Fluroxene and Isolated Heart Muscle

Alan H. Goldberg, M.D., Ph.D.,* Young Z. Sohn, M.D.,† W. P. C. Phear, B.Sc.‡

The direct myocardial effects of fluroxene were examined in isometrically and isotonically contracting isolated rat heart muscle. MAC, the minimum anesthetic concentration needed to prevent movement in response to tail clamping, was found to be 5.0 vol per cent fluroxene in the rat. At 4.6 vol per cent fluroxene, peak developed isometric tension and maximum rate of tension development were decreased 29 and 24 per cent, respectively. At 11 vol per cent, the depressions were 39 and 33 per cent. At 26.4 vol per cent, the depressions were around 60 per cent.

$V_{\text{max}}$ (the maximum shortening velocity of unloaded muscle) of the force-velocity relation was unaltered by fluroxene concentrations of 0.5, 4.6, and 11 vol per cent. Even at 26.4 vol per cent, the depression in $V_{\text{max}}$ was only 25 per cent. $P_{0}$ (the maximum force at zero velocity), work, and power were lowered much more, with reductions ranging from 15 to 27 per cent at 4.6 vol per cent, from 40 to 42 per cent at 11.0 vol per cent, and from 65 to 69 per cent at the 26.4 vol per cent. Series elastic extension was unchanged at 0.8 and 4.6 vol per cent fluroxene, but was decreased 16 per cent at 11.0 vol per cent and 46 per cent at 26.4 vol per cent fluroxene.

The data indicate the fluroxene has a direct negative inotropic effect that is associated with increased series elastic stiffness, but does not involve $V_{\text{max}}$ of the force-velocity relation until quite high anesthetic concentrations are reached.

Comparative studies were also carried out with halothane. MAC for halothane in the rat was 1.0 vol per cent. The relative potency of halothane compared with fluroxene in depression of $V_{\text{max}}$ was 13.2, and its relative potency in depression of $P_{0}$ was 4.0 (Key words: Anesthetics, volatile, fluroxene; Anesthetics, volatile, halothane; Heart, myocardial function.)

Fluroxene appears to be unique among general anesthetic agents in that arterial blood pressure and cardiac output are not depressed at moderate anesthetic concentrations, and may actually increase during deep anesthesia.† Although no change occurs in the ballistocardiogram in man, central venous pressure does increase, suggesting the possibility of direct myocardial depression.‡ The purpose of this paper is to describe the direct effect of this anesthetic on the mechanical properties of isolated heart muscle.

**Methods**

Twenty-seven rat left ventricular trabeculae carneaæ preparations were utilized. The average rat weight was 216 g ± 17; the maximum muscle length, 6.57 mm ± 0.39; the dried muscle weight, 1.94 mg ± 0.09 (means ± standard deviations).

Each preparation was placed in an oxygenated (95 per cent O₂, 5 per cent CO₂) muscle bath containing Krebs-bicarbonate solution, as previously described.§ Tempera-

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**ABBREVIATIONS**

$+\text{dP/dt} =$ maximum rate of tension development

$-\text{dP/dt} =$ maximum rate of tension relaxation

$P_{0} =$ maximum force at zero velocity

$RT =$ relaxation time (time for $T_{\text{des}}$ to decay by 90 per cent)

