Slide 1 Case 1 AANP Slide session 1960

D. G., 35 year old white woman first seen by the reporting physician in 1957 at the age of 35 years. In 1955 she developed polyuria and polydipsia, and after a diagnosis of diabetes insipidus was made she was treated with pitressin in oil. Following an attack of "stomach flu" in 1957, during which she had fever of 104°, nausea and abdominal cramps, she developed pain in the lower extremities and marked generalized weakness. She had amenorrhea since August 1956.

The family history indicated that her father died from carcinoma of the bowel, a sister died from carcinoma of the breast, and her mother had surgery for carcinoma of the breast.

Physical examination revealed swelling and tenderness of both lower extremities. She responded to aspirin and vitamins. In May 1957 she developed somnolence, severe pain in the lower extremities and a septic temperature. The only physical finding was severe hyperesthesia of the lower extremities. For a time she was disoriented. Skull films were negative and air studies showed a normal ventricular system. The cerebrospinal fluid protein was 126 mgm.%, the Pandy reaction 4 plus, serology negative, gold curve all zeros, spinal fluid glucose 56 mgm.%. She responded to penicillin but continued to have periods of disorientation, trance-like states and extreme weakness. Because of the possibility of an infiltrating glioma in the hypothalamus, a course of deep x-ray treatment was administered (5300 R for 38 days through five portals). She improved for a time but, in September 1958, she had another attack characterized by stupor and fever. She was admitted to the hospital for the last time in September 1959, exhibiting signs of myxedema. The cerebrospinal fluid pressure was 170 mm., with increased protein (167 mgm.%), 5 leucocytes and a glucose of 75 mgm.%. Because of the low protein-bound-iodine and radio-active-iodine determination she was given thyroid after which the stupor lessened and she was able to speak. After a test with 25 units ACTH the blood corticoids rose from 8.3 mgm. to 43.8 mgm. Despite supportive therapy she did poorly and died.

Slides 2A and 2B

ARMED FORCES INSTITUTE OF PATHOLOGY
Washington 25, D. C.

CASE FOR PRESENTATION AT BOSTON SLIDE SESSION
June 1960

The section is from the cerebellum. Diagnoses in this case (AFIP Acc. 320142) included platybasia, probable Arnold-Chiari malformation, and secondary hydrocephalus.

The patient was a 37-year-old sergeant in the United States Army. He was admitted to hospital 28 September 1958 for evaluation of episodic vertigo associated with head pain which first appeared in August 1958. The first indication of trouble occurred while he was taking a shower; it seemed that the room suddenly revolved and he fell to the floor. He was up again in a few minutes. Two weeks later he had another episode of vertigo, and during the attack he had blurred vision. From early November onward, attacks occurred almost daily and lasted as long as 4 to 6 minutes. Between attacks the sergeant felt "fine." He denied vomiting. There was definite facial asymmetry. "Cerebellar" signs were not elicited. Lumbar tap revealed an opening CSF pressure of 380 to 390 mm. H_2O. Ventriculogram revealed symmetrical dilatation of both lateral ventricles and of the IIId ventricle and aqueduct. On suboccipital craniectomy the arch of the first cervical vertebrae was removed. No abnormality was found in the posterior cranial fossa. The patient was returned to the ward in good condition. A few hours later he suddenly died (4 Dec. 1958).

There was no family history which seemed pertinent. The brain weighed 2400 gm. The cerebrum showed nothing beyond ventricular dilatation. Nor was anything abnormal remarked about the cerebellum grossly. There was "a moderate degree of platybasia of the shallow posterior fossa. . .as well as a prominent odontoid process. This may possibly have caused a pressure on the brain stem."

References to the pathological condition presented are the following:

1. Duncan, D., and Snodgrass, S.: Diffuse hypertrophy of cerebellar cortex (myelinated neurocytoma). Arch. Neurol. & Psychiat. 50:677, 1943.

2. Oppenheimer, D.: A benign "tumour" of the cerebellum. Report on two cases of diffuse hypertrophy of the cerebellar cortex. J. Neurol., Neurosurg., & Psychiatr. 18:199, 1955.

3. Russell, D., and Rubinstein, L. J.: Pathology of Tumours of the Nervous System, Edw. Arnold Ltd., London, 1959, p. 168.

