Selection of Patients with Truncus Arteriosus for Surgical Correction

Anatomic and Hemodynamic Considerations

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SUMMARY
Six years have passed since the first successful surgical correction of truncus arteriosus. A review of our experience enables some conclusions regarding the operation. Patients with mild or moderate truncal valve incompetence do not need truncal valve replacement. Patients with severe truncal valve incompetence require valve replacement, which is associated with a significantly increased surgical mortality. The surgical mortality is not increased in hemodynamically favorable patients who have only one pulmonary artery. However, these patients are especially likely to have early development of severe pulmonary vascular disease. The surgical mortality for the patient with uncomplicated disease and two pulmonary arteries, with pulmonary resistance of less than 8.0 units $\text{m}^2$, is 10%. In patients with pulmonary resistance between 8.0 and 12 units $\text{m}^2$, the mortality is approximately three times greater. Patients with pulmonary resistances greater than 12.0 units $\text{m}^2$ are probably inoperable. Different hemodynamic criteria must be applied in assessing the operability of patients with a single pulmonary artery. A systemic arterial oxygen saturation less than 85% in a patient with two pulmonary arteries and without pulmonary artery stenosis or a pulmonary artery band usually indicates inoperability. Elective operation usually is deferred until a patient is four years old, but if the patient's clinical condition warrants, the procedure can be done at any time after the age of one year, with a good chance of success. Follow-up on most operated patients has been encouraging.

Additional Indexing Words:
- Absent pulmonary artery
- Pulmonary artery banding
- Truncal valve incompetence
- Rastelli operation
- Hypertensive pulmonary vascular disease
- Aortic arch interruption
- Systemic arterial oxygen saturation

Truncus arteriosus is an uncommon, but not rare, congenital cardiovascular defect. In one series of 685 autopsy-proved cases of congenital heart disease, 27 (3.9%) had some form of truncus arteriosus. The embryologic explanation of the deformity remains controversial although explanations have been offered. The defect is characterized by a single, large arterial trunk originating from the base of the heart. There is always a large ventricular septal defect immediately subjacent to the truncus. Collett and Edwards have classified truncus arteriosus according to the sites of origin of the pulmonary arteries arising from the single arterial trunk. This classification remains clinically useful although patients with Collett and Edwards type IV truncus arteriosus are probably more correctly grouped embryologically with patients having pulmonary atresia.

In September 1967, a case of truncus arteriosus was successfully repaired surgically at the Mayo Clinic. This operation established continuity between the right ventricle and the pulmonary arteries by the use of a specially prepared homograft of ascending aorta, including the aortic valve and anterior leaflet of the mitral valve. Between September 1967 and March 1972, 40 patients with type I or II truncus arteriosus were catheterized and subsequently underwent this operation at our institution.
CORRECTION OF TRUNCUS ARTERIOSUS

We wish to report the anatomic and hemo-
dynamic considerations that we believe are of
importance in the proper selection of patients with
truncus arteriosus for surgical correction, drawing
on the data and experience accumulated during the
six years since the inception of this procedure.

Material and Methods

This report is concerned exclusively with types I and
II truncus arteriosus as defined by Collett and Edwards,4
conditions in which a single pulmonary artery arises or
two pulmonary arteries arise from the truncus a short
distance above the truncal valve. Between August 1967
and March 1972, 70 patients with type I or II truncus
were catheterized at the Mayo Clinic to assess their
suitability for complete repair. The ages of the patients
(41 males and 29 females) ranged from 13 months to
18 years. Large film biplane angiography was done on
all patients, many having had both a right ventricular
and a truncal root injection. All patients had at least
one series of oxygen saturation data obtained while
breathing room air or a mixture of nitrogen oxide and
oxygen, with a P'O2 of between 170 and 200 mm Hg.
Systemic arterial blood samples were obtained through
ea needle placed in the femoral artery or, in a few
patients, the brachial arterial. In every patient, the
pulmonary artery or arteries were entered with a
catheter. Calculations of pulmonary flow and resistance
were made in the usual manner.6,7

Forty of the 70 catheterized patients subsequently
underwent repair. There were 12 operative deaths, and
an autopsy was performed in each of these. Examination
of the heart and lungs, including histologic grading of
the severity of pulmonary vascular disease using the
Heath-Edwards criteria,8 was performed by one
cardiovascular pathologist.

