Respiratory Rate and Pattern Disturbances in Acute Brain Stem Infarction

MYOUNG C. LEE, M.D., ARTHUR C. KLASEN, M.D.,
LOIS M. HEANEY, R.N., AND JOSEPH A. RESCH, M.D.

ABNORMALITIES of respiratory rate and pattern may occur in patients with brain stem lesions. As previously described, these consist of Cheyne-Stokes respiration (CSR), Cheyne-Stokes variant pattern (CSV), cluster, apneustic and ataxic respirations and tachypnea or "central neurogenic hyperventilation" (CNH).2,3 The occurrence of these abnormalities may be related to the site and extent of the lesion2,3 as well as to prognosis.3 The mechanisms underlying these alterations of respiratory patterns are not well understood. Beyond sporadic case reports, information on the types and frequency of various respiratory pattern abnormalities in acute brain stem infarction is incomplete. The present study was undertaken to investigate the frequency of respiratory rate and pattern abnormalities in patients with acute brain stem infarction as well as the prognostic significance of the observed abnormalities.

Impedance pneumography has been used to detect respiratory rate and pattern abnormalities in patients with central nervous system lesions.1,4,5 This simple atraumatic technique accurately measures respiratory rate and pattern and, less reliably, the volume of respiration based on changes in transthoracic impedance.6,7

Methods

Twenty-three patients with acute brain stem infarction admitted to the University of Minnesota Neurological Intensive Care Unit within 72 hours after onset of symptoms were studied during the first two weeks of hospitalization (table 1). Diagnoses were made on the basis of clinical examination and laboratory evaluation; in six of eight patients who died, the diagnoses were confirmed by postmortem examinations. Only those patients with spontaneous respirations without assisted ventilation were included in the study. Using impedance pneumography, permanent records of respiratory rates and patterns were obtained with a chart recorder at approximately hourly intervals during the period of observation in the intensive care unit. This period of observation was varied in duration depending on the patient's clinical status. Arterial blood gas samples for pH, Pco2 and Po2 were obtained in all patients on the day of admission and occasionally thereafter. The frequency and type of respiratory pattern abnormalities were then related to the location of the lesions. The respiratory rate and pattern abnormalities were not quantitated, although the trends of the changes were noted in individual patients.

Figure 1 illustrates the types of respiratory rates and patterns observed in this group of patients. Periods of hyperpnea regularly alternating with periods of apnea were designated Cheyne-Stokes respiration (CSR). The term Cheyne-Stokes variant (CSV) was used to designate respiratory patterns in which phasic variations in depth of respiration without definite apneic periods occurred. Cluster respiration was used to refer to several breaths of varying depth interspersed with apneic episodes which were usually of irregular length. Rapid, regular respiratory rates greater than 30 per minute were designated as tachypnea. Other types of respiratory rate and pattern changes were not included because of difficulties in distinguishing them from artifactual changes using the impedance pneumography technique. As also observed in healthy subjects by others,8 occasional periods of very short duration of CSR and/or CSV were observed in all patients at some time during the period of observation, especially during sleep. These were not considered to be abnormal and these patients were thus categorized as having normal respiratory patterns. Patients with frequent or persistent abnormal respiratory patterns were classified on the basis of the abnormal respiratory pattern observed most frequently. Patients in whom both CSR and CSV patterns were observed were classified as having CSR.

Results

Abnormalities of respiratory rate and pattern were observed at some time in all patients. In most patients who
showed abnormal respiratory patterns, the abnormality was usually intermittent and was intermixed with periods of normal respiratory pattern.

The predominant respiratory abnormalities observed were correlated with the sites of the lesions (table 2). Nine of 23 patients had unilateral brain stem involvement whereas 14 had bilateral involvement. Pontine lesions were present in all cases, with coexistent infarction of the midbrain in four and of the medulla in nine. In general, patients with unilateral brain stem lesions were alert, while patients with bilateral lesions were either intermittently drowsy or comatose. In addition, ocular bobbing was prominent in two, and five patients represented examples of the "locked-in" syndrome.