4. Hallervorden, J.: Ueber die Hamartome (Ganglioneurome) des Kleinhirns. Deutsche Ztschr. f. Nervenheilk. 179:531, 1959 (latest compilation; 21 cases).

WEBB HAYMAKER, M. D.

Slide 3

History of Case Submitted by K. M. Earle, M.D.
University of Texas Medical Branch, School of Medicine, Galveston

This 15 year old Latin American boy died in San Antonio and the autopsy was performed there. The brain was sent to me by Dr. A. O. Severance of San Antonio. Records obtained from the hospital in San Antonio indicate that his birth was full term and uncomplicated. He developed normally for the first ten months at which time convulsions began and he was diagnosed as having "cerebral palsy" and "epilepsy". He was one of four children. One sister has epilepsy, but the other children and the parents are living and well. The exact nature of these convulsions and exact medications are not known. History was difficult to obtain from informants and was considered to be unreliable. The boy was admitted to a hospital in September of 1958, but the family took him home against advice and all that is known is that he was having convulsions at the time. He was readmitted in November, 1958, with "convulsions and fever". An informant stated that he had been unable to feed himself since September and had been fed by a nasal tube. Shortness of breath, episodic coughing and loss of sphincter control had been present since September. On admission to hospital in November he was described as a slender boy who was small for his age. He was comatose and respirations were labored. His neck was "stiff" and the pupils did not react to light or accommodation. Coarse rales were present in all lung fields. The extremities showed increased tone. Reflexes were hyperactive but there were no pathological reflexes.

Laboratory studies showed: RBC 4,000,000; Hgb. 11.5 gms.; WBC 18,450, Stabs 1, segs 76, neutro. 77, lymph 14, mono 1, eso. 8.

Urine showed 20-30 pus cells per h.p.f.; 6-8 rbc, sp. gr. 1.000; alb. and sugar negative.

V.D.R.L. negative.

X-Rays of the chest showed evidence of pulmonary edema. Despite therapy respiratory difficulty worsened and he died on the third hospital day. Spinal fluid examination is not recorded.

Autopsy revealed "multiple, pearly white, shiny nodules on the face, varying from 0.1 to 0.4 cm., which are well circumscribed, firm and distributed over the nose, cheeks and eyelids". Areas of brownish pigmentation and several large pigmented nevi were scattered over the abdomen and trunk. The kidneys contained well circumscribed, firm, round and oval nodules of light yellow and grey tumor masses in the cortex and medulla. The tumors varied from 0.2 to 4 cm. in diameter. The bladder showed evidence of acute cystitis and the lungs showed pulmonary edema and thickening of alveolar walls. The brain weighed 1165 gms. and showed greyish white nodules in the cortex and projecting into the ventricles. Some of these nodules were calcified.

AANP Slide Session 1960

Slides 4A and 4B

Phila. General Hospital (NP) 59-533

W.M. 44

Onset of symptoms of multiple sclerosis at age 24. Admitted PGH 1950. (Younger brother admitted at same time with similar C.N.S. symptoms) Anterior rhizotomy (T 11 - S 1) performed 1951 for pain and contractures. No notation of abnormality of lumbo-sacral cord at time of operation. Necropsy 1959 revealed typical plaques of multiple sclerosis in brain and cord. Cord was markedly enlarged in the lumbo sacral region. Section of dura revealed a firm, white lobulated mass covering and compressing the ventral half of the cord. This was not adherent to the dura or the cord surface. Spinal roots could be identified between the mass and the dura, and passing between the lobulations. Grossly, no tumor was visible at the lower sacral level, but circumscribed areas within the ventral roots presented architectural characteristics similar to those of the mass lesion.

Slide 5

UNIVERSITY OF MARYLAND
DEPARTMENT OF PATHOLOGY
DIVISION OF NEUROPATHOLOGY

NS-6796

A lad of 5 was admitted to University Hospital on April 19, 1957 because of progressive weakness of his legs and right arm.

The prenatal course of his mother was uneventful. Delivery was by difficult forceps. Birth weight was 5 pounds 10 ounces. Development appeared normal until April, 1956 when his mother noted that he began to walk stiffly, abduct his right leg and to throw his foot outward while walking. The child complained of stiff neck. He was unsteady on his feet and fell down frequently.

