Ischaemic stroke among young people aged 15 to 50 years in Adelaide, South Australia

Sroke can have disastrous con-
sequences for the individual
and society; it is Australia’s
second leading cause of death.1 The
national lifetime cost for all first-ever
strokes occurring in Australia in 1997
was estimated to be $1.3 billion.2
Strokes among young people account
for 18% of all strokes in Australia and
are particularly damaging, as patients
are often unable to resume independ-
ent living or return to the workforce.1

Population-specific studies are
essential to our understanding of
stroke risk, as considerable differences
in risk factors and aetiologies can exist
across different countries and ethnic
groups. To date, aetiological studies of
ischaemic stroke among young people
have been reported from several
countries in Europe, the Americas and
Asia3-17 but, to our knowledge, none
have been reported for the Australian
population. Furthermore, only a
minority of studies elsewhere have
presented detailed information regarding less well documented stroke risk factors and neuroimaging
features.3,7,17

The purpose of this study was to
determine risk factors, aetiologies,
and neuroimaging features among
young Australian people who have had
a first-ever ischaemic stroke, and
to evaluate these with regard to age,
sex and ethnicity. We aimed to be able
to compare our findings with those
for other countries.

Methods

South Australia is a state of 1.6 million
people. Adelaide, the largest city and
capital, has a population of 1.2 million
people and is the only major referral
centre for stroke in the state. Our
retrospective study included all seven
public hospitals in the greater
Adelaide area. Formal ethics approval
was sought and obtained from each
hospital ethics board.

The public hospital coding system
was searched for all patients aged 15
to 50 years with a primary diagnosis of
any form of stroke who were admitted
to a public hospital in Adelaide from
January 2006 to June 2010. We
reviewed discharge summaries, neu-
roimaging results and medical records
retrospectively and included only
patients with first-ever ischaemic
stroke with clinical symptoms.

We defined ischaemic stroke in
accordance with the Baltimore–Wash-
ington Cooperative Young Stroke
Study criteria as an acute onset of
focal neurological deficit lasting > 24
hours, or < 24 hours with correspond-
ing imaging evidence.18 Transient
ischaemic attack (TIA) was defined as
acute onset of focal neurological defi-
cit lasting < 24 hours, with normal
imaging. Patients were excluded if
they had TIA, cerebral venous throm-
bus, iatrogenic stroke secondary to a
procedure or operation, haemorrhage,
head or neck trauma, or a diagnosis
other than stroke.

Risk factors were investigated in
accordance with the 2006 guidelines
from the American Stroke Associa-
tion.19 We matched our risk factor
definitions closely with those of the
largest and most comprehensive
study of young people with ischaemic
stroke to date, the Helsinki Young
Stroke Registry Study, which allowed
us to make valid comparisons.3

Stroke aetiology was categorised by
subtype according to the Trial of Org
10172 in Acute Stroke Treatment
(TOAST) criteria.20 Subtypes were
assigned by two independent stroke
neurologists, with the intervention of
a third stroke neurologist in the case
of a split decision. To allow compari-
son with previous published studies,
both patent foramen ovale (PFO),
with or without atrial septal aneurysm
(ASA), and blood clotting abnormali-
ties were classified as stroke aetio-
lologies if the clinical setting was
consistent and no other source was
identified.

An independent neuroradiologist
who was blinded to the original
diagnosis classified the vascular
territory involved and the stroke location.
Vascular territory was classified as ante-
rior circulation, posterior circulation,
or both. Stroke location was lateral-
ised and classified as cerebral hemisphere, cerebellum, brainstem, or involving multiple sites.

**Statistical analysis**

Categorical variables were compared using the Pearson χ² test (P < 0.05 significant). Significant differences between categorical variables for different ethnic subgroups were confirmed using the Fisher exact test.

**Results**

Of 666 patients admitted to one of the public hospitals in Adelaide with a primary diagnosis of any form of stroke from January 2006 to June 2010, 326 patients were classified as having had a first-ever occurrence of ischaemic stroke with clinical symptoms.

