**Article abstract**—We observed acute onset of delayed psychosis in 8 patients 1 month to 11 years after right temporoparietooccipital (TPO) stroke or trauma. The psychotic disorder included hallucinations and, in some patients, delusions and agitation. All patients had spatioconstructional difficulties. None had an earlier psychiatric disorder. Seven of eight patients had clinical seizures, often in close temporal relationship to the psychosis. The pathophysiology of the psychosis may be related to that of the epilepsy.

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# Delayed psychosis after right temporoparietal stroke or trauma: Relation to epilepsy

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Psychosis characterized by hallucinations, delusions, agitation, and disorientation is often encountered in clinical states suggesting widespread brain dysfunction. These include systemic infection; severe renal, hepatic, or endocrine disease; withdrawal from alcohol or other sedative drugs; and numerous intoxications. Less commonly, psychosis has been described in association with focal brain lesions. Early authors emphasized brainstem or diencephalic lesions, such as the syndrome of peduncular hallucinosis<sup>1,2</sup> after vascular lesions of the midbrain tegmentum, or the hallucinosis and Parkinson syndrome after epidemic encephalitis.<sup>3-5</sup> Recent authors described psychosis after focal cerebral lesions, usually in the acute phase of cerebral infarction.<sup>6-8</sup> The lesions usually involved the temporo-occipital or parietotemporal cortex, although frontal lesions were also occasionally seen.

The relationship of psychosis to epilepsy has been examined in only a few of these clinical states and has not been clarified. Alcohol withdrawal has been best studied.<sup>9</sup> Seizures may occur, usually within 1 to 2 days, and hallucinosis or delirium may follow. Although seizures and psychosis often occur together, either may occur alone. A larger literature has addressed the relationship of psychosis to epilepsy among chronic epileptics, who may have a chronic paranoid psychosis, developing on average 14 years after onset of seizures,<sup>10,11</sup> or episodic psychosis, often preceding a seizure.<sup>12</sup> We now describe eight patients with psychosis after vascular lesions of the posterior portion of the right cerebral hemisphere (table). All were middle-aged or elderly adults with no history of earlier psychiatric illness. An interval of days to years intervened between the stroke and acute onset of psychosis. To our surprise, in reviewing these cases, we found that all but one had also developed seizures, although none had a prior history of epilepsy. In most of them, there was an obvious temporal relationship of the seizures and psychosis.

**Case reports. Case 1.** A 47-year-old, right-handed woman, previously in good health, awoke with headache, nausea, and vomiting. Later in the day she was lethargic. In a local hospital, CSF pressure was 320 mm  $H_2O$ , and the CSF contained 12 polymorphonuclear leukocytes. The next day she was transferred to our care.

She was lethargic but arousable. She spoke clearly in single words or short phrases and intermittently obeyed requests to perform simple movements, name objects, or repeat speech. Her speech and movements were slow and perseverative. On one occasion, the examiner left the room and returned 5 minutes later to find her still performing the finger-to-nose task. She would not stop until told to do so. Her limbs maintained cataleptic postures, and tone was somewhat increased. Visual fields were full to threat. The remaining cranial nerves, limb strength, tendon reflexes, and withdrawal from noxious stimuli were normal.

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Case no.	Age	Sex	Handedness	Injury	Psychosis
1	47	F	R	R parietooccipital hemorrhage	Agitation, delusions, hallucinations—
2	58	F	R	R parietooccipital hemorrhage	Mild delusions, hallucinations— 8 mos. after injury
3	23	F	R	R temporoparietal infarction	Agitation, delusions, hallucinations— 11 vrs. after injury
4	61	F	R	R temporoparietal contusion	Agitation, delusions, hallucinations— 3 mos. after injury
5	55	М	R	R temporoparietooccipital infarction	Recurrent episodes of agitation, delusions and hallucinations
6	85	F	R	R temporoparietal hemorrhage	Agitation, delusions, hallucinations— 1 mo. after injury
7	71	М	R	R temporoparietooccipital infarction	Mild delusions, hallucinations
8	78	М	R	R posterior embolic infarction	Hallucinations
Case no.	Other neurologic findings			Seizures	EEG findings
1	Constr	uctional a	apraxia,	L focal—6 days after injury	R temporal slowing
2	mild L HH (transient) Constructional apraxia, partial L HH (transient) mild L neglect (transient), mild L HS (transient)			L focal—8, 27 mos. after injury	R hemisphere slowing, R frontal sharp waves (postictal)
3	Constr	uctional a	apraxia	None	Bilateral frontotemporal slowing
4	Constr L H	uctional a H, mild L	apraxia, HP, L HS	L focal—3 mos. after injury	R hemisphere slowing (sharp wave activity during seizures)
5	Constr	uctional	apraxia	Generalized	R hemisphere slowing (occasional sharp waves)
6	Constr L H	uctional a P, L negle	apraxia, ect	L focal—2 wks. after injury	
7	Constructional apraxia, L HP, L HS, L HH, L neglect			L focal—days after injury. Generalized—3½, 7 mos. after injury	R temporal slowing
8	Constructional apraxia, L HH (transient)			L focal—15 mos. after injury Generalized—24 mos. after injury	L frontal slowing