$T_{\text{des}} =$ peak developed tension

$T_{p} =$ time to peak tension

$T_{r} =$ resting tension

$V_{\text{max}} =$ maximum velocity of shortening of unloaded muscle

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<table>
<thead>
<tr>
<th></th>
<th>Control Values</th>
<th>0.8</th>
<th>2.3</th>
<th>4.6</th>
<th>7.1</th>
<th>11.0</th>
<th>20.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>$T_p$ (g/mm²)</td>
<td>2.02 ± 0.37</td>
<td>1.92 ± 0.411</td>
<td>1.57 ± 0.391</td>
<td>1.45 ± 0.321</td>
<td>1.35 ± 0.271</td>
<td>1.19 ± 0.251</td>
<td>0.87 ± 0.191</td>
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<tr>
<td></td>
<td></td>
<td>(-7.8 ± 2.0)</td>
<td>(-16.3 ± 2.7)</td>
<td>(-29.2 ± 2.8)</td>
<td>(-33.0 ± 1.9)</td>
<td>(-38.7 ± 2.4)</td>
<td>(-58.4 ± 2.1)</td>
</tr>
<tr>
<td>$T_r$ (g/mm²)</td>
<td>0.52 ± 0.19</td>
<td>0.51 ± 0.20</td>
<td>0.52 ± 0.18</td>
<td>0.52 ± 0.17</td>
<td>0.49 ± 0.17</td>
<td>0.50 ± 0.19</td>
<td>0.53 ± 0.21</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(+0.5 ± 1.1)</td>
<td>(+0.4 ± 1.0)</td>
<td>(+0.3 ± 1.4)</td>
<td>(+2.3 ± 3.9)</td>
<td>(+0.7 ± 1.6)</td>
<td>(+0.9 ± 1.9)</td>
</tr>
<tr>
<td>+ dP/dt (g/mm²/sec)</td>
<td>32.46 ± 5.34</td>
<td>27.53 ± 2.13*</td>
<td>25.18 ± 3.011</td>
<td>22.23 ± 2.841</td>
<td>21.11 ± 2.251</td>
<td>20.62 ± 2.071</td>
<td>13.37 ± 1.811</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-5.6 ± 3.5)</td>
<td>(-14.9 ± 2.3)</td>
<td>(-23.7 ± 3.0)</td>
<td>(-28.8 ± 3.6)</td>
<td>(-32.5 ± 2.9)</td>
<td>(-60.1 ± 2.1)</td>
</tr>
<tr>
<td>- dP/dt (g/mm²/sec)</td>
<td>19.34 ± 3.91</td>
<td>16.48 ± 2.731</td>
<td>15.09 ± 2.651</td>
<td>13.91 ± 2.131</td>
<td>12.88 ± 1.941</td>
<td>10.92 ± 1.571</td>
<td>7.71 ± 1.131</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-10.4 ± 2.5)</td>
<td>(-18.3 ± 3.5)</td>
<td>(-27.3 ± 2.5)</td>
<td>(-31.2 ± 1.8)</td>
<td>(-38.6 ± 3.3)</td>
<td>(-61.0 ± 2.9)</td>
</tr>
<tr>
<td>TPT (sec)</td>
<td>0.065 ± 0.002</td>
<td>0.060 ± 0.002</td>
<td>0.060 ± 0.002*</td>
<td>0.061 ± 0.002*</td>
<td>0.059 ± 0.0011</td>
<td>0.055 ± 0.0011</td>
<td>0.057 ± 0.002*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(-7.2 ± 1.1)</td>
<td>(-8.8 ± 3.1)</td>
<td>(-7.8 ± 3.3)</td>
<td>(-8.8 ± 1.2)</td>
<td>(-15.0 ± 1.3)</td>
<td>(-5.8 ± 3.5)</td>
</tr>
<tr>
<td>RT (sec)</td>
<td>0.000 ± 0.006</td>
<td>0.001 ± 0.008</td>
<td>0.000 ± 0.006</td>
<td>0.089 ± 0.006</td>
<td>0.089 ± 0.007</td>
<td>0.088 ± 0.006</td>
<td>0.001 ± 0.005</td>
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<td></td>
<td></td>
<td>(+1.6 ± 2.0)</td>
<td>(-1.9 ± 2.9)</td>
<td>(-3.0 ± 1.8)</td>
<td>(-1.4 ± 3.7)</td>
<td>(-1.8 ± 2.3)</td>
<td>(-1.1 ± 4.4)</td>
</tr>
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</table>

Numbers in parentheses are percentage changes from control, calculated from individual rather than group data.

* $P < 0.05$.

† $P < 0.01$.

‖ $P < 0.001$. 

Table 1. Effects of Fluoxetine on Isometric Contractions (Means ± SEM)
Fluoxetine Concentration (Vol.%)

Fig. 1. Dose-response curves, illustrating effects of fluoxetine on peak developed tension ($T_{pd}$) and resting tension ($T_r$). Brackets in this and all subsequent figures indicate standard errors.