All patients had had a history taken and had undergone physical examination. Brain imaging had been performed on 325 patients (nearly 100%), vascular imaging of the neck on 299 patients (92%), and cardiac imaging on 252 patients (77%) (Box 1).

The mean age of patients in the study population was 40.7 years (SD, 7.9 years). To assess age-related trends, we divided the population into two groups of equal sizes: a younger cohort aged 15–42 years (162 patients) and an older cohort aged 43–50 years (164 patients). Overall, 56% of patients (184) were male (P = 0.02). The ratio of male to female patients was not significantly different in the younger cohort (P = 0.7), but in the older cohort 61% of patients (100) were men (P = 0.005).

People of European ancestry formed the largest ethnic subgroup (289; 89%), and Australian Aboriginals the next largest (19%; 6%).

Overall, the most frequent stroke risk factors were dyslipidaemia (57%), smoking (49%) and hypertension (32%) (Box 2). Obesity (28%) and illicit drug use (16%) were the fourth and eighth most prevalent stroke risk factors, respectively. The most commonly used illicit drug was marijuana (36 patients; 71% of illicit drug users), followed by amphetamines (19; 37%). Of amphetamine users, 17 (89%) administered the drug intravenously. Illicit drug use (P = 0.03), PFO (P < 0.001), and use of the oral contraceptive pill (OCP; P = 0.005) were more prevalent in the younger cohort, whereas hypertension (P = 0.006) and diabetes (P = 0.02) were more prevalent in the older cohort. More males were heavy alcohol drinkers (P < 0.001), and females had more migraines (P < 0.001). Cardiovascular disease (P = 0.01) and diabetes (P < 0.001) were more common among Australian Aboriginals compared with patients of European ancestry.

The most frequently identified stroke aetiology among our study population was cardioembolism (26%) (Box 3). Arterial dissection, a subtype of the “other aetiology” category, was identified in 15% of patients (49); and small-vessel occlusion was the aetiology in 10% (Box 3). Small vessel occlusion was more prevalent in male patients (P = 0.02). The most common aetiologies among the 85 patients with cardioembolic stroke were PFO (30), PFO plus ASA (11), infective endocarditis (7), left ventricle thrombus (6) and hypokinetic left ventricle segment (5). Intravenous amphetamine misuse was more common among patients with infective endocarditis (2/7; 29%) compared with the rest of the study population (15/319; 5%) (P = 0.04).

Strokes more frequently involved the anterior circulation compared with the posterior circulation (57% v 28%, respectively; P < 0.001) (Box 4). Strokes concurrently involving both vascular territories were observed in 9% of patients; intravenous amphetamine misuse was more common among patients with this type of stroke (7/29; 24%) compared with the whole study population (P < 0.001).

**Discussion**

To our knowledge, our study is the first specific analysis of a young population of patients with a first-ever ischaemic stroke in Australia. Our data showed some similarities between the population of patients from Adelaide and comparable groups of patients in other countries, but also some significant differences.

Dyslipidaemia, smoking and hypertension were the most frequent stroke risk factors among patients in our study, consistent with results of previous studies of ischaemic stroke among young people of similar age.3–6

Obesity and misuse of marijuana and amphetamines were more prevalent in patients from Adelaide compared with those from other countries. Of our study population, 28% were obese, compared with 11% and 5% among patients in the Finnish and Italian young stroke registry studies, respectively (P < 0.001 for each).6,6 Sixteen per cent of our study group reported use of illicit drugs, compared with 1% of patients in the Finnish study (P < 0.001).3 Most previous studies of young patients with ischaemic stroke have reported low rates of marijuana and amphetamine use, and that cocaine was the most commonly used illicit drug.21 Among our study population, marijuana and amphetamines were the most frequently used illicit drugs, and only one person used cocaine. Marijuana is the illicit drug most often used in South Australia; 12.5% of South Australians aged 14 years and over have used it at least once.22 Evidence supporting marijuana as a stroke risk factor is increasing. Recent studies have shown marijuana users to have increased levels of apolipoprotein C-III, a known cardiovascular risk factor, as well as an adjusted odds ratio of 1.76 for ischaemic stroke.3,24 Amphetamines increase the risk of stroke by several mechanisms, including elevation of blood pressure, vasculitis and cerebral vasospasm,25 and are frequently mis-
### 2 Stroke risk factors among patients admitted to public hospitals in South Australia with a primary diagnosis of first-ever ischaemic stroke, Jan 2006 – Jun 2010, by age, sex and ethnicity (n = 326)