### Table. Clinical, radiologic, and EEG findings in the patient group

In the next few days, she became less lethargic and better oriented but remained mildly confused. On the fourth hospital day, bifrontal headache recurred, accompanied by inability to count fingers in the left visual field. CT showed a right posterior intracerebral hematoma (figure 1). EEG showed right temporal slowing. The next day visual field defect had improved, but she did not spontaneously look to the left. There was no paralysis, but she used the left arm rarely and did not move it on command.

On the sixth hospital day, focal seizures began. She stared, became unresponsive, her head and eyes deviated

R =

L = M = F =

HH =

HP =

HS =

right.

hemiparesis.

Hemisensory loss.

homonymous hemianopia.

left. male. female.



Figure 1. CT scan of patient 1 showing right parietooccipital intracerebral hematoma. In this and other photographs of CT scans, the levels ascend as one proceeds rightward.

leftward, and there was jerking of the left arm and leg with extension of the right arm. Each seizure lasted up to 15 minutes and the seizures recurred every hour or two. She was treated with phenytoin and phenobarbital, and the convulsions ceased after 2 days. During and immediately following the seizures, there was little voluntary movement of the left arm, but movement returned to normal within 24 hours. She regained full alertness, and the subsequent course was remarkable only for frequent episodes of "depression" with outbursts of crying. At discharge, after 3 weeks, the only residuum was a left homonymous hemianopia and severe constructional apraxia.

After discharge, her moods became increasingly labile. She became agitated and aggressive and twice required psychiatric admission, in which she was confused, withdrawn, and violent, and unresponsive to phenothiazine and antidepressant medication. On the way home from her second hospital admission, she kicked out the windshield of her son's car. She proved to be completely unmanageable at home.

She was readmitted to the Massachusetts General Hospital 3 months after her previous discharge. Examination was unchanged except for her mental state. She was increasingly less cooperative as examination progressed. While she often followed simple commands, at other times she stared straight ahead without obeying. She identified the nurse as "a saint" and called the physician "son of God." She was frankly paranoid, saying, for example "Everyone is against me...I am doomed here. They are all paid off." At times she became agitated, sat up, and struck out at nurses and physicians. She was intermittently disoriented to place and time. At one moment she might say that she was "in a hospital in Beverly," but a few moments later would be oriented precisely to place and nearly accurately to date. At first she denied, but later admitted hallucinatory experiences. She wrote long, rambling, incoherent notes to her physician (figure 2). At times she would repeat words ("wait, wait, wait, ...") or short phrases ("wait one minute...") over and over again and could be observed talking to herself.

She remained in this state for 3 weeks. An EEG was normal except for right posterior slowing. She continued to receive phenytoin up to 900 to 1200 mg per day with blood levels of 15 to 25  $\mu$ g per milliliter and carbamazepine 800 per day. She also received increasing

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Figure 2. Letter written by patient 1 during her psychosis expressing her desperation and demonstrating her paranoid delusions.

doses of trifluperazine to 80 mg per day and became less agitated but increasingly listless, drowsy, and difficult to arouse. She died on the thirty-first hospital day.

At postmortem examination, the cause of death was a massive pulmonary embolus. The only cerebral lesion was the remnant of a large subcortical slit hemorrhage in the right parietooccipital region (figure 3). It straddled the intraparietal sulcus of the posterior parietal lobe and extended backward to approximately 1.5 cm anterior to the right occipital pole. **Case 2.** A 58-year-old, right-handed bookkeeper with a long history of psoriasis and arthritis suddenly had severe right temporoparietal headache. One hour later she vomited. After several hours the headache subsided, and she fell asleep but awoke with nausea and vomiting, and then felt well. Thirty-six hours later, a friend noted that she took wrong turns, lost her way in her own apartment, and walked into walls; she was then admitted.