Fluoxetine Concentration (Vol.%)

Fig. 2. Effects of fluoxetine on the maximum rate of tension development (+ dP/dt) and time to peak tension (TPT).

Fluoxetine Concentration (Vol.%)

Fig. 3. Effects of fluoxetine on the maximum rate of tension relaxation (− dP/dt) and relaxation time (RT).

ture was held constant at 37 C and pH at 7.4. The muscles were stimulated to contract 15 times per minute by a square-wave pulse 10 per cent above threshold and 6.0 msec in duration, applied through platinum electrodes placed 1 cm apart, parallel to the tissue.

After 30 minutes of isometric contractions, the length–tension curve of each preparation was determined. The muscle length was then held at the peak of the length–tension curve while isometric equilibration occurred over the next 30 minutes.

Five muscles, induced to contract isotonically, were exposed to four fluoxetine concentrations: 0.8, 4.6, 11.0, and 26.4 vol per cent. Six isometrically contracting muscles were exposed to two additional concentrations, 2.3 and 7.1 vol per cent. All anesthetic concentrations were delivered in random order for 30 minutes each. Control data were obtained prior to any anesthetic exposure, before the penultimate, and after the final fluoxetine concentration. Data from the three control periods were used to correct the observed fluoxetine effects for changes due to time alone.

An additional 16 muscles (ten isotonic and six isometric) were exposed to five concentrations of nitrogen: 5, 10, 15, 20, and 25 vol per cent, delivered in an identical manner. These data were used to correct the observed fluoxetine effects for changes due to reductions in delivered oxygen. The maximum change in any value with $N_2$ was −14 per cent (P$_o$ at 25 vol per cent $N_2$).

In force–velocity studies, the maximum initial velocity of isotonic shortening is plotted against the total load (force) lifted. The result typically is an inverse curvilinear curve. The ends of the curve are $V_{max}$ and $P_o$, where $V_{max}$ represents the maximum velocity at zero load and $P_o$ represents the maximum force at zero velocity. The equation for the curve is $(P + a) V = b (P_o - P)$, where P is any load and V is the velocity of shortening at that load. The asymptotic values of P and V are "a" and "b," respectively.

In order to compare the effects of halothane and fluoxetine on $V_{max}$ and $P_o$, 11
### Table 2. Effects of Fluoxetine on Isotonic Contractions (Means ± SEM)