<table>
<thead>
<tr>
<th>Age in years (no. [%])</th>
<th>Sex (no. [%])</th>
<th>Ethnicity (no. [%])</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male (n = 184)</td>
<td>Female (n = 142)</td>
</tr>
<tr>
<td>Dyslipidaemia</td>
<td>187 (57.4%)</td>
<td>100 (61.0%)</td>
</tr>
<tr>
<td>Smoking</td>
<td>161 (49.4%)</td>
<td>86 (52.4%)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>105 (32.8%)</td>
<td>67 (40.9%)</td>
</tr>
<tr>
<td>Obesity</td>
<td>92 (28.2%)</td>
<td>53 (32.3%)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>63 (19.3%)</td>
<td>36 (22.0%)</td>
</tr>
<tr>
<td>Migraine</td>
<td>59 (18.1%)</td>
<td>24 (14.6%)</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>53 (16.3%)</td>
<td>32 (19.5%)</td>
</tr>
<tr>
<td>Illicit drugs</td>
<td>51 (15.6%)</td>
<td>18 (11.0%)</td>
</tr>
<tr>
<td>Patien foramen ovale</td>
<td>49 (15.0%)</td>
<td>12 (7.3%)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>44 (13.5%)</td>
<td>30 (18.3%)</td>
</tr>
<tr>
<td>Transient ischaemic attack</td>
<td>35 (10.7%)</td>
<td>24 (14.6%)</td>
</tr>
<tr>
<td>Family history</td>
<td>32 (9.8%)</td>
<td>20 (12.2%)</td>
</tr>
<tr>
<td>Acute infection</td>
<td>29 (8.9%)</td>
<td>14 (8.5%)</td>
</tr>
<tr>
<td>Oral contraceptive pill</td>
<td>29 (8.9%)</td>
<td>7 (4.3%)</td>
</tr>
<tr>
<td>Obstructive/central sleep apnoea</td>
<td>18 (5.5%)</td>
<td>12 (7.3%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>15 (4.6%)</td>
<td>10 (6.1%)</td>
</tr>
<tr>
<td>Factor V Leiden mutation</td>
<td>15 (4.6%)</td>
<td>8 (4.9%)</td>
</tr>
<tr>
<td>Lupus anticoagulant</td>
<td>13 (4.0%)</td>
<td>6 (3.7%)</td>
</tr>
<tr>
<td>Anticardiolipin antibody</td>
<td>13 (4.0%)</td>
<td>4 (2.4%)</td>
</tr>
<tr>
<td>High homocysteine</td>
<td>13 (4.0%)</td>
<td>6 (3.7%)</td>
</tr>
<tr>
<td>Atrial septal aneurysm</td>
<td>12 (3.7%)</td>
<td>5 (3.0%)</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>9 (2.8%)</td>
<td>6 (3.7%)</td>
</tr>
<tr>
<td>Active malignancy</td>
<td>7 (2.1%)</td>
<td>4 (2.4%)</td>
</tr>
<tr>
<td>Prothrombin mutation</td>
<td>6 (1.8%)</td>
<td>1 (0.6%)</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>4 (1.2%)</td>
<td>2 (1.2%)</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>3 (0.9%)</td>
<td>1 (0.6%)</td>
</tr>
<tr>
<td>Antithrombin III deficiency</td>
<td>3 (0.9%)</td>
<td>1 (0.6%)</td>
</tr>
<tr>
<td>Hormone replacement therapy</td>
<td>1 (0.3%)</td>
<td>0</td>
</tr>
</tbody>
</table>

na = not applicable.

