CONVULSIONS DURING ETHER ANESTHESIA CONTROLLED
BY CURARE (INTOCOSTRIN)

IRVING GREENFIELD, M.D.*

Pittsburgh, Pa.

The use of curare (intocostrin †) to control anesthetic convulsions is the
subject of this report. It has not been reported previously. Anes-
thetic convulsions are serious because of the high mortality rate (18–
50 per cent) and the prospect of residual neurologic changes which may
persist postoperatively in those surviving (1). It is important that
the occurrence of these convulsions is recognized and means for con-
trolling them immediately instituted. Wilson, in 1927, was the first to
call attention to this phenomenon during anesthesia (2).

About thirty-five articles have been written expounding a variety of
theoretic considerations of convulsive seizures during anesthesia. Within
the past few years attention has been directed to anoxia of the
central nervous system as the single factor in producing this state
during anesthesia. It is not the intention in this paper to enter this
discussion, but rather to present one case and its treatment with
intocostrin.

The patient had been injured at 8 p.m. on October 25, 1944. He
arrived at the hospital at 5 a.m. the next morning, apparently in good
condition, and at 9:30 a.m. was admitted to surgery with a diagnosis of
a penetrating wound of the right lateral iliac region. Roentgenologic
examination was not wholly satisfactory in confirming clinical impres-
sion of an intra-abdominal penetration.

Preliminary consisted of morphine sulfate, 1/6 grain, and atro-
pine sulfate, 1/100 grain, intramuscularly, thirty minutes before in-
duction of anesthesia. The blood pressure was 120 mm. systolic and
80 mm. diastolic, pulse was 90, and the respirations were 20 before
operation.

The patient was anesthetized using nitrous oxide, oxygen, ether
and carbon dioxide absorption sequence. Induction was uneventful,
with only a slight excitement stage. It was noted at this time that the
pupils were dilated to about 65 per cent and the eyeball activity was
moderate. He was turned into a left Sims’ position for debridement of
the wound and the tract was followed down. An abdominal ex-
ploratory procedure was indicated when it was demonstrated that the
fragment had penetrated the peritoneal cavity. The patient was then
placed on his back and the abdominal exploration started. To over-

* Director of Anesthesia, Montefiore Hospital, Pittsburgh, Pennsylvania.
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come moderate laryngospasm, intubation was carried out through the mouth, using a number 38 Magill tube under direct vision. The pupils were still dilated, and some eyeball activity was present. There was good activity of the intercostal muscles.

One hour and thirty minutes after the start of the anesthesia, the patient was sufficiently relaxed, but because of his rapid jerky breathing, the liver and the adjacent abdominal contents obscured the surgeon’s exploration of the posterior aspect of the duodenum in the vicinity of the ligament of Treitz. Breathing was definitely diaphragmatic in character; the intercostal muscles were paralyzed and a prominent tracheal tug was evident. De-etherization with oxygen was started immediately. At the same time it was noticed that there were slight muscular twitches about the eyelids. This rapidly progressed upward to include the forehead. The face mask was removed and it was noted that the muscles about the mouth and nose were also starting to twitch. The left arm, which was extended and fixed to an intravenous arm-board, was soon involved. First, the muscles of the left fifth finger started to contract and relax slowly. This soon spread to involve the entire left hand and within a very few seconds the entire left forearm and arm were included in this neuromuscular phenomenon. The feet were quickly undraped and it was noted that the toes were also twitching. Within a relatively short time the entire trunk was involved and soon the entire body was in a state of clonic spasm. The duration of each spastic seizure was about two seconds and then a period of relaxation would follow which lasted for about four seconds. Anesthesia was immediately discontinued. Oxygen under slight positive pressure, with frequent emptying of the bag, was started and new soda-lime was inserted into the system. I hesitated to give the patient any pentothal at this time, because of the extremely deep anesthesia. The color of the blood was normal. Blood pressure was 146 mm. systolic and 60 mm. diastolic, the pulse 120, and the respirations were about 50. The temperature was checked at this time and was found to be 98° F. axillary. De-etherization was continued for about five minutes, and then sodium pentothal, 5 cc. of a 3.5 per cent solution, was given very rapidly. Additional pentothal in 2 cc. doses was then injected every ten seconds, until 30 cc. (1 Gm.) had been given over a five minute period. It was then decided to discontinue any additional barbiturate, as there was no indication that the patient’s condition was improving. The convulsions increased in frequency and severity, and at times it was necessary for three people to hold the patient on the table. The endotracheal tube, which was partly filled with mucus, was removed and another reinserted.

Twenty minutes after the onset of the convulsions and ten minutes after the pentothal had been given, it was decided to give the patient 1 cc. (20 units) of intecrin intravenously. The dose was given in approximately ten seconds. Within one minute the convulsions sud-
duly stopped. The patient went into apnea which was easily controlled by slight manual pressure on the bag. The blood pressure level and pulse remained good. Spontaneous respirations returned in about fifteen minutes. At first they were shallow, rapid and jerky, but they gradually returned to a more normal type. The surgeon was able to complete his exploratory procedure. The operation was finished at 12:30 p.m., and the patient was awake at 3:30 p.m. The postoperative course until the tenth day was uneventful. At that time he was discharged from the hospital.

**Comment**

The probable cause of the convulsions was the prolonged, deep anesthesia, with its resultant anoxia. An intravenous barbiturate was not used immediately because of the danger of additional respiratory depression. When sufficient ether had been removed from the lungs, pentothal was used, but the convulsions did not cease. The administration of intocostrin produced prompt cessation of the clonic spasms, and the musculature became flaccid. The violent heaving of the diaphragm, which pushed the abdominal contents out of the wound, stopped and the surgeon was able to proceed very rapidly. Apnea was not disturbing, for as long as good circulation was maintained with artificial respiration, it was felt that within a short time spontaneous respiration would return. It took fifteen minutes of artificial respiration before the patient was able to breathe spontaneously.

The use of curare in the treatment of convulsions dates back to 1867 when it was first employed for patients suffering from epileptiform seizures, rabies, chorea, strychnine poisoning, and a variety of tics. Recently, it has been used for patients about to undergo metrazol shock therapy to prevent the fractures and other injuries occurring during the convulsions (3).

The site of curare action in this particular is peripheral rather than central, interrupting the transmission of somatic nerve impulses to the effectors. This action of curare is performed without any apparent associated depression of the central nervous system.

**Conclusion**

A case of convulsions developing during ether anesthesia is presented. Control of the convulsions by the use of curare (intocostrin) after the intravenous barbiturates (sodium pentothal) had failed is described. This is the first time, according to the available literature, that curare has been used under such circumstances.

**References**