<table>
<thead>
<tr>
<th></th>
<th>Control Values</th>
<th>0.8 (Vol Per Cent)</th>
<th>1.6 (Vol Per Cent)</th>
<th>11.0 (Vol Per Cent)</th>
<th>26.4 (Vol Per Cent)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>V_max (muscle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>lengths/sec)</strong></td>
<td>1.11 ± 0.10</td>
<td>1.00 ± 0.26</td>
<td>0.99 ± 0.22</td>
<td>0.98 ± 0.23</td>
<td>0.64 ± 0.171</td>
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<tr>
<td></td>
<td>(+3.5 ± 3.7)</td>
<td>(-1.1 ± 4.0)</td>
<td>(+0.2 ± 1.7)</td>
<td>(-24.9 ± 9.0)</td>
<td></td>
</tr>
<tr>
<td><strong>P_o (g/mm²)</strong></td>
<td>4.52 ± 0.37</td>
<td>3.98 ± 0.66</td>
<td>3.51 ± 0.49*</td>
<td>2.56 ± 0.501</td>
<td>1.38 ± 0.251</td>
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<tr>
<td></td>
<td>(-4.6 ± 4.9)</td>
<td>(-23.7 ± 6.6)</td>
<td>(-40.1 ± 5.9)</td>
<td>(-64.5 ± 7.9)</td>
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<tr>
<td><strong>a (g)</strong></td>
<td>3.11 ± 0.35</td>
<td>3.10 ± 0.25</td>
<td>3.10 ± 0.37</td>
<td>2.90 ± 0.25</td>
<td>2.30 ± 0.25</td>
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<td>(-2.1 ± 3.8)</td>
<td>(+3.1 ± 11.0)</td>
<td>(-8.5 ± 5.0)</td>
<td>(-26.9 ± 8.9)</td>
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<tr>
<td><strong>b (mm/sec)</strong></td>
<td>5.13 ± 0.91</td>
<td>5.83 ± 1.08</td>
<td>6.32 ± 1.45</td>
<td>7.60 ± 1.01</td>
<td>8.46 ± 2.17</td>
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<tr>
<td></td>
<td>(+6.3 ± 2.2)</td>
<td>(+18.2 ± 8.27)</td>
<td>(+48.5 ± 13.1)</td>
<td>(+62.2 ± 18.1)</td>
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<tr>
<td><strong>Work (g-mm)</strong></td>
<td>0.61 ± 0.08</td>
<td>0.57 ± 0.11</td>
<td>0.39 ± 0.07</td>
<td>0.30 ± 0.05*</td>
<td>0.11 ± 0.02*</td>
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<tr>
<td></td>
<td>(+3.4 ± 8.0)</td>
<td>(-15.0 ± 4.5)</td>
<td>(-41.7 ± 3.1)</td>
<td>(-69.4 ± 5.2)</td>
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<tr>
<td><strong>Power (g-mm/sec)</strong></td>
<td>4.69 ± 0.84</td>
<td>3.88 ± 0.64</td>
<td>3.42 ± 0.351</td>
<td>2.91 ± 0.341</td>
<td>1.45 ± 0.381</td>
</tr>
<tr>
<td></td>
<td>(-9.1 ± 6.5)</td>
<td>(-26.6 ± 1.3)</td>
<td>(-39.8 ± 4.7)</td>
<td>(-64.9 ± 7.0)</td>
<td></td>
</tr>
<tr>
<td><strong>Series elastic</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>extension (mm)</strong></td>
<td>0.25 ± 0.06</td>
<td>0.20 ± 0.03</td>
<td>0.19 ± 0.03</td>
<td>0.18 ± 0.02*</td>
<td>0.11 ± 0.01*</td>
</tr>
<tr>
<td><strong>@ 1 g load</strong></td>
<td>(+0.5 ± 2.3)</td>
<td>(-7.2 ± 2.0)</td>
<td>(-15.5 ± 2.9)</td>
<td>(-46.4 ± 8.1)</td>
<td></td>
</tr>
</tbody>
</table>

Numbers in parentheses are percentage changes from control, calculated from individual rather than group data.

* P < 0.05.
† P < 0.01.
†† P < 0.001.

Other muscles were exposed to halothane for 30 minutes, preceded and followed by appropriate control periods. Halothane was administered in the following concentrations: 0.26, 0.34, 0.68, 0.98, and 1.49 vol per cent.

In the isotonic studies, V_max and P_o of the force-velocity relation, series elastic extension, and work and power curves were calculated as previously described. For the isometric twitches, resting tension (T_r) and peak developed tension (T_p), maximum rates of rise and fall of tension (± dP/dt), time to peak tension (TPT) and 90 per cent relaxation time (RT; time for T_p to decay by 90 per cent) were also calculated as previously described.

Relative potencies of fluoxetine vs. halothane in depression of V_max and P_o were calculated as ratios of minimum anesthetic concentrations (MAC) needed to produce equivalent depressions of these variables. These were obtained from linear portions of parallel dose-response curves.

MAC for the rat was determined for fluoxetine (n = 6) and halothane (n = 7). Each rat was positioned with its head completely inside a tight-fitting nose cone through which the anesthetic (in oxygen) was passed at 2 l/min. Rectal temperature was maintained at 37 C by a flow of warm air over the animal's body. MAC was computed as the average of the highest concentration at which the animal responded to a hemostat applied to the tail and the lowest concentration at which the animal did not respond. All concentrations were administered until an equilibration period of at least 10 minutes was achieved. During this time, at least four consecutive gas samples showed no change in the anesthetic concentration.

Gas chromatography was used to determine anesthetic concentrations. For the iso-
lated muscle studies, samples were taken from the gas inlet just proximal to the bath. In the MAC studies, samples were taken from a catheter located inside the cone and adjacent to the animal’s nose.