**Definitions for stroke risk factors:**
- Dyslipidaemia: Total cholesterol, > 5.0 mmol/L; low-density lipoprotein cholesterol, > 3.0 mmol/L; or high-density lipoprotein cholesterol, < 1.0 mmol/L.
- Smoking: Smoked =1 cigarette daily during the previous year.
- Hypertension: On antihypertensive therapy, or clearly stated.
- Obesity: Body mass index, > 30 or clearly stated as obese.
- Alcohol: Weekly intake of > 200 g of alcohol.
- Migraine: Clearly stated.
- Cardiovascular disease: Ischaemic heart disease, heart failure (ejection fraction, < 55%), previous myocardial infarction, or peripheral vascular disease.
- Illicit drugs: Taken within the month before stroke occurred.
- Patien foramen ovale: As reported on echocardiogram.
- Diabetes: Fasting plasma glucose, > 7.0 mmol/L or clearly stated (type 1 or 2).
- Transient ischaemic attack: Acute onset of focal neurological deficit lasting < 24 hours, with normal imaging.
- Family history: Stroke in a first-degree relative.
- Acute infection: Confirmed at stroke onset.

These results are similar to results from previous studies. Comparisons by ethnic subgroup revealed a higher prevalence of cardiovascular disease and diabetes among patients who were Australian Aboriginals compared with those of European ancestry — the contrast was particularly striking for diabetes (47% vs 11%, respectively). Australian Aboriginals were over-represented in our study population, constituting 6% — more than double the proportion of Aboriginal and Torres Strait Islander people among the Australian population in 2006 (2.3%). Comparisons between ethnic subgroups should be interpreted with caution, as the absolute number of Australian Aboriginals included in our study was small. Further studies involving greater numbers of Australian Aboriginals from more areas of Australia are needed.

Cardioembolism, arterial dissection, and small-vessel occlusion were the most frequent stroke aetiologies in our study. The prevalence of cardioembolism was higher among the pop-
3 Stroke aetiologies among patients admitted to public hospitals in South Australia with a primary diagnosis of first-ever ischaemic stroke, Jan 2006 – Jun 2010, by age, sex and ethnicity

<table>
<thead>
<tr>
<th>Aetiology</th>
<th>Total (n = 326)</th>
<th>15–42 (n = 162)</th>
<th>43–50 (n = 164)</th>
<th>P</th>
<th>Male (n = 184)</th>
<th>Female (n = 142)</th>
<th>P</th>
<th>European ancestry (n = 289)</th>
<th>Australian Aboriginal (n = 19)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardioembolism</td>
<td>85 (26.1%)</td>
<td>50 (30.9%)</td>
<td>35 (21.3%)</td>
<td>0.09</td>
<td>50 (27.2%)</td>
<td>35 (24.6%)</td>
<td>0.7</td>
<td>70 (24.2%)</td>
<td>9 (47.4%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Large artery atherosclerosis</td>
<td>12 (3.7%)</td>
<td>4 (2.5%)</td>
<td>8 (4.9%)</td>
<td>0.3</td>
<td>8 (4.3%)</td>
<td>4 (2.8%)</td>
<td>0.5</td>
<td>11 (3.8%)</td>
<td>0</td>
<td>0.4</td>
</tr>
<tr>
<td>Small vessel occlusion</td>
<td>31 (9.5%)</td>
<td>12 (7.4%)</td>
<td>19 (11.6%)</td>
<td>0.2</td>
<td>24 (13.0%)</td>
<td>7 (4.9%)</td>
<td>0.02</td>
<td>31 (10.7%)</td>
<td>0</td>
<td>0.2</td>
</tr>
<tr>
<td>Other determined aetiology</td>
<td>95 (29.1%)</td>
<td>46 (28.4%)</td>
<td>49 (29.9%)</td>
<td>0.8</td>
<td>48 (26.1%)</td>
<td>47 (33.1%)</td>
<td>0.2</td>
<td>87 (30.1%)</td>
<td>1 (5.3%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Multiple possible aetiologies</td>
<td>25 (7.7%)</td>
<td>15 (9.3%)</td>
<td>10 (6.1%)</td>
<td>0.3</td>
<td>15 (8.2%)</td>
<td>10 (7.0%)</td>
<td>0.7</td>
<td>23 (8.0%)</td>
<td>2 (10.5%)</td>
<td>0.7</td>
</tr>
<tr>
<td>Undetermined aetiology, extensive evaluation</td>
<td>52 (16.0%)</td>
<td>25 (15.4%)</td>
<td>27 (16.5%)</td>
<td>0.8</td>
<td>25 (13.6%)</td>
<td>27 (19.0%)</td>
<td>0.2</td>
<td>44 (15.2%)</td>
<td>5 (26.3%)</td>
<td>0.2</td>
</tr>
<tr>
<td>Undetermined aetiology, incomplete evaluation</td>
<td>26 (8.0%)</td>
<td>10 (6.2%)</td>
<td>16 (9.8%)</td>
<td>0.3</td>
<td>14 (7.6%)</td>
<td>12 (8.5%)</td>
<td>0.8</td>
<td>23 (8.0%)</td>
<td>2 (10.5%)</td>
<td>0.7</td>
</tr>
</tbody>
</table>

