THE TERRIBLE THREAT OF NERVE GAS

By JOHN KOBLER

Workers at Edgewood Arsenal's Toxic Agent Test Site collect samples which measure nerve-gas concentrations at various heights. Despite progress in developing an antidote, no system of instant detection exists.

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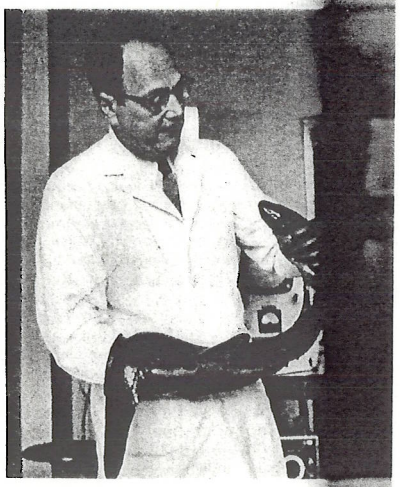

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THREAT GAS

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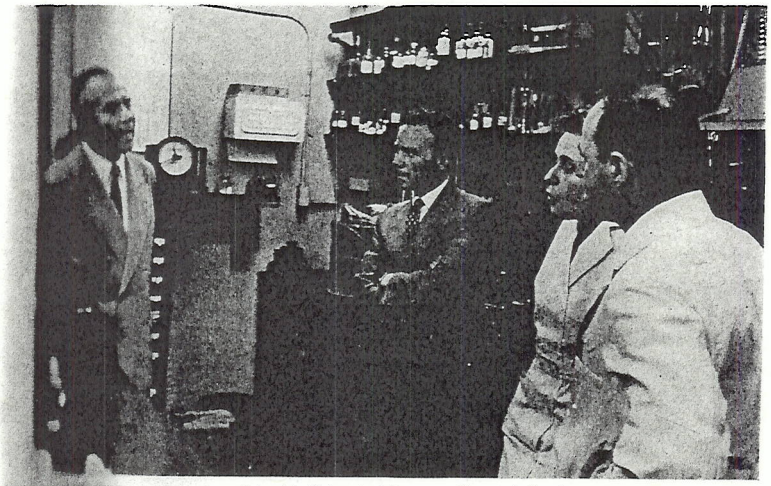

Nerve-gas researchers at a Columbia University laboratory: From left: Dr. David Nachmansohn; Dr. Helmut Krewitz, who is a Ford Foundation Fellow from Germany; Dr. Sara Ginsburg, and Dr. Irwin Wilson.

Photographs by Gus Pasquarella

A daring experiment on human guinea pigs will be performed in the not too distant future by the United States Army Chemical Corps in Edgewood, Maryland. A group of Army chemists, headed by director of research Dr. William H. Summerson, will deliberately expose themselves to nerve gas—the deadliest known chemical warfare weapon—which paralyzes the nervous system.

Nerve gas was invented and manufactured in large quantities by the Germans during World War II, though never used, and now it is being stockpiled by both the United States and Russia. It might well be the weapon of choice of an enemy planning to occupy a city. Odorless and invisible, the vapor from three drops can kill in four minutes. Under optimum conditions, with a brisk wind blowing at ground level, guided missiles containing the gas could probably destroy 90 per cent of all life within an area of many square miles.

The Army chemists, right after taking the poison, will inject themselves with one of a number of new compounds, the products of more than sixteen years' research by the Chemical Corps and by various civilian scientific groups. If their calculations have been correct, they will suffer no ill effects. They will have gone far toward eliminating the terrible menace of nerve gas.

The prospects of success are bright. Two of the compounds have protected laboratory animals against two types of nerve gas. The first compound was developed at New York's College of Physicians and Surgeons, a branch of Columbia University; the second was developed at Edgewood.

The search for those compounds set in motion one of the strangest secret missions of World War II. Early in the war, when the Chemical Corps received intelligence reports of nerve-gas manufacture by the Germans, it sought help from a scientist who probably knows more about the chemical make-up of nerves than any man alive. He is fifty-eight-year-old, Russian-born Dr. David Nachmansohn, professor of biochemistry at Columbia.