**Results**

In isometric contractions (table 1), $T_{pd}$, $+\text{dP/dt}$ and $-\text{dP/dt}$ were depressed in relation to the concentration of fluroxene (figs. 1, 2, and 3). While these changes are all statistically significant, the curvilinear dose-response relationships indicate that the relative effects decrease as the anesthetic concentration increases.

There was no difference from control in $T_r$ (fig. 1) or RT (fig. 3) at any anesthetic concentration. TPT was significantly decreased to approximately the same extent (9 per cent) at all fluroxene concentrations (fig. 2).

$P_o$ of the force–velocity relationship (table 2, figs. 4 and 5) and work and power maxima (fig. 6) were affected in a manner similar to that observed with developed tension. $V_{max}$, however, was not altered up to and including the 11 vol per cent concentration (figs. 4 and 5). At 26 vol per cent fluroxene, the depression in $V_{max}$ was 25 per cent, in comparison with the 65 per cent decline in $P_o$. In addition, the constants “a” and “b” of the force–velocity relationship were not altered in a statistically significant manner by any anesthetic concentration.

Fluroxene did not significantly affect the extension of the series elastic element at the two lowest concentrations studied (table 2, fig. 7). At 11.0 vol per cent fluroxene, there was a 16 per cent reduction, and at 26.4 vol per cent, there was a 46 per cent reduction.

MAC's (vol per cent) were found to be 5.00 ± 0.37 (SD) for fluroxene and 1.00 ± 0.32 (SD) for halothane.

**Fig. 4.** Effects of fluroxene on $P_o$ and $V_{max}$ of the force–velocity relationship.

**Fig. 5.** Effect of fluroxene on force–velocity curves. $V_{max}$ is the intercept on the velocity axis, and $P_o$ is the intercept on the load (force) axis.
The relative potency of halothane compared with fluroxene (fig. 8) in depression of \( V_{\text{max}} \) was 13.2, and its relative potency in depression of \( P_o \) was 4.0.

**Discussion**

The MAC values we obtained in the rat for fluroxene (5.0 per cent) and halothane (1.0 per cent), measuring inhaled concentrations after steady-state conditions had been achieved, are close to data obtained by others who used either similar experimental techniques or end-expiratory gas sampling. We therefore felt justified in considering the 11 vol per cent concentration equivalent to a high anesthetic dose, and 26.4 vol per cent fluroxene to be well beyond the clinically useful range.

Our isolated heart muscle experiments indicate that fluroxene does have direct dose-related negative inotropic effects. However, dose–response curves representative of isometric data, including \( P_o \), work, and power maxima, and studies of elastic extension are all concave to the dose axis, resulting in decreasing relative effects with increasing anesthetic concentrations. This is in contrast to the dose–response curve for \( V_{\text{max}} \), which is unaffected even at 11.0 vol per cent fluroxene, a concentration equivalent to more than 2 MAC.

The \( V_{\text{max}} \) dose–response relationship appears to be at the bottom of a typical S-shaped curve, while the other variables may be at the upper ends of their curves, even though the maximum effects seen with a 5-MAC dose are well below 100 per cent depression.

At 1 MAC, \( V_{\text{max}} \) was reduced 48 per cent by halothane, but was unaffected by fluroxene. In the linear portions of their dose–response curves, which involve concentrations of fluroxene beyond 2 MAC, halothane is 13.2 times as potent as fluroxene in depressing \( V_{\text{max}} \).

At 1 MAC, fluroxene lowered \( P_o \) 22 per cent, and halothane reduced it 58 per cent. Relative potency calculations indicate that halothane is four times as potent as fluroxene in depressing \( P_o \).