* n = 308 (18 were other ethnicity).

4 Features seen on neuroimaging among patients admitted to public hospitals in South Australia with a primary diagnosis of first-ever ischaemic stroke, Jan 2006 – Jun 2010, by age, sex and ethnicity

<table>
<thead>
<tr>
<th>Location</th>
<th>Total (n = 326)</th>
<th>15–42 (n = 162)</th>
<th>43–50 (n = 164)</th>
<th>P</th>
<th>Male (n = 184)</th>
<th>Female (n = 142)</th>
<th>P</th>
<th>European ancestry (n = 289)</th>
<th>Australian Aboriginal (n = 19)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior circulation</td>
<td>185 (56.7%)</td>
<td>96 (59.3%)</td>
<td>89 (54.3%)</td>
<td>0.5</td>
<td>96 (52.2%)</td>
<td>89 (62.7%)</td>
<td>0.2</td>
<td>161 (55.7%)</td>
<td>14 (73.7%)</td>
<td>0.3</td>
</tr>
<tr>
<td>Posterior circulation</td>
<td>92 (28.2%)</td>
<td>44 (27.2%)</td>
<td>48 (29.3%)</td>
<td>0.7</td>
<td>61 (33.2%)</td>
<td>31 (21.8%)</td>
<td>0.06</td>
<td>86 (29.8%)</td>
<td>1 (5.3%)</td>
<td>0.05</td>
</tr>
<tr>
<td>Both</td>
<td>29 (8.9%)</td>
<td>14 (8.6%)</td>
<td>15 (9.1%)</td>
<td>0.9</td>
<td>16 (8.7%)</td>
<td>13 (9.2%)</td>
<td>0.9</td>
<td>23 (8.0%)</td>
<td>3 (15.8%)</td>
<td>0.3</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location</th>
<th>Total (n = 326)</th>
<th>15–42 (n = 162)</th>
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<th>Australian Aboriginal (n = 19)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right CH</td>
<td>141 (43.3%)</td>
<td>78 (48.1%)</td>
<td>63 (38.4%)</td>
<td>0.2</td>
<td>76 (41.3%)</td>
<td>65 (45.8%)</td>
<td>0.5</td>
<td>124 (42.9%)</td>
<td>8 (42.1%)</td>
<td>1</td>
</tr>
<tr>
<td>Left CH</td>
<td>161 (49.4%)</td>
<td>79 (48.8%)</td>
<td>82 (50.0%)</td>
<td>0.9</td>
<td>94 (51.1%)</td>
<td>67 (47.2%)</td>
<td>0.6</td>
<td>140 (48.4%)</td>
<td>12 (63.2%)</td>
<td>0.4</td>
</tr>
<tr>
<td>Right cerebellum</td>
<td>28 (8.6%)</td>
<td>11 (6.8%)</td>
<td>17 (10.4%)</td>
<td>0.3</td>
<td>19 (10.3%)</td>
<td>9 (6.3%)</td>
<td>0.2</td>
<td>24 (8.3%)</td>
<td>1 (5.3%)</td>
<td>0.7</td>
</tr>
<tr>
<td>Left cerebellum</td>
<td>29 (8.9%)</td>
<td>14 (8.6%)</td>
<td>15 (9.1%)</td>
<td>0.9</td>
<td>18 (9.8%)</td>
<td>11 (7.7%)</td>
<td>0.5</td>
<td>21 (7.3%)</td>
<td>2 (10.5%)</td>
<td>0.6</td>
</tr>
<tr>
<td>Right BS</td>
<td>19 (5.8%)</td>
<td>8 (4.9%)</td>
<td>11 (6.7%)</td>
<td>0.5</td>
<td>12 (6.5%)</td>
<td>7 (4.9%)</td>
<td>0.6</td>
<td>18 (6.2%)</td>
<td>1 (5.3%)</td>
<td>0.9</td>
</tr>
<tr>
<td>Left BS</td>
<td>23 (7.1%)</td>
<td>8 (4.9%)</td>
<td>15 (9.1%)</td>
<td>0.2</td>
<td>18 (9.8%)</td>
<td>5 (3.5%)</td>
<td>0.04</td>
<td>21 (7.3%)</td>
<td>1 (5.3%)</td>
<td>0.9</td>
</tr>
<tr>
<td>Multiple sites</td>
<td>61 (18.7%)</td>
<td>31 (19.1%)</td>
<td>30 (18.3%)</td>
<td>0.9</td>
<td>27 (14.7%)</td>
<td>34 (23.9%)</td>
<td>0.06</td>
<td>54 (18.7%)</td>
<td>3 (15.8%)</td>
<td>0.8</td>
</tr>
</tbody>
</table>