Ten patients had predominantly normal respiratory rates and patterns; in six the lesions were unilateral while the other four had bilateral brain stem involvement. All of these patients were alert or only intermittently drowsy. In four patients with pontine lesions (three unilateral and one bilateral) the predominant abnormal pattern was CSV. CSR was the predominant respiratory pattern abnormality in four patients; all had bilateral pontine involvement with coexistent involvement of the midbrain in one and of the medulla in one. All of these patients were comatose. Sustained tachypnea with probable hyperpnea was observed in five patients, all of whom showed clinical evidence of bilateral pontine involvement. Two of these patients were intermittently drowsy while the other two were comatose. It should be noted that frequent episodes of CSR or sustained tachypnea were present only in patients with bilateral brain stem lesions. Apneustic respirations were not observed in any of these 23 patients; cluster respirations were noted infrequently in four patients (two with CSR, two with CSV).

In ten of 23 patients there was evidence of cardiac, pulmonary and/or other systemic diseases which may have played a significant role in the development of respiratory rate and pattern abnormalities. Such systemic disorders were present in three of our patients with CSR, two of four with CSV, and three of five with tachypnea, but in only two of ten with normal patterns.

Some apparent correlations between the presence of respiratory abnormalities and prognosis were noted. Eight of 23 patients died during the period of initial hospitalization (table 3). Nine of ten patients with predominantly normal respiratory pattern survived; one died of pneumonia. Three
of four patients with CSV survived; the fourth died due to myocardial infarction. Two of four patients with CSR died; in both, death appeared to be related to the CNS lesions. Four of five patients with sustained tachypnea died; all deaths appeared to be related to extensive brain stem pathology. In six of eight fatal cases, postmortem examination was obtained (table 4). All patients with predominantly CSR or tachypnea had extensive bilateral pontine lesions involving both basal and tegmental portions. Two patients with unilateral pontine lesions had normal or CSV patterns.

Arterial gas studies showed varying degrees of respiratory alkalosis and mild hypoxemia in most patients with either CSR or tachypnea (fig. 2). Respiratory alkalosis was most severe in patients with sustained tachypnea. In patients with normal or CSV patterns, respiratory alkalosis, if present, was mild.

Discussion

The regulatory centers for automatic respiration are located in the lower pons and medulla. These centers are constantly influenced by other complex neurogenic as well as metabolic control mechanisms. Thus, the presence of pathological lesions involving these centers may be expected to result in changes of respiratory function as reflected by alteration of rates and patterns. This study demonstrates the frequent occurrence of respiratory rate and pattern abnormalities and arterial gas changes in patients with acute brain stem infarction. It may be difficult or impossible to separate neurogenic from non-neurogenic factors resulting in the observed changes of respiratory rates and patterns. However, it is interesting to note that no apparent systemic disease was found to be present at the time of acute brain stem infarction in one of four patients with CSR, two of four with CSV, and two of five with tachypnea. This observation indicates that acute brain stem ischemic lesions may result in respiratory rate and pattern abnormalities without the presence of concomitant systemic diseases, as observed by others in patients with CSR.8

Other investigators4,9 have suggested that specific respiratory rate and pattern abnormalities may be correlated with the location of the central nervous system lesions. In this study, however, the types of respiratory rate and pattern abnormalities in acute brain stem infarction were not specifically related to the level of the lesions but rather to the extent and bilaterality of the lesions. Patients with lesions of similar size and location did not always demonstrate identical respiratory pattern abnormalities.

Normal or CSV respiratory patterns were usually present in patients with mild neurological deficit and unilateral involvement. All of these patients survived except for one patient in whom fatal pneumonia developed. Prognosis therefore seems excellent in this group of patients with acute brain stem infarction.

It is of considerable interest that apneustic respiration was not observed in any of our cases. Cluster respiration occurred occasionally in four patients in whom either CSR or CSV patterns predominated. Thus neither apneustic nor cluster respiration is a common phenomenon in patients with acute brain stem infarction.