Concerning the mechanics of nerve gas, little was then understood beyond its general effect. Nachmansohn was given the top-secret assignment of investigating its action in detail and, if possible, contriving an antidote. "Besides serving my adopted country," he says, "I welcomed the opportunity of working with the toxic as a tool of basic research. Here was a substance that in some mysterious way violently affected the nerves. What could we learn from it about the nervous system?"

To a Chemical Corps procurement officer who asked him if he needed any special materials, Nachmansohn blandly replied, "Yes, please, one hundred electric eels from the Amazon."

The Chemical Corps promised its co-operation, but the actual masterminding of Operation Eel fell to Christopher W. Coates, then as now curator of the New York Aquarium, and a world-renowned authority on the behavior of electric eels.

Coates' main supplier of exotic fish was Paramount Aquarium, Inc., of Dobbs Ferry, New York. The firm, originally German, was the largest in the business, with collectors and collecting stations scattered all over the world, but the war had brought its operations to a standstill. Moreover, though its personnel were anti-Nazi and had fled Germany at the beginning of Hitler's rise, they were still under surveillance as aliens. Nineteen of them had been interned in various Allied countries.

Coates undertook the delicate task of first getting them clearance for a semiofficial mission; then negotiating a deal whereby Paramount agreed to procure the eels if permitted to import other fish for its own account.

Of the free-lance collectors frequently retained by Paramount none knew the upper reaches of the Amazon better or had greater skill at trapping electric eels than a wiry, dauntless, middle-aged Frenchman named J. Auguste Rabaut. The native fishermen of the Amazon will not go near electric eels, which *en masse* can kill a man. Rabaut was then in New York on a visitor's visa and his status presented Coates with another knotty poser. On the one hand, Rabaut had had some slight difficulties with the Brazilian Government and, on the other, even if admitted to Brazil, he would not budge from the United States without a re-entry permit. To make things more difficult, the real reason for the expedition could not be disclosed to Paramount, to Rabaut or to any of the intermediaries. Coates himself was not supposed to know of the nerve-gas project—the word

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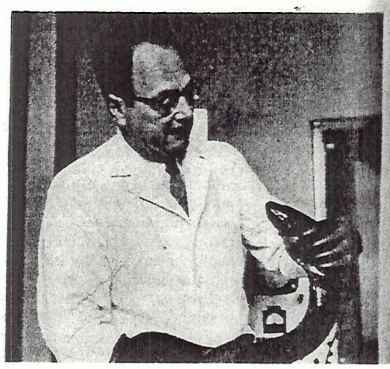

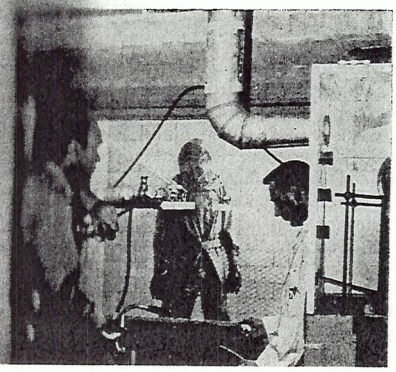

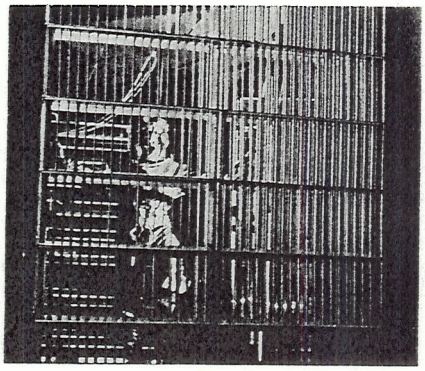

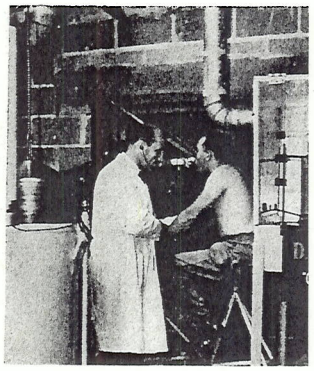

The Terrible Threat of Nerve Gas

(Continued from Page 29)

never passed between him and Nachmansohn—though he later guessed it.