Intraoperative, postoperative pressure measurements
were obtained on 28 of the 40 operated patients after
they had stabilized following discontinuation of cardio-
pulmonary bypass.

Results

The incidence of associated cardiovascular abnor-
malities in these patients is shown in table 1. Lesions such as atrial septal defect and partial
anomalous pulmonary venous connection can be
dealt with at the time of repair and do not
apparently add to the risk of operation.

Table 1

<table>
<thead>
<tr>
<th>Defect</th>
<th>Patients Operated on</th>
<th>Surgical deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atrial septal defect</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Partial anomalous pulmonary</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>venous connection</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Interrupted aortic arch</td>
<td>4</td>
<td>3</td>
</tr>
<tr>
<td>Truncal valve insufficiency</td>
<td>25</td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>14</td>
<td>11</td>
</tr>
<tr>
<td>Moderate</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Severe</td>
<td>4</td>
<td>3†</td>
</tr>
<tr>
<td>Unilateral absence of</td>
<td>11</td>
<td>3</td>
</tr>
<tr>
<td>pulmonary artery</td>
<td></td>
<td>33</td>
</tr>
<tr>
<td>Previous pulmonary artery</td>
<td>7</td>
<td>4</td>
</tr>
<tr>
<td>banding</td>
<td></td>
<td>1*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>25</td>
</tr>
</tbody>
</table>

*Same patient.
†Valve replacement.

Anastomosis to the homograft, leaving the ductus
to function as the arch.9 Aortic arch interruption
of this type seems not to add to the risk of operation.

One of the three patients who underwent operation
was a surgical death, but death was attributed to
technical problems encountered secondary to a
previous bilateral pulmonary artery banding which
this patient had undergone during infancy. At re-
pair, both bands were located far peripherally near
the hila of the lungs. Removal of the bands with
subsequent anastomosis of the homograft to the
pulmonary arteries was technically very difficult,
and postoperative bleeding from these distal
anastomoses probably was the major contributing
factor in this patient’s death.

Truncal Valve Insufficiency

Twenty-five of the 70 patients (36%) had a
blowing diastolic murmur of truncal valve incom-
petence heard during clinical evaluation. At cat-
theterization, all 25 had truncal root angiocardiog-
raphy performed, and an attempt was made to
grade the regurgitation as mild, moderate, or
severe.

Fourteen patients had mild regurgitation, and 11
of these subsequently underwent operation. All 11
survived and have continued to do well, although
they still have mild incompetence of the semilunar
valve. Seven patients were judged to have moderate
detruncal valve insufficiency. Five underwent oper-
ation and two died; the three survivors continue
to do well. In retrospect, one of the patients who died
had severe truncal valve insufficiency, which was a
significant factor in his death. Three patients had

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Repair of truncus arteriosus with interruption of aortic arch. (A) Excision of pulmonary arteries from posterior aspect of truncus. (B) Repair of resulting defect in posterior wall of truncus, leaving ductus (now functioning as a substitute aortic arch) in continuity with truncus. (C) Orifice in pulmonary artery to which aortic homograft is anastomosed to establish right ventricular to pulmonary artery continuity. (Reprinted with permission from Gomes MMR, McGoon DC.9)

their trunal valve replaced with a Starr-Edwards prosthesis at the time of repair. Two of these patients were surgical deaths. The third patient survived the immediate postoperative period but died at home 2½ months later. Postmortem examination in this case revealed dehiscence of the prosthetic valve, and the resulting severe perivalvar insufficiency was probably the major factor contributing to her death. Severe trunal valve incompetence, which necessitates valve replacement, adds to the risk of operation although we believe it does not necessarily preclude a successful surgical result.