The presence of CSR is usually thought to be indicative of deep bilateral cerebral dysfunction.6,8,12 Plum4 further observed that CSR implies bilateral dysfunction of neurological structures, usually deep in the cerebral hemispheres or diencephalon and, rarely, of those located as low as the upper pons. This study demonstrates that CSR occurs frequently in patients with extensive bilateral pontine lesions involving both basal and tegmental regions. These patients usually showed depression of level of consciousness.

Two of four patients with prominent CSR died. In agreement with Rout et al.,7 this observation suggests that the presence of predominantly CSR pattern in patients with acute brain stem infarction may indicate a grave prognosis. Patients with sustained tachypnea with probable hyperpnea had the most extensive brain stem involvement; most of these patients died. One patient survived but with severe neurological deficit. It is possible that in some of these five patients, the observed tachypnea may represent "central

<table>
<thead>
<tr>
<th>TABLE 4</th>
<th>Site of the Brain Stem Lesions and Predominant Respiratory Pattern in Six Autopsy-Verified Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pt.</td>
<td>Level of consciousness</td>
</tr>
<tr>
<td>M.S. Alert</td>
<td>R midpons, unilateral (base)</td>
</tr>
<tr>
<td>A.K. Coma</td>
<td>L midpons, bilateral (base and tegmentum)</td>
</tr>
<tr>
<td>J.R. Coma</td>
<td>Pons, old; bilateral R medulla, now</td>
</tr>
<tr>
<td>E.S. Drowsy</td>
<td>Midpons, bilateral (base and tegmentum)</td>
</tr>
<tr>
<td>A.V. Drowsy</td>
<td>L midpons (base)</td>
</tr>
<tr>
<td>E.S. Coma</td>
<td>Midbrain andpons, bilateral (base and tegmentum)</td>
</tr>
</tbody>
</table>

R = right, L = left, CSV = Cheyne-Stokes variant, CSR = Cheyne-Stokes respiration.
Hemodynamic Derangement for the Induction of Cerebrovascular Fat Deposition in Normotensive Rats on a Hypercholesterolemic Diet

YUKIO YAMORI, M.D., PH.D.,* RYOICHI HORIE, M.D.,† MASAYASU SATO, M.D.,‡ AND MASAICHI FUKASE, M.D., PH.D.§

SUMMARY Cerebrovascular ring-like fat deposition, which was noted only in hypertensive rats but never observed in normotensive rats even after they had been fed a high-fat cholesterol (HFC) diet for a long time, was successfully developed in the posterior communicating or other cerebrobasal arteries in normotensive rats fed an HFC diet for ten weeks after bilateral or unilateral carotid artery ligation or basilar artery ligation. These posterior communicating arteries with fat deposits were clearly dilated to a significant extent. These findings corroborated the fact that not only high blood pressure but also hemodynamic derangements induced by hypertension or other causes were important factors for the development of fat deposition in cerebral arteries.

Introduction

THE EXTREMELY RAPID development of ring-like arterial fat deposition in spontaneously hypertensive rats (SHR) fed a high-fat cholesterol (HFC) diet for two weeks was first reported by Yamori1 and its detailed mechanism was studied in SHR and other experimental hypertensive rats.2-4 Our previous studies further showed that fat depositions quickly developed even in the circle of Willis, especially in stroke-prone spontaneously hypertensive rats (SHRSP) and other experimental hypertensive rats on an HFC diet.5,6 Because normotensive Wistar-Kyoto (WK) rats and antihypertensive agent-treated experimental hypertensive rats on an HFC diet never had cerebrovascular fat deposition, hypertension was confirmed as the decisive factor for cerebrovascular fat deposition. We observed the dis-
Respiratory rate and pattern disturbances in acute brain stem infarction.
M C Lee, A C Klassen, L M Heaney and J A Resch

Stroke. 1976;7:382-385
doi: 10.1161/01.STR.7.4.382

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/7/4/382

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/