She was alert and cooperative. Her affect was flat. When asked, she denied anything was wrong. She was oriented to person, place, and approximately to time. Recall of recent events was good. There was no aphasia. She neglected the left-most portions of compound visual stimuli, such as lines of print or thematic pictures. She drew from memory and copied diagrams poorly. An incomplete left homonymous hemianopia was present, more marked in the lower quadrants. The remainder of the neurologic examination was normal, except for occasional extinction on the left of simultaneous bilateral tactile stimuli, and diminished stereognosis bilaterally, more marked on the left.

CT (figure 4) demonstrated a corticosubcortical hem-



Figure 3. Location of subcortical "slit hemorrhage" (horizontal hatching) in patient 1 at autopsy. Drawings were made from tracings of the coronally cut brain slices. (A)—4.5 cm anterior to the occipital pole, 1.5 cm posterior to the splenium of the corpus callosum. (B)—3.5 cm anterior to occipital pole. (C)—2.5 cm anterior to occipital pole.

orrhage in the right parietotemporal region. EEG showed periodic sharp slow waves at a frequency of one per 3 seconds in the right temporal region. During the first 3 hospital days, she was intermittently disoriented and uncooperative. Several times she was found wandering in her room or in the halls. Her affect remained unchanged. At the end of this period, however, she was alert, fully oriented, and cooperative. The left visual neglect, left homonymous hemianopia, and left somatosensory findings disappeared, but the constructional apraxia persisted. She was discharged on her ninth hospital day. An EEG 5 months later showed frequent rhythmic 4-5 per second slowing in the right temporal area.

She remained well until 8 months later, when she suddenly had a sensation of a "soft cloak" over both shoulders and left arm. The left hand became numb for about 20 seconds. A strange medicine-like odor was briefly present. The next day she momentarily felt nauseated, and it appeared as though her living room had been rearranged. That night, she turned the radio off to fall asleep, but she continued to hear the broadcaster's voice repeating the same things. She slept fitfully and whenever she awoke, the voices were present. The following night, she heard classical music. Often the singers interrupted the performance to sing "Happy Birthday." There were also loud, insulting voices. She believed that an x-ray machine was located beneath the bed, and that the voices were attempting to move her into position to be x-rayed. Although the hallucinations were predominantly auditory, she also saw God as a glorious light that followed her. She could not tell if she was awake or asleep during these episodes. The hallucinations continued during the next 2 days. During the day, she appeared perfectly lucid, reporting on the voices in detail to her relatives. A left occipital headache was intermittently present. She was readmitted.

Examination was normal except for constructional difficulties. Intermittent auditory musical hallucinations were still present. An EEG showed right temporal theta and delta slowing, more pronounced than before, but without paroxysmal activity. Nevertheless, phenytoin was administered, and the hallucinations disappeared in the next 2 days.

Soon after discharge, she stopped taking the anticonvulsants. She remained well for 19 months, when she suddenly developed tingling in her left hand and arm and went to bed and lost consciousness. When brought



Figure 4. CT scan of patient 2 showing right temporoparietal intracerebral hemorrhage.

to the hospital, her head and eyes were deviated to the right, she spoke little, responded only to some simple commands, and showed continuous chewing movements. Her left arm was rigid with little spontaneous movement, and there was diminished withdrawal of the left extremities from noxious stimuli. Her tongue had been bitten.

She was given intravenous phenytoin. Within 3 hours, her mental state improved to full orientation, intact memory, and lucid thought. However, during the next 24 hours, she showed intermittent confusion, agitation, and hallucinations. An EEG showed high voltage slowing over the entire right hemisphere, with attenuation of background activity and some right frontal sharp waves. The intermittent confusion disappeared the next day, and an EEG 6 days later showed persistence of the right hemisphere slowing with return of the background rhythms.

In the following year she had several similar focal seizures, but no psychosis.

**Case 3.** A 23-year-old, right-handed woman suddenly began to drop objects from her left hand 9 days after giving birth to her first child. She quickly improved and a diagnosis of conversion reaction was made. When she returned home, she had difficulty caring for her baby and doing housework. She gradually improved and resumed most of her previous activities. However, there was evidence of a subtle change in her personality. Several deep personal friendships became shallower; she no longer met friends as frequently, and they no longer enjoyed each other's company. Her relationship with her husband changed similarly. She became more irritable and occasionally spiteful.