Unilateral Absence of a Pulmonary Artery

Eleven of the 70 patients (16%) had unilateral absence of a pulmonary artery. Seven had an absent right pulmonary artery and four an absent left pulmonary artery. Three patients were surgically repaired and one died. Unilateral absence of a pulmonary artery probably does not significantly increase the surgical risk if the patient is hemodynamically operable.

Previous Pulmonary Artery Banding

Seven patients had undergone previous pulmonary artery banding. Their ages at the time of banding ranged from 6 weeks to 15 months. Five of these patients, all of whom had two pulmonary arteries, were hemodynamically operable from 4 to 8 years after banding. Four have undergone operation, and one is presently awaiting surgery. Of the four who underwent operation, three survived surgery and have continued to do well. One patient was a surgical death (the previously mentioned patient with peripherally located bands who also had interruption of the aortic arch). Two patients, both of whom had unilateral absence of a pulmonary artery, had undergone banding of their single pulmonary artery at the ages of 6 weeks and 4 months, respectively. At catheterization, both...
patients were inoperable because of severe pulmonary vascular disease in the lung supplied by the pulmonary artery, despite their early banding.

**Pulmonary Vascular Resistance**

Accurate assessment of pulmonary vascular resistance is vital in the proper selection of truncus patients for operation. In our group of patients with two pulmonary arteries, 22 patients had pulmonary resistances of less than 8.0 units m\(^2\) (table 2). There were four surgical deaths in this group, an overall surgical mortality of 18%. Postmortem histologic examination of the lungs in these four patients revealed grade I Heath-Edwards changes in the pulmonary arterioles of two patients and grade II changes in the two others, confirming the preoperative assessment of mild to moderate pulmonary vascular disease. Two of the surgical deaths in this group had complicating features. One was the previously mentioned patient who had peripherally placed pulmonary artery bands, and the other patient had severe truncal valve insufficiency, necessitating valve replacement. If these two patients are excluded from the group, there remain 20 cases with two deaths, a surgical mortality rate of 10%.

Thirteen surgical patients had pulmonary resistances ranging between 8.0 and 12.0 units m\(^2\). There were five deaths in this group, an overall surgical mortality rate of 38%. Postmortem histologic examination of the lungs in these patients revealed that all five patients had grade III Heath-Edwards changes. One patient in this group had severe truncal valve insufficiency, necessitating valve replacement. If this patient is not included, there remain 12 patients, of whom four died, a surgical mortality rate of 33%.

Two patients with pulmonary resistances greater than 12.0 units m\(^2\) underwent operation, and both died. At autopsy, one patient had grade III Heath-Edwards changes and the other had grade IV changes.

After correction, the ratios of the peak systolic pressures in the pulmonary artery and the aorta were measured at surgery in 28 of the 40 patients and are shown in table 3. The mean ratio of 15 patients with preoperative pulmonary resistances less than 8 units m\(^2\) was 0.4, and only two (13%) had ratios greater than 0.5. In nine patients with pulmonary resistances between 8.0 and 12.0 units m\(^2\), the mean ratio was 0.7, and seven of these (78%) had ratios greater than 0.5. The level of pulmonary resistance, as estimated from these postoperative measurements, therefore showed good correlation with the preoperative calculated resistances.