The next hurdle was transport. As his base camp, Rabaut chose Letitia, a native village in the wilds of Colombia. The only feasible approach to it was by amphibian plane along the Amazon from Belém, some 1100 miles away, on the east coast of Brazil. No such plane could be spared stateside. But Coates heard that the United States Rubber Development Corporation, a Government agency, had a PBY in Belém. After weeks of dickering, an Army plane flew Rabaut to Belém, and the PBY flew him thence up the Amazon.

The PBY returned to Belém after depositing Rabaut with a supply of food. He built a raft and began cruising the river, eyes peeled for electric eels. Most of the time they lie torpidly in shallow water. Rabaut would drag them ashore with seines, wait for them to dry and, donning rubber gloves, toss them into tanks of water.

The eel collector lived in isolation, stretching out his food rations with what game he could catch. His only means of communication with the outside world was through an occasional native boatman who ventured downriver. In this way Rabaut sent word to Coates via Belém that he desperately needed new rubber gloves. Coates enclosed several pairs in airmail letters to Belém. Almost all were returned without comment by the United States military censor, but toward the end a few slipped through.

When the PBY returned for Rabaut, he had collected more than 100 eels. But before the plane could take off again, the State Department, moving in its own mysterious way, decreed that the plane should not transport either Rabaut or the eels across Brazilian territory. The alternative route out was north through a gorge with walls 8000 feet high. The eels together with their tanks weighed almost two tons. They had to be stowed in the nose of the PBY; then, as soon as the plane cleared the river, shifted to the tail.

The eels traveled the last leg of the journey—by rail from Miami to New York—in style. Freight cars were too cold for them so they rode in heated private compartments.

Behind Operation Eel and its contribution to a nerve-gas antidote lies one of the most far-reaching quests in scientific annals: The life-saving drugs are only by-products of basic research into the human nervous system. The larger goal is nothing less than the discovery of the nature of thought, how it arises, how impulses are translated into action.

Among the phenomena involved in that formidable enigma are two extraordinary biological entities—choline esterase and acetylcholine. Nachmansohn has been exploring them for the last twenty years and expects to devote the rest of his career to them.

Choline esterase is an enzyme. Manufactured by all living cells, enzymes are the maintenance crew of the body. Without them vital functions like breathing and digesting would be fatally slow. In fractions of a second they cause speed-up and regulate chemical changes throughout the animal organism—the conversion of food, for example, into elements that the blood can absorb. There are hundreds of different enzymes, each acting upon a specific substance.

To illustrate further, our bodies need sugar for energy. But before sugar can be released, it must be split into simpler components, then still simpler ones. A series of fifteen enzymes performs that splitting.

The substance upon which an enzyme acts is called a substrate. Acetylcholine is such a substrate—the substrate of choline esterase.

It was first found to exist in animal tissue twenty-seven years ago by a British physiologist, Henry Hallett Dale. He noted its presence specifically at the nerve endings. What was it doing there?

Nerve and muscle cells, Dale knew, generate electricity and the electricity is the conductor of impulses from the brain. He concluded that if a pianist, say, wishes to strike middle C with his index finger, the impulse travels from the brain to the nerve endings, where it foments a discharge of acetylcholine. In a sense the pianist thinks acetylcholine into being. In the instant of its discharge Dale further demonstrated, it causes a momentary muscular contraction, and that contraction is the driving power which moves the pianist's finger. Every heartbeat, every intake of breath, all of our countless movements are triggered in the same way by the potent compound.