Past history revealed admission to another hospital for tonsillectomy; however, this was not performed because a displaced cervical vertebra was discovered. He had frequent bouts of vomiting since birth. However, x-ray examination of the GI tract was normal. He had pneumonia & tracheotomy in October, 1954. The child wore a cervical cast and collar for awhile and in 1954 underwent a cervical laminectomy with fusion. In 1956 he began to show the progressive stiffness of his legs and weakness of his right arm. For about 5 to 6 weeks prior to the April, 1957 admission, he had been unable to walk and for 3 weeks prior to admission he complained of headache and vertigo.

Physical examination showed a chronically ill, irritable, alert white male of stated age. He lay on his right side. The following neurological abnormalities were noted: Decreased tone in the right arm; the right wrist held extended with elbow flexed; very limited use of right arm; increase in muscular tone in both legs which were held in extension. There was inability to dorsiflex in right foot. Deep tendon reflexes were absent in the right arm; hyperactive in both legs. The plantar response was extensor. There was no nystagmus or gross ataxia. The gait was markedly spastic, and the child was unable to walk without support.

Cervical myelogram with Pantopaque was performed on 23rd of April and revealed complete obstruction of the subarachnoid space at the level of the 10th thoracic vertebra. Lumbar puncture revealed slightly xanthochromic fluid with an abnormal Queckenstedt sign. Pantopaque was next injected into the cisterna magna where it was retained.

On May 6, 1957, a laminectomy from 3rd cervical through first thoracic vertebrae was performed. There was an intrinsic swelling in the cervical spinal cord which was very hard and firm. The wound was closed without biopsy.

On May 20, laminectomy was performed from the 8th through the 10th thoracic vertebrae. After the dura was opened, a large, soft, swollen spinal cord was noted. A needle was introduced into the cord and 20 cc. of xanthochromic fluid was withdrawn. Incision was next made over the dorsal portion of the cord in the midline and a cystic space was exposed. A tiny fragment of the cyst wall was removed. A pathologic diagnosis of syringomyelia was made. Two silk setons were left in the cystic cavity and were embedded in the perispinal muscles.

Following operation, spasticity remained. His right arm now became completely paralyzed and flaccid. He was discharged on June 13 but was readmitted August 4 because of difficulty in breathing. The interval since discharge had been characterized by progressive paralysis, so that now he was quadriplegic and in respiratory distress, with recurrent bouts of cyanosis, apnea and loss of consciousness. Spastic paralysis continued in the lower extremities with ankle clonus and extensor plantar response. The upper extremities were flaccid. The patient died quietly on August 7

UNIVERSITY OF COLORADO MEDICAL CENTER
4200 East Ninth Avenue, Denver 20, Colorado
DEPARTMENT OF PATHOLOGY

Autopsy No. 59-328
Date 1-26-60

Slide 6

HISTORY: The patient was a 73 year old Spanish-American female. At age 64 in 1951, she had a carcinoma of the cervix which was treated with irradiation. In 1955, she incurred a fracture of the right hip but thereafter remained well until the spring of 1959, when she began experiencing difficulty with swallowing. Neurological examination on 5-1-59 revealed inability to look upward and to the right, weakness of the right lower face, deviation of the uvula to the left, a vocal cord paresis, diminished gag reflex and hyper-reflexia of the left arm and leg. It was felt that the patient had some type of neoplastic lesion involving the brain stem. Irradiation therapy was planned but before it could be initiated the patient's symptoms began subsiding and neurological examination on 6-30-59 was essentially within normal limits. She was discharged to a nursing home and for several months got along without any major difficulty. Early in October of 1959, she had fever followed by gross unresponsiveness which developed rapidly over a period of a day or two. On physical examination on readmission (10-16-59) her head was constantly turned to the left and she was unable to move her right arm and leg. There was a horizontal nystagmus with a slow component to the left. Further findings included right lower facial weakness, a diminished right corneal reflex, absent gag reflex on the right, increase of deep tendon reflexes on the left, positive bilateral Babinski's reflex, nuchal rigidity, a positive Kernig's sign, and bilateral bronchopneumonia. Spinal fluid examination showed normal pressure, protein 129, sugar 69, chlorides 113, 4 red cells and 3 white cells. The patient's course was continuingly downhill and she expired on 11-2-59.

