CVP) may incur significant errors, as the vast majority of patients do not have a CVP of 0. The percentage error incurred when the CVP is not included can be calculated from equation 1.

Percentage error in FFR = \[ \frac{P_{cvp} - (P_a + P_d)\times100}{P_d - P_{cvp}} \]  

(1)

Where \( P_a \) = arterial pressure; \( P_d \) = distal pressure; and \( P_{cvp} \) = central venous pressure.

This is graphically illustrated in fig 1A, which shows that the percentage error incurred is significant.

Finally, even though catheter technology has advanced significantly over the past decade resulting in decreased physical size and increased flexibility, sources of error resulting from the physical size of the catheters should be appreciated if correct interpretations of the readings and derived interpretations of the readings and derived measurements and fractional flow reserve. Fractional flow reserve is useful in evaluating the influence of an epicardial coronary stenosis on myocardial blood flow. Circulation 1999;99:2318–93.


**Diamorphine and British cardiology: so we are right!**

Editor,—Diamorphine has been used extensively in cardiology in the UK in the management of acute left ventricular dysfunction and myocardial infarction. Our European and American colleagues however remain firmly committed to the use of morphine for the same clinical situations. To date no studies have revealed any difference in efficacy between morphine and diamorphine.

Ischaemic preconditioning is thought to play an important role in reducing the severity of myocardial damage due to coronary artery occlusion, and for relieving pain during a myocardial infarction. Our European and American colleagues however remain firmly committed to the use of morphine for the same clinical situations.

The recent discovery that activation of the opioid \( \delta \) receptor on the myocardium can exert a protective effect on myocardial ischaemia similar in extent to classic ischaemic preconditioning may have important implications. Morphine is known to act predominantly via the opioid \( \mu \) receptor whereas diamorphine acts at the \( \delta \) receptor. By implication, diamorphine could offer the benefits of ischaemic preconditioning to patients who receive it compared to those who receive morphine.
Unfortunately adequate numbers and practicalities of patient stratification would make a clinical study difficult, especially for two off patent drugs; however, we may still be right.

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Decrease of plasma fibrinogen after eradication of Helicobacter pylori infection in patients with ischaemic heart disease

EDITOR,—Infectious agents such as Chlamydia pneumoniae or Helicobacter pylori have been linked to ischaemic heart disease (IHD). Raised plasma fibrinogen has been claimed as a possible link between H pylori infection and IHD; however, fibrinogen as an acute phase protein may only reflect systemic inflammation from other underlying diseases (usually not considered in previous publications).

Our aim was to evaluate retrospectively a possible relation between H pylori infection, plasma fibrinogen, and IHD. We then planned to test the hypothesis that raised fibrinogen induced by H pylori is only important in patients with IHD in the absence of other systemic inflammation parameters. Finally, we attempted to lower plasma fibrinogen in this subgroup of patients by eradicating H pylori.

We examined the notes of all patients referred for coronary artery angiography (at least two weeks after myocardial infarction) from August 1994 to August 1995 with suspected or proved IHD for routinely analysed plasma fibrinogen and H pylori status. Systemic inflammation was deemed absent if body temperature, leucocytes, C reactive protein (CRP), and erythrocyte sedimentation rate (ESR) were within normal ranges. Subgroups were defined according to H pylori status, IHD, and plasma fibrinogen in the presence or absence of systemic inflammation. Of 317 patients, 245 (77%) had IHD with a stenosis ≥ 70% of at least one vessel, 127 (40%) had IHD and H pylori infection. Forty nine of these 127 (15% of all patients) also had raised fibrinogen (> 3.5 g/l). Only 20 of these 49 patients had normal systemic inflammation parameters (6% of all patients). A causal association between IHD and H pylori infection was suspected for these patients. This hypothesis was supported by a higher prevalence of raised fibrinogen in H pylori positive patients with IHD in the absence of systemic inflammation (35.1% v 17.5%; p = 0.05, one sided χ² test; relative risk (RR) 1.7; odds ratio (OR) 2.0; 95% confidence interval (CI) 0.9 to −4.6). Comparing only patients with increased fibrinogen without systemic inflammation, the prevalence of IHD was higher in H pylori positive patients (95% v 63.6%; p < 0.05, one sided χ² test; RR 1.3; OR 7.3; 95% CI 0.7 to −73). By performing multiple regression analysis, serologically determined positive H pylori status was significantly (p < 0.001) associated with raised plasma fibrinogen after adjusting for age, history of peptic ulcer, CRP, ESR, and leucocyte count. Bivariable analysis of these parameters influenced fibrinogen levels (but not IHD). Because of their dyspeptic symptoms, 12 of the above mentioned 20 patients agreed to be tested for active H pylori infection by 14C urea breath test and 11 of 12 were positive and included in an H pylori eradication trial described elsewhere, 10 of them becoming negative. After obtaining written informed consent we had the opportunity to follow up these 10 patients for 6 months (table 1).

In conclusion, H pylori infection may be regarded as a risk factor for IHD in a very small proportion of patients (6%, borderline significance). Our results may explain why even large epidemiological studies do not show a significant association between H pylori infection and IHD. Raised plasma fibrinogen could be a link for the development of IHD in this predefined subgroup.

Treatment (H pylori eradication) leads to a decrease of plasma fibrinogen in single cases with a known low risk of H pylori reinfection. However, in the individual patient there is striking evidence to assess fibrinogen repeatedly: spontaneous fluctuations (as seen in two of 10 patients) can possibly reflect the stability or instability of IHD or interfering concomitant diseases. Before making a decision whether to treat hyperfibrinogenaemia with H pylori eradication, the absence of all other signs of systemic inflammation is essential.

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Table 1 Inflammation parameters and plasma fibrinogen before and after H pylori eradication

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before eradication</th>
<th>After eradication</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>First contact</td>
<td>Before treatment*</td>
</tr>
<tr>
<td>Leucocytes (&gt;10⁹/l)</td>
<td>7.14</td>
<td>6.81</td>
</tr>
<tr>
<td>CRP (g/l)</td>
<td>0.007</td>
<td>0.006</td>
</tr>
<tr>
<td>ESR (mm 1st h)</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Fibrinogen (g/l)</td>
<td>4.13*‡</td>
<td>3.45†</td>
</tr>
<tr>
<td>(SD)</td>
<td>(0.43)</td>
<td>(0.89)</td>
</tr>
</tbody>
</table>

Values are means.

* to 12 months after first contact.
†Raised values because two patients had respiratory infections at that time.
‡ p < 0.01 (paired t test) compared with first contact or 6 months.
§ p < 0.01 (paired t test) mean of first contact and before treatment compared with the mean of 3 and 6 months after eradication.

Transcatheter closure of atrial septal defects

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Heart 1999 82: 644
doi: 10.1136/hrt.82.5.644

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