Sleep apnoea is associated with increased cardiovascular risk. Sleep apnoea is common after stroke and associated with increased blood pressure variability as described by Turkington and co-workers in this issue of Clinical Science. Both sleep apnoea and blood pressure variability confer a poor prognosis after stroke and are potentially treatable. Many studies of CPAP (continuous positive airway pressure) demonstrate decreases in cardiovascular risk markers in other patient groups. Although difficult to apply in these patients in the short term, CPAP has some potential benefits in medium-term rehabilitation and secondary prevention following stroke, which warrants further study.

Stroke is common, with an incidence of 2–18/1000 per year, and is the most common cause for neurological admission to hospital, and accounts for a large burden of community disability [1]. Sleep disordered breathing has been recognized in stroke since the early 19th century [2], but recent studies suggest a very high prevalence (> 50 %) of sleep apnoea after stroke compared with control groups [3,4], although there may be no increased risk after TIA (transient ischaemic attack) [5]. Central, as opposed to obstructive, apnoea may be more common after infratentorial stroke [6], although both can occur in the same individual and neither predicts ischaemic or haemorrhagic subtypes nor neurological location [7].

The prevalence of sleep apnoea is highest early following stroke and may fall during recovery [8]. This reflects a decrease in sleep apnoea in most patients, but also hides a higher mortality in those with more severe sleep disordered breathing. In addition to increased mortality, sleep apnoea following stroke predicts worse stroke scores, cognitive deficits, disability, length of hospital stay and long-term functional outcome [9–12].

So, if stroke causes sleep apnoea, does sleep apnoea cause stroke? Since sleep apnoea is so common [13] it may, by chance, occur with stroke. However, work to date linking sleep apnoea and stroke includes only small numbers of subjects with sleep apnoea. These report relative risk for stroke of approx. 2–3 in self-reported habitual snorers (a weak surrogate for sleep apnoea) [14,15], and of approx. 5–8 in subjects with obstructive sleep apnoea.

Sleep apnoea may cause cardiovascular disease at various levels, for example, by causing known risk factors (hypertension or insulin resistance) or multiplying the effects of these known intermediary risk factors (through hypoxia or sympathetic activation), by initiating critical events (plaque rupture and thrombosis) or by increasing injury at the time of event or impairing subsequent repair processes (apnoea and hypoxia lower baroreflex sensitivity and increase sympathetic activation causing raised and variable blood pressure with platelet activation and endothelial damage extending thrombosis and ischaemic territory). Which of these precise mechanisms is involved is not yet clear, but recent studies at the cellular and molecular level have demonstrated many physiologically plausible mediators between sleep apnoea and vascular damage, including stroke [16]. Thus there are several mechanisms to explain the poorer prognosis in stroke patients with sleep apnoea, although other factors may also be important. Upper airway obstruction, for example, suggests bulbar or pseudobulbar dysfunction, and may precipitate aspiration of pharyngeal contents.

In this issue of Clinical Science, Turkington et al. [17] report increased blood pressure and blood pressure variability post stroke compared with controls, increased blood pressure variability in stroke with upper airways obstruction (respiratory disturbance index > 10/h) and strong positive association between upper airway obstruction and blood pressure variability. Both high blood pressure and variability are associated with poor outcome following stroke and this study [17] therefore suggests one intermediary between sleep apnoea and poor prognosis.

In the report by Turkington et al. [17], blood pressure variability is raised in all patients post stroke compared with controls. Blood pressure variability may be raised by altered sleep–wake cycles, arousals...
through noise or nursing procedures, or by central
dysautonomia and altered chemico- and baro-reflexes.
Despite all these potential confounders, the authors [17]
report a really quite close relationship between blood
pressure variability and sleep apnoea severity assessed by
respiratory disturbance index ($r = 0.642$ compared with
10 mmHg dips). This is similar to the blood pressure
fluctuations entailed in subjects with isolated sleep
apnoea. The authors [17] also demonstrate deeper and
longer falls in oxygen saturation during the studies in
patients with upper airway obstruction. Cerebral blood
flow autoregulation is disrupted following stroke and
perfusion pressure may fall during episodes of sleep
apnoea. It is mooted that the blood pressure dips
reported here [17] could extend areas of critical ischaemia,
especially in conjunction with apnoea-associated hypoxia
and hypercapnia.

Our main aims of stroke treatment are to reduce
mortality and disability. Recent advances have been made
with thrombolytic treatment and there is convincing
evidence for several drugs in primary and secondary
prevention. Conversely, tight blood pressure control
acutely following stroke and prevention of hypoxaemia
with oxygen therapy have been disappointing. Upper
airway obstruction presents a potential therapeutic target
to improve outcomes, as control of sleep apnoea may
prevent both hypoxaemia, large fluctuations in blood
pressure and cerebral hyperperfusion. There are some
theoretical benefits for protection of cerebral blood flow
following stroke by the slight rise in CO$_2$ which occurs
with hypoventilation. And concerns are expressed that supportive CPAP (continuous positive airway pressure)
treatment or non-invasive ventilation may lower CO$_2$
supportive CPAP (continuous positive airway pressure)
variability as described by Turkington et al. [17] in this
issue of Clinical Science. Both sleep apnoea and blood
pressure variability confer a poor prognosis after stroke
and are potentially treatable. Many studies of CPAP
demonstrate decreases in cardiovascular risk markers in
other patient groups. Although difficult to apply in these
patients in the short term, CPAP has some potential be-
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vention following stroke which warrants further study.

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