It follows that some force must intervene to destroy each discharge of acetylcholine after the power has been utilized. Otherwise the pianist's finger would go on twitching indefinitely, the whole human machine would be shaken apart. Dale postulated an enzyme of which acetylcholine is the substrate. Just as there are special enzymes to split up sugar, he reasoned, so must there be a special enzyme to split up acetylcholine. He visualized a pair of chemical teammates in continuous off-and-on action—the one flicking a switch, so to speak, to furnish power, the other breaking the circuit. Later researchers identified and named the enzyme choline esterase. At about that point Nachmansohn entered the picture.

He was born in Ekaterinoslav, Russia, the second of three children of a well-to-do importer. Soon after, the family moved to Berlin. At the age of twenty-four he graduated from the University of Berlin with a medical degree. At the university he met a gifted girl student, Edith Berger. They were married four years later and had a daughter, Ruth. Today Dr. Edith Nachmansohn is a psychiatrist, Ruth a Columbia fine-arts student.

Nachmansohn studied enzymes at Berlin's Chemical Institute; then biochemistry at the Kaiser Wilhelm Institute. In 1933, as the tramp of Hitler's bully boys grew louder in the streets of Berlin, he took his family to Paris, where he was appointed *maître de recherche* at the Sorbonne.

In his early studies of choline esterase and its substrate, Nachmansohn minutely analyzed nerves in lobsters, rabbits, mice, chickens and human brain tissue. He made the original discovery that the chemical teammates do not operate just at the nerve endings, as Dale contended, but throughout the entire nervous network. From this he arrived at a revolutionary concept. "Nature," he says, "does not make meaningless arrangements. Such a general distribution of substances must have a functional reason. I began to suspect that the body's prime generator of electricity was acetylcholine itself."

To verify his hunch, he cast about for some living organism that expends much more electricity than those he had been using, a natural amplifier. Only two members of the animal kingdom would fill the bill—the electric ray and the electric eel, which discharge up to 150 and 600 volts respectively, as compared to the one tenth of a volt discharged by ordinary animals, including man. "Nature's gift to the biochemist," Nachmansohn fondly calls the unlovable creatures, which have since become the constant companions of his working hours. "The point is, man doesn't need much electricity to communicate an impulse or a thought. But to electrocute, which is the way these rays and eels attack or defend themselves, obviously requires high voltage. Thus, they offer an enlarged reproduction of the human nervous system, where electrochemical phenomena are greatly amplified and so easier to study."

On display in the Paris World's Fair of 1937 were two specimens of the *Torpedo marmorata*, an electric ray from the Mediterranean, their flat, turnip-shaped bodies measuring ten inches in diameter. When the fair ended, Nachmansohn persuaded the exhibitor to part with them. Installing the baby monsters in a tank in his laboratory and insulating himself with rubber gloves, he sliced out bits of their natural batteries. As he expected, choline-esterase-acetylcholine activity was rife.

To obtain enzyme extract for further examination in test tubes, he journeyed to a marine laboratory on the Mediterranean coast, well stocked with torpedo rays. Of the electric eels, generating still higher voltage, its tail being 60 per cent battery,

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he managed to get only a single specimen. They come from the fresh-water rivers of tropical South America and importing them was prohibitively expensive. Running up to ten feet in length, they have fetched as much as $100 a foot.

A month before the war broke out, Nachmansohn accepted an invitation from Yale to continue his research under its auspices. He sailed aboard the Normandie with his wife, daughter and fifteen quarts of milk-white enzyme extract.

Meanwhile, German production of nerve gas, rendering all other poison gases obsolete, was under way. The German code designation for them was Trilon, actually an innocuous water softener; the real names: Tabun, Sarin and Soman. Why the Germans never used them—fear of retaliation, or that sudden wind shifts would imperil their own troops—is still debated by military historians. At the Nuremberg trials Albert Speer, the Nazi Minister of Munitions, testified:

"I knew that they (Hitler and his political advisers) were discussing the question of using our two new combat gases, Tabun and Sarin. They believed that these gases would be of particular efficacy, and they did in fact produce the most frightful results . . . there was no respirator and no protection against them that we knew of. For the manufacture of this gas (Tabun) we had about three factories, all of which were undamaged and which, until November, 1944, were working at full speed."