Different hemodynamic criteria must be used to assess operability of patients with unilateral absence of a pulmonary artery, if calculations of pulmonary flow and resistance are made in the usual manner and an attempt is made to correlate these parameters with the degree of histologic pulmonary vascular obstructive disease. For a given degree of anatomic pulmonary vascular disease, the

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**Table 2**

*Surgical Mortality Rate Related to Pulmonary Resistance in 40 Patients*

<table>
<thead>
<tr>
<th>Rp, units m(^2)</th>
<th>Patients operated on</th>
<th>Overall</th>
<th>Excluding &quot;complicated cases&quot;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Surgical deaths</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Health-Edwards grade</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(pulmonary vascular disease)</td>
</tr>
<tr>
<td>&lt;8.0</td>
<td>22</td>
<td>4(^*)/22 (18%)</td>
<td>2/20 (10%)</td>
</tr>
<tr>
<td>8.0–12.0</td>
<td>13</td>
<td>5(^*)/13 (38%)</td>
<td>4/12 (33%)</td>
</tr>
<tr>
<td>&gt;12.0</td>
<td>2</td>
<td>2/2 (100%)</td>
<td>2/2 (100%)</td>
</tr>
<tr>
<td>12.3, 15.0, 17.1</td>
<td>3</td>
<td>1/3 (33%)</td>
<td>1/3 (33%)</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>12/40 (30%)</td>
<td>9/37 (24%)</td>
</tr>
</tbody>
</table>

Complicated cases: *one patient, peripheral pulmonary artery banding, and one patient, truncal valve replacement; †one patient, truncal valve replacement.

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**Table 3**

*Pressure Measurements Related to Pulmonary Resistance in 28 Patients from Whom Intraoperative Postrepair Measurements Were Obtained*

<table>
<thead>
<tr>
<th>Rp, units m(^2)</th>
<th>No. of patients</th>
<th>Ratio of systolic pressures (distal pulmonary artery to systemic)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Two pulmonary arteries (26 patients)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;8.0</td>
<td>15</td>
<td>0.2–0.6</td>
</tr>
<tr>
<td>8.0–12.0</td>
<td>9</td>
<td>0.4–1.0</td>
</tr>
<tr>
<td>&gt;12.0</td>
<td>2</td>
<td>0.8–1.0</td>
</tr>
<tr>
<td>Single pulmonary artery (2 patients)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15.0, 17.1</td>
<td>2</td>
<td>0.6–0.7</td>
</tr>
</tbody>
</table>
patient with only one pulmonary arteriolar bed exposed to systemic driving pressure would be expected to have approximately one-half the pulmonary flow and hence twice the calculated pulmonary resistance of the patient with comparable pulmonary vascular disease but two pulmonary arteries. Because of the presence of bronchial supply to the lung with absent pulmonary artery, this is obviously an oversimplification, but clearly different hemodynamic criteria for operability seem necessary. In our series, three patients with a single pulmonary artery underwent operation and one died, a mortality rate of 33% (table 2). The patient who died had a calculated pulmonary resistance of 12.3 units m², and histologic examination of the lungs revealed grade I Heath-Edwards changes in the pulmonary arterioles of the lung supplied by the pulmonary artery. Grade I changes were found at autopsy in only two of our cases in which two pulmonary arteries were present; both of these patients had pulmonary resistances of less than 8.0 units m². One can reason that if the patient had had two pulmonary arteries he would have been perfusing, at systemic driving pressure, two pulmonary arteriolar beds rather than just one. If both beds had the same degree of anatomic pulmonary vascular disease as found in the lung supplied by the single pulmonary artery, he would have had approximately twice the pulmonary flow and one-half the calculated pulmonary resistance. If the calculated pulmonary resistance of 12.3 units m² in this patient is halved, it falls into the group in which the finding of grade I changes might be anticipated. One of the two survivors of the group with a single pulmonary artery is doing well 2½ years after operation. However, the other survivor has had progression of her pulmonary vascular disease. If a pulmonary resistance of 12.0 units m² is considered the dividing line between operability and inoperability in patients with two pulmonary arteries, only 23 of 59 (39%) of these patients were inoperable at the time of study (table 4). For the patient with truncus arteriosus, unilateral absence of a pulmonary artery with a secondary obligatory increase in flow at high pressure through the other lung may lead to development of significant pulmonary vascular disease in the lung supplied by the pulmonary artery. Work by Vogel et al. and Rudolph et al. has suggested that ligation of one pulmonary artery shortly after birth, with secondary obligatory increase in flow through the other lung, may predispose to changes in the pulmonary arteriolar structure in that lung.