AUTOPSY EXAMINATION: There was bilateral bronchopneumonia. No evidence of tumor recurrence was found. The swollen brain weighed 1310 gm. The base was soft gray-brown and ragged. A granular gray-pink, generally firm but focally softer, fairly well demarcated mass replaced the medial portion of the left temporal, parietal, and occipital areas. The left thalamus and internal capsule as well as the left cerebral peduncle were also involved; tumor tissue extended along the tegmentum of the pons almost to the medulla. The dorsum of the pons was soft and hemorrhagic. The pineal gland could not be identified. A 3 cm. area of recent softening involved both cortex and white matter of the superior medial portion of the left occipital pole.

K. T. Neubuerger, M.D.

Slide 7

AANP Slide Session 1960

UA 44-59 A

This 28 year old woman is known to have had Hodgkin's disease for about 5 years. She had been treated initially by x-ray, and more recently, by nitrogen mustard and cortisone. An infection of the mouth was present 4 months prior to death, and white plaques were noted in the pharynx 1 month prior to death. There were no neurological abnormalities until the day of death, when Jacksonian seizures accompanied by a fever of 106°F. were observed for a short time.

The brain revealed considerable exudate over the leptomeninges. On section, numerous areas of green discoloration up to 3 cm in diameter, were present in the cortex and white matter of the cerebrum, cerebellum and brain stem.

The process illustrated was limited to the brain.

Slide 8

AANP Slide Session 1960

Jefferson Medical College NP #56-39 (Submitted by R. G. Berry)

This 19 year old college student first showed occasional episodes of confusion 10 months prior to death. There was a relentless progression of psychiatric symptoms: lack of initiative, apathy and subjective clouding of his thinking ability. These were later associated with bizarre activity, posturing and the inability to keep up with his school work, all of which eventually necessitated admission to a psychiatric hospital 4 months prior to death.

Abnormal tic-like movements incorrectly called myoclonic were the only neurological findings. He was diagnosed schizophrenic reaction, catatonic type and was given insulin therapy. Occasional tremblings of the body and extremities developed. Facies gradually became immobile and voluntary movement ceased. These culminated in a febrile reaction 6 weeks prior to death, with rapid deterioration in 2 weeks to profuse sweats, intermittent extensor spasms, opisthotonus and head-eye deviation to the left. Within a week, he was in a continuous state of decerebrate rigidity.

It became increasingly difficult to arouse him. Opisthotonus increased. Reflexes became more active and a grasp reflex was present on the left.

The only constant laboratory finding during this neurological process was a first zone colloidal gold curve. An hypotensive episode associated with low prothrombin time and gastrointestinal bleeding occurred 5 days prior to death. This was quickly and adequately treated. The rigidity began to recede, but death occurred 10 months after the onset when signs of an empyema developed in his right chest.

Autopsy performed one-half hour after death confirmed the empyema and atelectasis. There was a superficial stomach ulceration which yielded candida albicans. The brain weighed 1410 grams. There was an hemorrhagic lesion in the internal capsules and the splenium and the genu of the corpus callosum, which extended down the long descending tracts to the level of the 8th cranial nerve. In addition, some small patchy grey plaques with an irregular outline were noted in the white matter, particularly in the frontal gyri.

Slides 9A, 9B, 9C.

CASE No. 59-650

SPECIES: Feline BREED: Cross AGE: 3 years SEX: Female

HISTORY: Animal spayed May 5, 1959. Sudden onset of paraplegia on Sept. 25, 1959. Physical examination by local practitioner said to be normal. Sept. 29, 1959: Pulse, respiration, and cardiac auscultation normal. Reflex examination revealed normal cranial nerve reflexes, normal postural reflexes with the exception of the extensor postural thrust which was absent. The hind limb special reflexes were absent. Sensation was dull in both hind limbs. The animal evidenced urinary incontinence. X-ray examination of the vertebral column was normal. The animal was maintained on symptomatic treatment until Oct. 16, 1959 at which time the skin of the distal right hind leg sloughed away and the cat was necropsied. Cerebro-spinal fluid studies from the cisterna magna revealed 11/WBC/mm^3 and a protein of 13.5 mg.%.