Allied intelligence got its first inkling of Tabun in 1944. The Allies did not obtain the formula until after the war, when they seized all three German plants. One at Dyhernfurth in Poland fell to the Russians.

The idea of a volatile toxic, undetectable by the senses, was no novelty. As early as 1940 the Allies had the formula for a gas, also of German origin, called di-isopropyl fluorophosphate—DFP for short. It proved less lethal than Tabun and was superseded in 1945 by what the Chemical Corps now refers to as the G-series, including Tabun.

Nerve gases were evolved from certain insecticides with which they share the same nerve-paralyzing properties. Dr. Gerhardt Schrader, research chemist for the huge German I. G. Farben trust, developed a series of these insecticides so lethal as to make them impractical for agricultural purposes—though modifications of them are now widely employed. They were adapted instead to chemical warfare.

Ever since Allied intelligence penetrated the Nazi secret, one of the United States Army Chemical Corps' highest-priority projects has been both to stockpile its own nerve gases and to devise defenses against them. In 1951 Gen. Anthony C. McAuliffe, the then Chief of the Chemical Corps, announced an expenditure on that project of a billion dollars. The Russians, he added, were making nerve gas. With the Dyhernfurth plant at their disposal, it could be assumed that they had a fair start.

American stockpiles of the G-series are now abundant and still growing. A plant at Muscle Shoals, Alabama, prepares the basic ingredients, consisting of cheap, common chemicals. They are distributed among the Chemical Corps centers, the principal one being the Rocky Mountain Arsenal near Denver. There end production proceeds amid elaborate safety devices. Ultra-sensitive electronic-signaling devices respond to the faintest traces of escaping vapor. Each laboratory keeps on hand canaries, rabbits or fruit flies, which react to the poison faster than humans. Workers handling large amounts of the toxic wear hermetically fastened, head-to-toe clothing, treated with detoxifying chemicals.

Despite these precautions there have been occasional casualties. In 1954 at least seventy Rocky Mountain technicians suffered exposure. The dosage was light, yet many of them were hospitalized for days. They complained of blurred vision—the first symptom—cramps, contractions, chest pains, shortness of breath, nausea. Some told of wild dreams, extreme anxiety and an inability to make decisions. These mental symptoms led Army neurologists to speculate that even mild exposure might suffice to distort the judgment of commanders and troops in combat. More recently, during field tests in which goats were tethered in a target area, a Chemical Corps officer ventured too close. It took doctors twelve hours to revive him.

The manufacture of G-gas, however, has been an elementary operation compared to the search for countermeasures. Standard gas masks (Speer notwithstanding) do resist the nonpersistent or vaporized varieties; impermeable rubberized raincoats, boots and gloves give protection against the persistent liquid kinds—though dense saturation can penetrate even rubber.

The Chemical Corps in collaboration with RCA has developed an automatic warning device. Weighing twenty-five pounds and contained in a compact portable metal case, it can be connected to a 110-volt AC or 24-volt DC power source to operate continuously for twelve hours. After this period a simple readjustm... requiring only a few minutes, will p... again in operation. At the first trac... nerve gas a red lamp lights, a loud bu... sounds. A filtering system prevents i... ference by heavy dust or smoke.