**Table 4**

Incidence of Severe Pulmonary Vascular Disease in 59 Patients With Two Pulmonary Arteries and 11 Patients With Single Pulmonary Artery

<table>
<thead>
<tr>
<th>Two pulmonary arteries (59 patients)</th>
<th>Rp &lt;12.0 units m²</th>
<th>Rp &gt;12.0 units m²</th>
<th>Inoperable</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>36</td>
<td>23</td>
<td>23/59 (39%)</td>
</tr>
<tr>
<td>Age range, yr</td>
<td>1-18</td>
<td>1-18</td>
<td></td>
</tr>
<tr>
<td>Mean age, yr</td>
<td>6</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Single pulmonary artery (11 patients)</th>
<th>Rp &lt;20.0 units m²</th>
<th>Rp &gt;20.0 units m²</th>
<th>Inoperable</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>3</td>
<td>8</td>
<td>8/11 (73%)</td>
</tr>
<tr>
<td>Age range, yr</td>
<td>7-14</td>
<td>3-12</td>
<td></td>
</tr>
<tr>
<td>Mean age, yr</td>
<td>11</td>
<td>6</td>
<td></td>
</tr>
</tbody>
</table>

Age at Operation

The ages at operation and the number of surgical deaths in the various age groups are shown in table 5. It is our present policy to defer operation until the age of four or five years if the patient is doing well clinically. If he is not doing well on medical management or if there is evidence of increasing

**Table 5**

Surgical Mortality Rate Related to Age in 40 Patients

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>Patients operated on</th>
<th>Surgical deaths</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>2</td>
<td>1</td>
<td>50</td>
</tr>
<tr>
<td>2-4</td>
<td>7</td>
<td>3</td>
<td>43</td>
</tr>
<tr>
<td>4-8</td>
<td>18</td>
<td>5</td>
<td>28</td>
</tr>
<tr>
<td>8-12</td>
<td>6</td>
<td>2</td>
<td>33</td>
</tr>
<tr>
<td>≥12</td>
<td>7</td>
<td>1</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>40</td>
<td>12</td>
<td>30</td>
</tr>
</tbody>
</table>
pulmonary vascular disease, the operation is offered earlier.

**Relationship of Systemic Arterial Saturation to Pulmonary Resistance**

In the patient with truncus arteriosus, as pulmonary blood flow decreases secondary to increasing pulmonary vascular disease, the systemic arterial oxygen saturation decreases. Systemic arterial saturation, therefore, reflects in part the status of the pulmonary vascular bed. Table 6 shows the systemic arterial saturation, with the patients at rest and breathing room air, of our 47 patients with two pulmonary arteries who had neither a pulmonary artery band nor naturally occurring pulmonary artery stenosis—that is, they had systemic levels of driving pressure in two pulmonary arteries. The data suggest that the hemodynamically most favorable patients, those with pulmonary resistances of less than 8.0 units m², will have a systemic arterial saturation of 88% or greater. Conversely, a systemic saturation less than 88% in a patient with two pulmonary arteries and without a pulmonary artery band or pulmonary artery stenosis probably means the patient is inoperable. Since this series of patients was compiled, we have had a patient who achieved a systemic saturation of 87% despite a pulmonary resistance of 12.8 units m². This patient had very favorable streaming of blood, with a systemic arterial saturation of 87% when saturation in both pulmonary arteries was 80%. Most patients with systemic saturations between 85 and 88% will be operable, although they may fall into the relatively higher risk group with pulmonary resistances between 8.0 and 12.0 units m². However, as our recent patient demonstrated, an occasional patient with a saturation greater than 85% may have a pulmonary resistance greater than 12.0 units m², attaining this saturation by virtue of very favorable streaming of blood rather than secondary to a relatively large pulmonary blood flow.

