A possible relationship between the carotid body and schizophrenia has never been investigated, so far as can be ascertained. Coincidentally, in the course of routine forensic necropsies, I have observed that the carotid bodies in schizophrenics are even smaller than average and especially difficult to detect. Upon microscopic examination, the reduced size appears due to contracted, thickened arterioles and empty vessels. Normally the carotid bodies are profusely vascularized; in fact no other organ in the body has a richer blood flow. Many examples of peripheral, vasomotoric disturbances in the schizophrenic are cited in the literature. Since these would point directly to involvement of the carotid body, it is indeed intriguing that such a relationship apparently never has been considered.

Acrocyanosis and other vasomotoric disturbances in the schizophrenic have been observed by many investigators. Cornell as well as Bleuler considered the cyanotic hand of the schizophrenic to be pathognomonic. Shattock (1950) found that peripheral cyanosis was present in more than half of catatonic schizophrenics. He also observed a lowered skin temperature, low blood pressure, and very low pulse in the same patients. Acrocyanosis in schizophrenics, especially of the fingernail folds, has been reported by others (Abramson 1944 and Altschule and Sulzbach 1949). At necropsy, a constant finding is a small heart, in which the walls of the auricles are reduced in thickness (Lewis 1923 and Shattock 1950). These characteristics appear to be independent of the physical activity of most patients; but they are certainly related to their mental condition. In young schizophrenics, in the initial phase of the illness, transient cyanosis of the hands reflects the degree of emotional tension (Shattock 1950). The cyanotic hand of the schizophrenic thus would seem to deserve further consideration.

The local anatomical substrate for the vascular response leading to acrocyanosis is primarily the large number of arteriovenous anastomoses termed "glomer" situated in the deeper layer of the corium (Johnston and Whillis 1946). The artery to each glomus divides into a number of fine arterioles having myoepithelioid-like sphincters. The sympathetic provides a very rich supply of unmyelinated fibers to the walls of these vessels having myoepithelial-like sphincters. The sympathetic provides a very rich supply of unmyelinated fibers to the walls of these vessels which are thus capable of complete closure (Lewis and Pickering 1931-33).

The carotid body is, in respect of vessels and efferent innervation, structurally identical to the cutaneous glomera (Adams 1958). In 1928, de Castro, intrigued by the profuse vascularization and constant innervation of the carotid body in a variety of species, realized that it is a sense organ which "tastes" the blood gases. It is the site of origin of the chemoreceptor afferent nerve fibers of the glossopharyngeal nerve (sinus nerve) which run to the respiratory and cardiovascular autonomic centers. It is not unlike a set of electrodes sensing the PACO₂, PAO₂, and pH (Hornbein 1968). A very important factor in this mechanism is the blood flow through the carotid body. Its structure is that of a vascular glomus, consisting of arterio-venous anastomoses with myoepitheliod cells. The structural analogy with the cutaneous glomera has been pointed out by many authors (see Adams 1958, p. 139). Its flow is normally extremely high, as is its oxygen consumption (Daly, Lambertsen, and Schweitzer 1954).

The blood vessels are very richly supplied by sympathetic fibers from the superior cervical ganglion. Electrical stimulation of these fibers elicits a consistent rise in chemoreceptor discharge because of the reduced blood flow through the organ due to vasoconstriction of its arterioles (Biscoe 1971 and Eyzaguirre and Lewin 1961). And Biscoe and Purves (1967) demonstrated in animal experiments that sympathetic activity greatly influences the carotid body chemoreceptor signals.

The increased sympathetic tone in the schizophrenic, which is a prerequisite for cyanosis of his hands, must accordingly also affect his carotid bodies, producing vasoconstriction, reduced blood flow, and oxygen depletion within them. This must result in continuous—and erroneous—signals in the sinus nerve. These signals, for example, might misinform the brain of a
general anoxia in the organism which in fact does not exist. The situation may best be illustrated by studying the diving animal (James and Daly 1968). Here the chemoreceptor is strongly stimulated and produces three phenomena: peripheral vasoconstriction, low pulse, and low blood pressure. It is the same three somatic symptoms said to be characteristic of catatonic schizophrenia.