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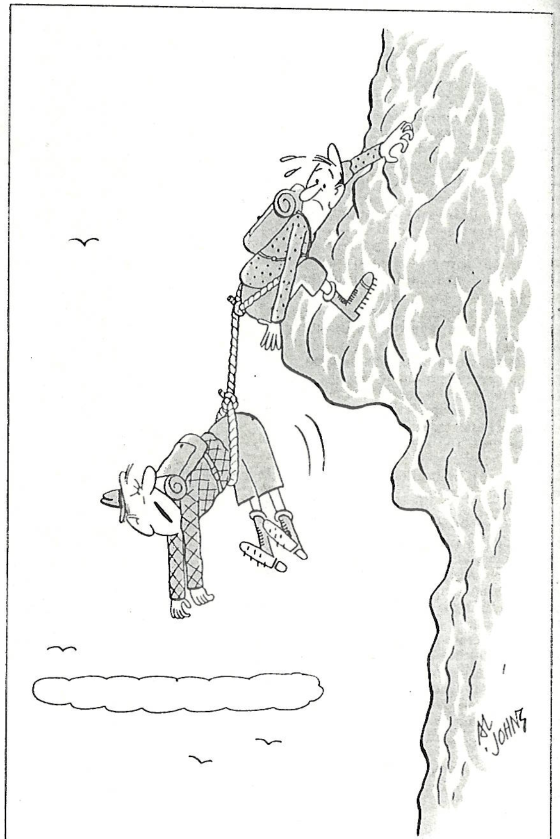

"No, you may not borrow my knife!"
THE SATURDAY EVENING POST

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Atropine sulfate suggested itself as an antidote at an early stage because of its known effectiveness in combating poisons of similar action. It was found that intramuscular injections would counteract symptoms produced by fairly heavy exposure. The drug has since saved not only victims of laboratory accidents like the Rocky Mountain technicians but also farmers, aerial crop dusters and chemical-plant packers contaminated by nerve insecticides. Of the latter group scores, not treated in time, have died.

At present atropine is the standard, recommended antidote—indeed the only one available. The Federal Civil Defense Administration has released a thirty-minute color film showing how an aggressor might attempt to destroy civilian populations with nerve gas in an attack on American cities, and how atropine can be self-injected through syrettes. Atropine-filled syrettes are now available to civil-defense organizations.

But the effectiveness of atropine, powerful as it is, would be limited against massive saturation by nerve gas to be expected in an all-out onslaught. And so the search for something superior continues.

When Nachmansohn first tackled the problem, he killed a rabbit with DFP—Tabun was not yet available—and analyzed the nerve tissues. The scientific consensus at the time was that the toxic destroyed the entire nervous system. But the only significant chemical change Nachmansohn noted was a total absence of choline esterase. He observed the same change in a test tube by adding DFP to enzyme extract from his Amazon eels. The poison completely inactivated it. That meant prolonged, uncontrolled acetylcholine activity, leading to a fatal overfunctioning of the nerves. The switch was closed with the other half of the chemical team not there to open it.

When Nachmansohn reported his findings to the Chemical Corps experts they concluded that the enzyme had been abolished. He disagreed. He thought it likelier that it had only been inhibited. Accordingly, he felt that the best antidote would be one with the power to reactivate choline esterase rapidly and so restore the normal off-and-on cycle.

Before Nachmansohn could begin to look for such a reactivator he had to determine exactly what changes DFP caused. He and a team of Columbia associates studied nerve tissue with some of the most sensitive instruments known to science. Because of the wider implications of their work, their research was supported by grants not only from the Chemical Corps but also from the Surgeon General of the Army, the United States Public Health Service and the Atomic Energy Commission.

With a micromanipulator they isolated single eel cells. They charted the speed and strength of impulses flashing through nerve fibers with an oscillograph. To separate choline esterase from other substances in tissue, they used an ultracentrifuge that cost Columbia $16,000. Now and again they were reminded of the deadliness of DFP when, in transferring fresh supplies from vials to tubes, wisps of vapor escaped and their pupils constricted. Some of the researchers suffered from blurred vision for several days.

They had barely covered the groundwork five years later when a fellow professor recommended to Nachmansohn as an assistant twenty-eight-year-old Dr. Irwin B. Wilson, of Columbia's Chemistry Department. It was a happy choice. So readily did Wilson grasp the underlying principles that he was soon able to take up the specific challenge of a nerve-gas antidote. By 1951 he had deduced the precise manner in which the toxic kills. We have seen how choline esterase acts as a kind of circuit-breaker to control discharges of acetylcholine. Nerve gas contains atoms of a chemical group called phosphoryl. These atoms attach themselves, barnaclelike, to the molecules of the enzyme. In doing so they obstruct the circuit-breaking mechanism. Acetylcholine runs wild, convulsing the nervous system.