**Table 6**

| Systemic Arterial Oxygen Saturation (Breathing Room Air) Related to Pulmonary Resistance in 47 Patients* With Two Pulmonary Arteries |
|---|---|---|
| Rp, units m² | No. of patients | Systemic arterial saturation, % |
| | | Range | Mean |
| < 8.0 | 12 | 88–94 | 91 |
| 8.0–12.0 | 13 | 85–89 | 88 |
| >12.0 | 22 | 61–85 | 79 |

*Excluding 12 patients with pulmonary artery banding or stenosis.

**Discussion**

Although there are several case reports of patients with truncus arteriosus surviving to middle age without surgery, certainly the natural history of this condition usually runs a much shorter course. The initial results of operation for complete correction of truncus arteriosus have been encouraging. Although follow-up beyond six years is not yet available in corrected patients, we believe that the initial results justify recommendation of com-
complete repair to patients who are anatomically and hemodynamically favorable.

The risk of surgical correction depends on a number of factors. Patients with severe truncal valve incompetence are clearly a relatively high-risk group. In our series, these patients exhibited massive cardiomegaly before operation and had been in severe congestive heart failure for some time, factors which probably contributed more to the increased surgical risk than did the actual additional technical considerations involved in valve replacement. Relatively early repair, while there is still a reasonably good cardiac reserve, is no doubt desirable in such patients.

Previous pulmonary artery banding, provided the bands are not located far peripherally, does not appear to add to the risk of operation, although a previous thoracotomy inevitably makes surgical dissection more tedious. Our experience indicates, however, that banding does not always protect against the development of, or allow regression of, severe pulmonary vascular obstructive disease. This is particularly true in the especially susceptible group of patients having a single pulmonary artery.

With advancing degrees of pulmonary vascular disease, the operative mortality naturally increases. It is clearly not realistic to establish a single specific value of pulmonary resistance below which patients are operable and above which they are not. However, in our group of patients with two pulmonary arteries, patients with a pulmonary resistance greater than 8.0 units m\(^2\) were at substantially greater risk than those with values below this level, and the only two operated patients with pulmonary resistances greater than 12.0 units m\(^2\) both died.

We advise follow-up with periodic arterial blood samples in patients with truncus arteriosus who are less than four years old. If the arterial saturation shows a significant decrease, particularly if it has fallen below 88%, prompt recatheterization is indicated, with consideration toward early surgical correction if there has been a significant increase in pulmonary resistance.

A significant problem area is the management of the very young infant with this lesion who is in severe congestive heart failure that does not respond to medical therapy. Pulmonary arterial banding may provide effective palliation if the failure is secondary only to a large left-to-right shunt.\(^{15, 16}\) However, banding is not without significant operative risk and does not always prevent the subsequent development of significant pulmonary vascular disease. Moreover, as Gelband, Van Meter and Gersony\(^{17}\) have pointed out, a significant percentage of these young infants in refractive heart failure have severe truncal valve incompetence, in addition to the large shunt, and these patients are not benefitted by banding. The management of such infants is, at this time, unsatisfactory.

All surgically corrected patients in this report had continuity established between the right ventricle and the pulmonary artery by use of a conduit of homograft aorta and aortic valve. Although the early results were encouraging, we are now correcting a series of patients by using a prosthetic Dacron conduit that contains a porcine aortic valve (Hancock conduit). This change was made because of the nearly invariable calcification of the homograft, the need to replace the homograft in two patients\(^{12}\) (both had anomalies other than truncus) because of progressive cardiac failure related to stenosis of the graft, and the tendency of the homograft to bend at the level of the valve, thus distorting it. The most satisfactory type of conduit to use in establishing right ventricle to pulmonary artery continuity in complete surgical correction of truncus arteriosus has not yet been determined. However, the operation itself now seems well established as the treatment of choice in this condition.

Acknowledgment

Dr. J. L. Titus performed the histologic grading of the pulmonary arteriolar disease in the autopsy cases.

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