PERTINENT NECROPSY FINDINGS included distal dry gangrene of the right hind leg from the hock down. The heart, lungs, aorta, and proximal femoral arteries appeared grossly normal. The right kidney was small with several large infarcts. The musculature of the left hind leg was small and atrophied. The musculature of the right hind leg below the stifle (knee) was atrophied, firm, and white. The muscle lateral to the lumbar spine on the right side was white, firm, with a streaked appearance. Section of the lower lumbar cord revealed cavitation with a semi-liquid consistency of the gray matter.

Sections: Right kidney, right and left musculature, lumbar spinal cord (L_3,L_4,L_6).

Slides 10A and 10B CASE SUBMITTED BY DR. R. A. CIASEN
PRESBYTERIAN-ST. LUKE'S HOSPITAL
CHICAGO, ILLINOIS

A58-158

CLINICAL HISTORY: This 68 year old white female was readmitted to the Presbyterian-St. Luke's Hospital with complaints of diplopia, dizziness and nausea of five days duration. Twenty two months prior to admission the patient had a right radical mastectomy for carcinoma of the breast. This was followed by radiation therapy because of positive axillary lymph nodes. Three months later she had a bilateral oophorectomy.

PHYSICAL EXAMINATION: The patient was described as a well-developed, well-nourished, pale, white female. The pupils were equal and reacted to light and accomodation. There was nystagmus on lateral gaze, more to the right. Convergence of the eyes was poor and the discs were flat.

HOSPITAL COURSE: While in the hospital the patient developed coarse nystagmus on left lateral gaze, a left lower facial weakness, adiadochokinesis of the right hand and foot and a poor finger-to-nose test on the right. She complained of severe dizziness. Therapy included analgesics and meticorten for cerebral metastases. She expired of pneumonia in the 8th hospital week.

POSTMORTEM FINDINGS: There was considerable metastatic tumor in the lungs and mediastinal lymph nodes. Incidental findings included a large tubular adenoma of the left kidney and two 0.5 cm. meningiomas, one located on the right supra-orbital ridge and the other over the occipital bone at the midline. On sectioning of the fixed brain no gross tumor was seen. Both dentate nuclei appeared shrunken and atrophied, but this was more prominent on the right. The degenerative changes seen on the accompanying slides were confined to the white matter of the cerebellum, the fibers surrounding the olivary nuclei of the brain stem, and the pons. The brachium conjunctivum showed demyelinization and fiber degeneration not accompanied by the macrophage reaction seen elsewhere. There was a single microscopic focus of metastatic tumor in the pons.

Slide 11

Case#
D-576, contributed by Ellsworth C. Alvord, Jr., M. D.

This 59 yr. old farmer developed progressive weakness, weight loss, pains and cramps, with death 15 months after onset. He had been in good health until August 1956, when he awoke one morning with pain in the left hip radiating down the medial aspect of the left leg. The following week he developed numbness in the right knee, hand and face, and then abdominal pain, and constipation. Gradually numbness of both hands and dragging of the left foot developed. In December spinal fluid contained 140 mg.% protein, and chest x-ray was normal.

By February 1957 he had become more weak, unable to write or handle objects, and was hospitalized for 4 months. Examination revealed muscle strength and coordination slightly impaired in both hands. There was muscular atrophy in the quadriceps and slightly in both hands. The gait was slightly ataxic, and there was a postive Romberg. Touch, pain and position sensations were decreased bilaterally and distally, more in the arms than in than in the legs. Vibration sense was normal except in the left big toe. Biceps, triceps, and patellar reflexes were absent; the ankle jerks were normal. A left Babinski sign was recorded. X-rays revealed no significant lesion in the chest, gall bladder or gastrointestinal tract. X-rays of the spine revealed slight osteo-arthritis. Routine laboratory work was normal including total serum protein, A/G ratio, fasting blood sugar, VDRL, thymol turbidity, bilirubin, Schwartz-Watson, and LE preparations. Serum cholesterol varied from 222 to 381 mg.% on 7 occasions. The first examination of the urine for arsenic revealed 0.6 mg./L, but the next 4 examinations were all negative. Lumbar puncture on February 25 was normal except for protein 108 mg.%. Subsequent lumbar punctures revealed protein 113 mg.% on March 13 and 85 mg.% on April 26.