Wilson had thus defined his target—the phosphoryl group. There remained the problem of fashioning a chemical bullet that would knock it off the enzyme. "I could have spent the rest of my life," he says "searching the pharmacopoeia for the right compound. It seemed easier to design a new one from scratch on the basis of what properties we knew an antidote must possess."

At the twenty-fifth attempt he developed, and with an associate, Dr. Sara Ginsburg, synthesized a yellowish crystalline substance—pyridine aldoxime methiodide, nicknamed PAM. Forty white mice were then divided into two equal groups. Dr. Helmut Kewitz, a pharmacologist from the Free University of Berlin, long associated with the project, injected them all with DFP. Next he gave the first twenty an injection of PAM. In five minutes the second twenty mice were dead. The first twenty survived.

Because the chemistry of the human nervous system does not differ basically from that of mice, it could be predicted that PAM would protect humans against DFP as well as against the nerve insecticides which they resemble. Said Nachmansohn, "I will be astounded if it does not."

But would PAM counteract the deadlier G-series, which, though they affect the nerves in the same way, embody variations of structure? Tests were run at Edgewood. The mice died. But Nachmansohn and Wilson were in no wise perturbed. The principle which they had formulated, they felt sure, was still valid. An antidote to other nerve gases merely required further trials along identical lines. "The problem is now practical," said Nachmansohn; "no longer theoretical."

Not long after, the Chemical Corps, following a modification of principles similar to those of the Columbia group, came up with a compound that protected mice against the G-series. The Army chemists are now on the trail of a method to confer immunity to nerve gas for prolonged periods before and after exposure.

Between the successful tests of these two compounds and their general use as antidotes a good many factors remain to be determined, such as dosages, the physical form, the techniques of administering them. Then, as the final phase of the long search, must come tests on humans. "We will have no alternative," says director of research Summerson. THE END

"No, you may not borrow my knife!"

THE SATURDAY EVENING POST

...ts and moderate incomes found themselves able to acquire luxuries that none but the very rich once could afford.

The question which now plagues the economic psychologist, as it troubles every thoughtful man, is simply this: Can it last? Is prosperity its own gravedigger? Must a decade of fantastic growth, of almost limitless wish fulfillment, end inevitably in saturation and recession?

At the moment, the economic psychologist would answer this question with a wary "No." The deep-felt wants of the American people are still unsatisfied. They are still intent on improving their standard of living. They may have bought the new split-level house in the "nice" neighborhood which they had dreamed of. Now they want to air-condition it or add a room for a new baby. They may have the two-tone fin-tail car with power brakes and power steering. Now they'd like a second car, or a sailboat, or small cabin cruiser for weekends on the lake. They have the refrigerator and the washing machine. Now they want the freezer and the automatic dryer. They have black-and-white TV, but they keep think-

waiting for the price drop. This waiti mood prevailed until the summer 1950, when the Korean War began. Th before there was any noticeable crease of Government spending or in money supply, there was a rush of pa buying.

Fearful of World War III, and reme bering the shortages, restrictions and tioning so recently ended, buyers storr the stores, grabbing everything they cc lay hands on before the anticipated p rise. They lit a quick, hot fire of inflati then quickly put it out. As the Kor front stabilized, and it became clear t the fighting in the Far East would explode into a global war, consu thinking did a quick about-face.

The mood of acute fear, accordin Professor Katona, gave way to on uneasy anxiety. The expectation of sh ages disappeared as the people real that the country could provide both g and butter during a prolonged perio cold war. The rush to buy slowed d in 1951 and the slowdown continued into 1952, as families held on to t cash, despite stable prices and the