At age 34, she was admitted to a psychiatric hospital after a few weeks of progressively increasing anxiety, emotional lability, paranoid delusions, and hallucinations. She misidentified people and had visual hallucinations and illusions of human forms. She believed she was being spied upon, and that listening devices had been placed in her home. She was extremely fearful and had outbursts of screaming and crying. She and her husband believed several episodes had occurred in the previous 10 years, but none as severe as the present illness. There had been no history of blackouts, memory lapses, recurrent brief sensations or thoughts, or convulsions.

In the hospital, this behavior continued for several weeks. She was suspicious and afraid of physicians and nurses. Projective psychological testing<sup>13</sup> was "clearly full of schizophrenic material and breaks in reality testing, including visual hallucinations during the course of the interview . . . Her manner of communicating was peculiar and chaotic, and verbal communication was frequently invaded by autism." On the Wechsler Adult Intelligence Scale, verbal IQ measured 71 and performance IQ was 51. There was marked intratest scatter; she often failed easy items and succeeded on more difficult ones.

On neurologic examination, she was alert and oriented to person or place, but unsure of the date. There was no aphasia. Recall of a short story after 5 minutes was normal. She was extremely concrete in the interpretation of proverbs, had moderately severe constructional difficulty, and some trouble with mental calculations. Cranial nerves, including visual fields, were normal. No



Figure 5. CT scan of patient 3 showing right temporoparietal low-density lesion, presumed to be old infarction.

sensory, reflex, or motor deficits were present, and there were no pathologic reflexes.

EEG was abnormal because of bursts of generalized sharp slowing while awake and shifting frontotemporal slowing during sleep. The EEG was unchanged 1 month later. CT (figure 5) demonstrated an irregular area of decreased density involving extensive areas of the right temporoparietal region, with no mass effect or enhancement of the lesion. The findings were consistent with old infarction.

She was treated with phenothiazines and haloperidol. Within 2 months the agitation, hallucinations, and delusions had disappeared. When interviewed 18 months later, she had returned to steady work as a cashier and was functioning responsibly at home. She complained of insecurity, lack of self-confidence, anxiety, and depression. Her relationship to her husband had returned to its previous suboptimal state.

**Discussion.** Clinical features. Eight patients, age 23 to 85, developed psychosis after right TPO damage. Four were men and four were women. All were right-handed. The brain injury was infarction, probably embolic, in four patients, superficial subcortical hemorrhage in three, and cerebral contusion in one.

The interval between the original lesion and the onset of psychosis, known in six cases, was 1 month to 11 years. In the other two patients, evidence of an old, previously undetected infarction was discovered only after the psychosis developed.

The psychosis developed acutely, in hours or days, and lasted from days to months. Two patients had multiple psychotic episodes, and recurrences were suspected in two others.

During the psychosis all of the patients experienced hallucinations, which included both auditory and visual impressions of identifiable, meaningful objects. The hallucinations were accepted as real objects, although the patients differed widely in the extent to which they persisted in this belief.

In three patients (cases 2, 7, and 8) the hallucinosis was relatively pure without major alterations of orientation, intellect, memory, or affect. However, even these patients showed persecutory ideation and agitation that subsequently disappeared. The other patients showed more severe and persistent agitation, paranoid delusions, and confusion. The agitation consisted of screaming, crying, expressions of fear and, at times, aggressive behavior that included directed, dangerous physical assault. Agitation usually occurred with persecutory delusions, which often accompanied the hallucinations. The confusion was characterized by disorientation and incoherent thoughts that fluctuated markedly from moment to moment. Intermittent inattentiveness to the examiner's requests and perseveration of thought were also present.

In at least three patients (cases 3, 4, and 5), there were subtle changes in behavior during periods when the patient was not psychotic. These changes included irritability, suspiciousness, and intermittent agitation.

Associated neurologic findings included left hemiparesis in three patients, left homonymous hemianopia in five patients, and left hemisensory deficit or neglect in four. These abnormalities were generally mild and tended to improve. The only consistent neurologic finding, present in all patients, before, during, and after episodes of psychosis, was constructional apraxia. In two patients (cases 3 and 5), who were examined between psychotic episodes, constructional apraxia was the only abnormality on neurologic examination.