The lack of effect of fluroxene on \( V_{\text{max}} \) at clinically useful concentrations could be indicative of unchanged velocity of chemical reactions involved in the contractile process; this would allow force-generating bonds to move at normal speed. This is consistent with the absence of significant change in \( "a" \) and \( "b" \) of the force–velocity relation, indicating no alteration in the amount of heat produced per unit of shortening or in the change in the rate of heat production per unit change in load. The depressions in \( P_o \), \( +dP/dt \), and \( -dP/dt \) probably reflect decreased numbers of bonds being formed and a reduction in the rate of calcium binding to troponin, possibly due to a limitation in the availability of myofibrillar calcium.
Increased stiffness of the series elastic component results in improved effectiveness in the transmission of tension from the contractile elements to the ends of the muscle units and would tend to modulate reductions in \(+\text{dP}/\text{dt}\). The constant, minor alteration in TPT reflects a small decrease in the duration of the active state. The constancy of \(T_\text{r}\) indicates no change in parallel elasticity; this may indicate the absence of any change in diastolic compliance, but further studies would be needed to prove this point.\(^9\)

The most striking finding we observed was the lack of effect of fluroxene on \(V_{\text{max}}\). This is in contrast to the results of Kemmotsu et al.,\(^10\) who reported 32 per cent depression in \(V_{\text{max}}\) and 45 per cent in \(F_m\) (\(P_0\)) at 1 MAC. However, their observations were made at a mean anesthetic concentration in the perfusate of 14.3 mg/100 ml. Assuming a water/gas partition coefficient of 0.84, this is equivalent to a vapor concentration of 17.0 vol per cent, which corresponds to 3.4 MAC. At this concentration, our results would be \(-11\) per cent for \(V_{\text{max}}\) and \(-52\) per cent for \(P_0\). The remaining difference in \(V_{\text{max}}\) is most likely to be due, at least in part, to the difference in temperature: 32 C for the study of Kemmotsu et al.\(^10\) and 37 C in this investigation.

The sparing of \(V_{\text{max}}\) until quite high concentrations are reached, when combined with decreased series elastic compliance and, in the intact organism, enhanced sympathetic nervous system activity,\(^2\) is consistent with the reported absence of reduction in the ballistocardiogram, arterial blood pressure, and cardiac output.\(^12\) These findings suggest that any myocardial depression that may occur with fluroxene can be easily counteracted by an increase in venous return to the heart.

The authors thank Mr. David M. McKay for excellent technical assistance.
References

CPAP WITH A FACE CHAMBER IN EARLY TREATMENT OF IDIOPATHIC RDS To avoid intubation in patients with IRDS (idiopathic respiratory distress syndrome) the authors have developed a face chamber consisting of a disposable latex ring fitted with styrene particles and a detachable lid. Using a bacteria-proof filter and air conditioner, gas is introduced at a flow rate of 12–15 l/min. Pressures as high as 15 cm H2O may be developed by use of a regulatory valve at the gas-outlet port. Ten unselected neonates were treated initially with CPAP (continuous positive airway pressure). Two infants died. In one neonate (CPAP) was discontinued due to apneic spells. Seven infants were successfully treated, with no complications noted.

The authors feel their apparatus is an advance on earlier equipment that involved the risk of venous congestion of neck veins and possible cochlear damage due to high noise levels. Easy access and nursing are other favorable factors. (Ahlström, H., Jonson, B., and Scenningsen, N.: Continuous Positive Airway Pressure with a Face Chamber in Early Treatment of Idiopathic RDS. Acta Paediatr Scand 62:433–436, 1973.)

ABSTRACTER’S COMMENT: A radical effect on prognosis of IRDS by the early use of CPAP claimed by the authors is unsubstantiated.

PROSTAGLANDIN F2α EFFECT ON THE NEONATE Possible clinical and biochemical alterations in the neonate were evaluated subsequent to induction of labor with prostaglandin F2α (PGF2α) and oxytocin. Twenty-three infants were studied in a double-blind investigation. Clinical observations and laboratory studies were performed on each neonate for 72 hours after birth. Umbilical cord PGF2α levels were not significantly higher in the group induced with PGF2α, compared with the group induced with oxytocin. There was no significant difference between the two groups of neonates in the clinical and metabolic parameters studied. (Blackburn, M. G., and others: Effects on the Neonate of the Induction of Labor with Prostaglandin F2α and Oxytocin. Am J Obstet Gynecol 116:847–853, 1973.)