CH = cerebral hemisphere. BS = brainstem.

ulation in Adelaide (26%) than among patients in Finland (20%), Greece (13%), Spain (17%), Canada (14%), Indiana in the United States (14%), and Taiwan (18%), but not significantly different compared with Georgia, Italy, Sweden, Switzerland, Iowa in the US, Brazil, India and Korea.3-16 Infective endocarditis was more frequent among our study population than has been reported in most other studies.3,13,14 Since intravenous injection can trigger infective endocarditis, our higher observed rates of cardioembolic stroke might be partly explained by a high prevalence of intravenous amphetamine misuse.

The few studies that have reported ischaemic stroke topography among young adults have shown that stroke involving the anterior cerebral circulation are more frequent than those involving the posterior circulation, observations which are consistent with our results.3,7,17 However, more strokes in South Australia (9%) concurrently involved both the anterior and the posterior circulations compared with Finland (5%) and Sweden (3%) (P < 0.05 for each).3,7 Intravenous amphetamines have the potential to cause infarction in more than one vascular territory simultaneously, due to their pathophysiological effects. Such imaging findings in our study are thus probably partly explained by a high prevalence of intravenous amphetamine misuse among patients with this type of stroke.

There were several limitations to our study. First, it was retrospective in nature. It covered a period of several years over which imaging technology and diagnostic testing varied. It also involved several hospitals, so the approach to documentation and diagnostic testing was not standardised. The retrospective determination of risk factors is of particular concern in that the more atypical risk factors (such as illicit drug use) may have been underestimated, as they are not routine components of history-taking. Second, the Australian health care system comprises coexisting public and private sectors, and access to patient details in the private system is difficult. We were unable to include private patients in our study. However, the actual number of younger patients admitted to private hospitals with ischaemic stroke during our study period was low — roughly seven patients per year, before applying exclusion criteria (South Australian Department of Health, unpublished data, 2010), and it is therefore unlikely that these patients would have
affected our results significantly. Third, some younger patients with ischaemic stroke treated solely as outpatients may have been missed by our study. However, it is hard to imagine that there would be many patients in this group, as young patients with a first-ever ischaemic stroke with clinical symptoms would routinely be admitted to one of the tertiary hospitals included in our study for inpatient workup. Finally, since risk factor definitions vary from one study to the next, any comparison of risk factors across different studies must be interpreted with caution. Although we matched our risk factor definitions with those of the Helsinki study, any comparisons with other studies of ischaemic stroke in young people are subject to this limitation.