All patients had thorough investigation to exclude known causes of psychosis. None had a previous history of psychiatric illness or drug abuse. None had significant abnormalities in fluid, electrolyte, or acid-base balance, and all had normal renal, hepatic, pulmonary, cardiovascular, and endocrine function. One patient (case 5) had a history of significant alcohol consumption, but withdrawal from alcohol could have contributed to only one of his many psychotic episodes.

*Epilepsy and psychosis.* All but one of these patients had seizures that initially developed after the right hemisphere injury. In six patients the seizures had focal aspects, with leftward turning of the head and eyes or left-sided twitching or paresthesias. One patient had generalized seizures. In all cases, the seizures were accompanied by altered awareness and affect.

The onset of seizures preceded the psychosis in five patients and followed the onset of psychosis in two others. In some, the temporal relationship was close; patient 2 was well for 8 months after a right temporoparietal hemorrhage until an epileptic aura occurred, followed the next day by a psychosis that lasted 6 days. Patient 4 suffered a right temporoparietal contusion and improved steadily for 3 months until focal seizures started and were followed by a psychosis lasting 6 weeks. In patient 8, hallucinations developed several days after a seizure on several occasions, but other episodes of hallucinations were not preceded by seizures. In patients 5 and 7, hallucinations and delusions developed hours or days before seizures.

Both the high incidence of seizures and temporal proximity of seizures and psychosis suggested a relationship. Assuming an incidence of epilepsy as high as 25% after cortical stroke<sup>14</sup> or closed head trauma, epilepsy in seven of eight such patients would be expected with p < 0.0005. Selecting patients for psychosis and stroke, as we did, enhanced the incidence of epilepsy over selection for stroke alone.

Notwithstanding the high coincidence and temporal contiguity of epilepsy and psychosis in these patients, the precise relationship between the two phenomena is not clear. The psychoses were unlike the ictal phenomena, because they were long-lasting, did not usually respond to anticonvulsants (whereas the seizures did), and EEG did not show the paroxysmal activity present during seizures. Although many of the psychoses followed seizures, they were not typical "postictal" phenomena, because they were slower to develop and much longerlasting than postictal hemiparesis or visual defects. In several patients, under direct observation, postictal paresis cleared within hours, before the hallucinations and agitation were prominent. However, postictal stupor and confusion could be a more severely abnormal state that had been present in the immediate postictal period and then evolved into psychosis.

*EEG*. The EEG was recorded in all patients on one or more occasions and was abnormal at least once in each of them. Patients 1 and 2 had the first EEG within 3 days of right posterior hemorrhage. Rhythmic, sharp, high-voltage activity was present over much of the right hemisphere, although no clinical epilepsy was noted. Patient 3 had her first EEG during focal seizures; again rhythmic, sharp high-voltage slowing was present on the right. The same three patients all had EEGs in a postictal period, several hours to 3 days after termination of seizures, and all three showed rightsided slowing that was less prominent than in the initial record. Finally, all three had EEGs during a psychotic episode; these were nonspecifically abnormal with some theta and delta slowing, often more prominent on the right, varying from patient to patient, but always less prominent than in the postictal period. No spike or sharp wave activity was seen.

Pathophysiology of the psychoses. All of these patients, by clinical and radiologic criteria, had a lesion affecting the TPO portion of the convex surface of the right hemisphere. We have not observed a similar incidence of psychoses in patients with lesions situated in homologous areas of the left hemisphere. The significance of this asymmetry is unclear. It may reflect the occurrence of aphasia with left hemisphere lesions, making detection of the syndrome more difficult. It may also reflect the greater prominence of spatial disabilities with right hemisphere damage, predisposing to misinterpretation of environmental events and to the development of hallucinations and delusions.

The clinical and EEG data in some of our patients suggested that an epileptic discharge, resulting in abrupt and widespread dysfunction of the right hemisphere, preceded the psychosis. The location of the underlying lesions in the right TPO region may have predisposed to such widespread seizure discharges. In primates<sup>15,16</sup> this region of cortex receives projections from all of the modality-specific sensory association areas, and projects widely to frontal granular cortex and to limbic structures in the medial temporal lobe, which have the lowest thresholds for seizure discharge among cerebral structures.<sup>17</sup> An epileptic discharge, originating in the parietotemporal convexity, could spread through the ipsilateral hemisphere, and even transcallosally, with only minimal convulsive manifestations. In our patients, widespread epileptiform discharges over the right hemisphere were associated with defects of awareness and affect with only mild contralateral clonic activity.