During his hospitalization he was treated with steroids, which afforded some improvement for a while, but then he began to complain more about paresthesias in the arm and face, and became negativistic, hypocondriacal and depressed and cried all the time. It was thought that he had a passive dependent personality with a neurotic reactive depressive reaction to his organic disease, and in September he received 10 electro-shock treatments for his psychoneurosis. His depression improved, but his weakness continued to become worse, so that he became bedfast. He was unable to swallow solids and occasionally choked on liquids and developed constant nausea. He had lost 90-100 lbs. over a 15 month period (normal weight 200 lbs). Diplopia and tremor gradually appeared. He was hospitalized for the last time on Nov. 2, 1957, when he was noted to be cachetic but alert and cooperative, moving frequently because of cramps. Neurological examination revealed marked muscular atrophy of all muscles including the shoulder girdle and small muscles of the hand. There was a coarse tremor of the hands with cogwheel rigidity. Extra-ocular movements were markedly weak with vertical nystagmus. Facial movements and sensation and hearing were normal, but he was unable to open his mouth widely, and there was slight weakness of the tongue and masseter muscles. The gag reflex was hyperactive. Bowel sounds were increased, and there was markedly increased anal sphincter tone. Sensory examination was variable and not reliable. Deep tendon reflexes were absent, but superficial abdominal reflexes were present. There was no Babinski sign.

Lumbar puncture on Nov. 5 revealed no cells and 67 mg% protein. EEG was slightly slow but within normal range. X-rays of the skull, hips, pelvis and abdomen were normal. X-rays of the chest on Nov. 4 revealed a 5 x 5 cm. tumor mass in the medial right base posteriorly. He became progressively worse and died on Dec. 10, 1957, about 15 months after the onset of symptoms.

Autopsy revealed bronchogenic carcinoma with metastases only to regional lymph nodes and left adrenal, and incidental carcinoma of thyroid and adenoma of parathyroid.

AANP Slide Session 1960

Slides 12A and 12B
From adjacent blocks

Case Submitted By Dr. Richard Lindenberg CAL. 827/59

This 44 year old white widow was living by herself. According to her physician, she never was seriously ill. On December 5, 1959, at 9:30 A.M., she was found dead in bed. The back door of the house was open. The body was lying with the head jammed against the headboard of the bed. Except for the head and neck, the body was covered with bed sheet and blanket which showed no derangement. The pillow was under the chest and the pajama pants were pulled down in the back. One button of the pajama top was torn off together with a religious medal. The bed sheet showed some staining with blood which was of a type other than that of the deceased. There was no derangement of the contents of the room. The handbag of the deceased was missing.

On December 6, 1959, at 9:45 A.M., a medicolegal autopsy was performed. The body measured 5' 4" in length and weighed 132 lbs. A small amount of serosanguinous fluid had drained out of the left corner of the mouth. Over the midportion of the helix of the right ear, there was a single small excoriated area; and a 1 centimeter long superficial bruise was found over the right anterior neck. The lips and the tongue showed no pathology. The upper canines and incisors were covered with chewing gum, which was embedded deep between the teeth. There were no external alterations indicating a fight. The external genitalia showed no bruises or lacerations. Sperms were present in the vagina. Over the superior horn of the thyroid cartilage, there were submucosal fresh hemorrhages. Similar hemorrhages were found on the posterior surface of the epiglottis. Histologically, there was also some fresh hemorrhage between the submucosa and muscle layer in the area of the larynx. The lungs were edematous, weighing 520 and 470 grams respectively. There was no obstruction of the respiratory system. The heart weighed 380 grams. The coronary arteries showed some non-obstructive arteriosclerosis. The heart muscle revealed no scarring. All other organs revealed no pathology.

The brain, which weighed at time of autopsy 1460 grams, weighed 1520 grams after fixation. It showed no signs of intra vitam swelling. There was no arteriosclerosis. The only pathologic alteration within the brain consisted of a small round-shaped, probably granulomatous lesion containing cholesterol crystals. This was located in the dorsal portion of the left posterior thalamus. It measured 2 to 3 mm. in diameter and was surrounded by a brownish discolored zone. Ventrally from this zone, there was some grayish discoloration of the thalamic tissue.

The case was declared a homicide. All efforts of the police to solve the case have shown no result so far. One week after the body was found, a sweater belonging to the deceased was found along the roadside about one-half mile away from the home.

The microscopic slide submitted shows the thalamic lesion. What is the nature of the lesion? Does the lesion have any bearing on the fatal outcome of this case?