The catatonic schizophrenic, however, is not a diving animal. Increased chemoreceptor discharge—whether erroneously elicited or released by real physiological requirements—should result in hyperventilation. Schizophrenics indeed do have an above normal pulmonary ventilation—although this, of course, must be within limits compatible with life. When the schizophrenic is subject to a restricted oxygen intake, his abnormal respiratory response is clearly revealed. In 1942 Horvath, Dill, and Corwin, in parallel with their study of the military problems of parachute escape, exposed catatonic schizophrenics to an anoxic situation severe enough to induce unconsciousness. These patients proved to be unexpectedly resistant to anoxia. But the most striking feature was the slow return to normal breathing during the recovery phase. In normal subjects a period of overventilation is immediately followed by a period of hypoventilation or apnoea (Heymans and Neil 1958). The catatonic schizophrenics, in contrast, continued hyperventilating for several minutes after the oxygen supply had returned to normal. Although Horvath, Dill, and Corwin do not mention it, this abnormal oxygen response in catatonic schizophrenics is most certainly due to a chemoreceptor malfunction.

What effect persistent, erroneous chemoreceptor signals may have, especially to the brain itself, is difficult to surmise. The carotid body is, for all practical purposes, a sense organ which relays the blood gas levels to the brain. The impulses reach the hypothalamus and presumably the formatio reticularis (palaeostriatum), and there is little doubt that such impulses reach the cerebral cortex. The signals would normally never be perceived. But if erroneous and continuous, they might trouble the mind of the schizophrenic, perhaps accounting at least in part for his introversion and the delusion of being influenced by radiation or electrical impulses.

The concentration of dopamine in the carotid body is allegedly very high (Dearnaley, Fillenz, and Woods 1968). All the modern chemotherapeutic agents used in the treatment of schizophrenia have one feature in common: an antidopaminergic effect (Fog 1972). If dopamine indeed is the major transmitter of the carotid body and its cranial nerve, then the chemoreceptor function would not escape the effect of antidopaminergic medication. Should this account for the therapeutic effect of these drugs, then other measures would certainly seem to be far more effective and reliable: severing of the sympathetic fibers to the carotid bodies or—if necessary—even the sinuses nerves is suggested as an effective measure.

Summary

Acrocyanosis and other vasomotoric disturbances in catatonic schizophrenics have been observed by many investigators. The local anatomical substrate for acrocyanosis is the glomera in the corium. The carotid body is, in respect of vessels and efferent innervation (sympathetic), structurally identical to the cutaneous glomera. The increased sympathetic tone in the schizophrenic, which is a prerequisite for cyanosis of his hands, must accordingly also affect his carotid bodies, producing vasoconstriction, reduced blood flow, and oxygen depletion within them. This must result in continuous—and erroneous—signals in the chemoreceptor nerve, which again produce peripheral vasoconstriction, low pulse, and low blood pressure: precisely the somatic symptoms of catatonic schizophrenia. The schizophrenic's physiological situation cannot be far from that of the diving animal, and studies of the military problems of parachute escape have demonstrated that catatonic schizophrenics most certainly have a chemoreceptor malfunction. Persistent and erroneous chemoreceptor signals to the brain may perhaps disturb its flow or—if perceived—may trouble the mind of the schizophrenic. Severing of the sympathetic fibers to the carotid bodies or, if necessary, the sinus nerves is suggested as an effective measure.

References


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**job exchange—a free service**

The National Society for Autistic Children, Information and Referral Service, operates a job exchange as a free service. The job exchange receives listing of both employees and employers all over the country for services to handicapped children and adults, especially those who are autistic or autistic-like. The job exchange, which attempts to match employers and employees, has names of special education teachers, teacher aides, psychologists, nurses, administrators, social workers, and therapists of all kinds (including speech, occupational, drama, art, and adapted physical education therapists). The job exchange also hears from students in these areas looking for summer camp jobs. Jobs are in public and private programs of many kinds.

Information on more job openings and more prospective employees is needed. With the new mandates for services and the concomitant shortage of personnel, it is important to cut the search time for employers and employees who are interested in serving autistic persons. Suggestions are welcome. Those wishing further information about this free service should write to: Job Exchange, National Society for Autistic Children, Information and Referral Service, 306 31st Street, Huntington, W. Va. 25702.