The emergence of psychosis hours or days after such an epileptic discharge, with eventual recovery after weeks or months, suggests that the psychosis represents an unstable pathophysiologic state, associated with improving but still abnormal function within the right temporoparietal, prefrontal, and limbic structures. Serial EEGs showed a progressive diminution in the extent, amplitude, and rhythmicity of the abnormal discharges as the patient progressed from the acute lesion or clinical seizure to the period of psychosis.

Because the psychoses were intimately associated with epilepsy, they resemble the episodic psychotic disorders of patients with long-standing psychomotor epilepsy.<sup>12</sup> Our experience demonstrates that similar psychosis can develop with the onset of seizures late in life, and after only a single episode of clinical seizures. Vascular or traumatic lesions of the temporoparietal region may cause epileptic psychosis.

Despite the close relationship with epilepsy, not all psychotic episodes in all patients were associated with clinical seizures. Patient 3 had no clinical or EEG evidence of epilepsy at all; in this case psychosis may have been associated with preceding seizure discharges that were clinically detectable but not observed, or there could have been an abnormal "subepileptic discharge," which might have reverted to normal instead of worsening to a frank seizure discharge.<sup>12</sup> This might also explain the occurrence of agitated delirium in the acute phase of cerebral infarction.<sup>6-8</sup> Unwitnessed seizures may have preceded admission of these patients to the hospital in a psychotic state, or EEG in the earliest phases of infarction might have shown epileptic discharges over the right hemisphere. However, the delirium might also have been associated with a "subepileptic discharge" without clinical epilepsy or electrographic seizure discharges.

#### References

- 1. von Bogaert L. L'hallucinose pedonculaire. Rev Neurol (Paris) 1927;1:608-17.
- Lhermitte J. L'hallucinose pedonculaire. Encephale 1932;27:422-35.
- Guiraud P. Les delires chroniques. Encephale 1925;20:663-73.
- Steck H. Contribution à l'étude des sequelles psychiques de l'encephalite lethargique. Schweiz Arch Neurol Neurochir Psychiatr 1927;21:214-37.
- Claude H, Ey H. Troubles psycho-sensoriels et etats oniriques dans l'encephalite epidemique chronique. Nouv Presse Med 1933;41:1281-5.
- Horenstein S, Chamberlain W, Conomy J. Infarction of the fusiform and calcarine regions: agitated delirium and hemianopia. Trans Am Neurol Assoc 1967;92:85-9.
- Medina JL, Rubins FA, Ross E. Agitated delirium caused by infarctions of the hippocampal formation and fusiform and lingual gyri: a case report. Neurology (Minneap) 1974;24:1181-3.
- Mesulam M, Waxman SG, Geschwind N, Sabin TD. Acute confusional states with right middle cerebral artery infarctions. J Neurol Neurosurg Psychiatry 1976;39:84-9.
- 9. Victor M. Alcoholism (chap 22). In: Baker AB, Baker LH, eds. Clinical neurology, vol 2. Hagerstown, MD: Harper & Row, 1978.
- Slater E, Beard AW. The schizophrenia-like psychoses of epilepsy. Br J Psychiatry 1963;109:95-150.
- Flor-Henry P. Psychoses and temporal lobe epilepsy: a controlled investigation. Epilepsia 1969;10:363-95.
- Dongier S. Statistical study of clinical and electroencephalographic manifestations of 536 psychotic episodes occurring in 516 epileptics between clinical seizures. Epilepsia 1959;1:117-42.
- Rorschach H. Psychodiagnostics. New York: Grune and Stratton, 1969.
- Richardson EP Jr, Dodge PR. Epilepsy in cerebral vascular disease. Epilepsia 1954;3:49-74.
- Pandya DN, Kuypers HGJM. Cortico-cortical connections in the rhesus monkey. Brain Res 1969;13:13-36.
- Jones EG, Powell TPS. An anatomical study of converging sensory pathways within the cerebral cortex of the monkey. Brain 1970;93:793-820.
- Ajmone Marsan C, Gumnit RJ. Neurophysiological aspects of epilepsy (chap 2). In: Vinken PJ, Bruyn GW, eds. Clinical neurology, vol 15. New York: Elsevier/North Holland, 1974:30-59.



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