British Journal of Anaesthesia 1995; **75**: 366–368

HISTORY

Thiopentone anaesthesia at Pearl Harbor

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Summary

A wartime embargo on casualty figures and an imprecise contemporary editorial contributed to the persisting belief that a grossly excessive mortality rate from barbiturate anaesthesia for surgery of the injured occurred after the Japanese attack on the American bases in Hawaii in December 1941. From accounts by surgical staff and official hospital records which have become available through US Freedom of Information legislation, it is clear that the rumoured death rate from this cause has been greatly exaggerated. (*Br. J. Anaesth.* 1995; **75**: 366–368)

Key words

Anaesthetics i.v., thiopentone. History, anaesthesia.

In 1939 fewer than 10 % of the 140 000 or so anaesthetic procedures carried out annually in Great Britain involved the use of a barbiturate. Evipan (hexobarbitone) and thiopentone (Pentothal) were equally regarded at the time in Europe but the local product, thiopentone, proved more popular in the United States. At the Mayo Clinic, where it had been introduced by Lundy and co-workers in June 1934, 3 months after the first clinical use by Ralph Waters in Madison, Wisconsin, progressive increase in use had occurred during the pre-war years (table 1) [1]. We can be sure that there was a general awareness of practical i.v. anaesthesia among American doctors and nurses when their country entered the war.

For those concerned with military anaesthesia, i.v. anaesthetics offered obvious advantages, and experience with the barbiturates was gained during the Spanish Civil War (1936–9). Simplicity of administration, portability, non-flammability and apparent lack of need for elaborate ancillary equipment suggested that anyone who could depress the plunger of a syringe in response to movement in a patient could give an anaesthetic.

Not surprisingly, overdosage was an important factor in mortality associated with thiopentone during its early days. Trainee anaesthetists of the 1940s and 1950s were exhorted to be cautious in dosage in shocked patients, and it was (and perhaps still is) widely believed that ... "i.v. anaesthesia was the cause of more fatal casualties among the servicemen at Pearl Harbor than were the enemy bombs" [2]. After 50 yr of rumour, the move towards Freedom of Information in the USA has made wartime documents available and allowed us to gain a better idea of what really happened.

Pearl Harbor

Stockpiling of plasma and a re-training programme immediately before the Japanese attack suggest that the medical and nursing staff in the town of Honolulu and at the naval base in nearby Pearl Harbor, on the Hawaiian island of Oahu, were in a high state of readiness and efficiency. One of the few American surgeons experienced in major war surgery and the treatment of mass casualties, Dr J. J. Moorhead of New York, had been invited by the Honolulu Medical Society to give a series of lectures on traumatic surgery. He arrived on December 3, 1941, 4 days before the attack. An audience of some 300, a large proportion of whom were army and navy medical personnel, attended his presentation on "Treatment of wounds, civil and military" given approximately 36 h before the raid; it was, as he said, "virtually a rehearsal" [3].

At 07:55 on Sunday, December 7, 1941, before the declaration of war, successive Japanese bombing raids were directed on the US Pacific Naval Base at Pearl Harbor and on many of the other military installations on Oahu. Heavy casualties were sustained by naval and Army personnel (the US Air Force at that time was part of the Army), and by civilians. Eighteen ships, including eight battleships, were sunk and 188 planes destroyed. These events led to the entry of the United States into World War II and so to the eventual downfall of the Axis powers. The declaration of war meant an embargo for the duration of hostilities on casualty figures and on firm information about what really happened at and around Pearl Harbor.

The casualty figures set out in table 2 indicate the scale of the disaster. On the battleship *Arizona* alone, which was hit and capsized rapidly, 1227 sailors and marines were trapped and killed [4]. Between 1000 and 2000 casualties presented to the 13 or 14 military and civilian hospitals on Oahu during and immediately after the onslaught. A substantial proportion of the injured would have required anaesthesia. It has not proved possible to find out how many of the injured were anaesthetized solely with thiopentone, so the precise contribution this agent made to the surgical fatality rate remains uncertain. We can, however, learn a great deal from the contemporary accounts.

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An account of this work was presented to the History of Anaesthesia Society, June 1990.

Table 1 Numbers of patients receiving thiopentone at the Mayo Clinic: annual and cumulative totals up to the attack on Pearl Harbor (from [1])

1934	1935	1936	1937	1938	1939	1940	1941	1934–41
73	1333	2745	3810	4157	5874	6629	7310	31931 cases

Table 2 Casualties: Pearl Harbor (Oahu) attack, December 7, 1941

	Killed, missing, died of wounds		Wounded
US Navy	2008	(1227 died on *USS Arizona and Utah*)	710
Marines	109		69
Army (including Air Force)	218		364
Civilians	68		35
Total	2403		1178

First-hand accounts

The situation at an Army Hospital, North Sector General Hospital, Schofield Barracks, where about 160 wounded were admitted within 2 or 3 h of the start of the attack, was described long after the war by one of the surgeons there at the time: "Anesthesia was a problem. Some of the debridements were done under local anesthesia. But general anesthesia was needed in many cases and the only nurse anesthetist was busy in the operating room. Captain Bob Hoagland of the Medical Service came over to help. We had just been issued with a new drug called Pentothal which could be given i.v. for anesthesia. We got out a box, read the directions and Bob used it on many of my cases over the next 48 hours with excellent results."

There seems no reason for this 1988 account by Dr R. M. Hardaway, a Texas Professor of Surgery, to be biased or lacking in critical data. No mention is made of any excess mortality or morbidity from Dr Hoagland's activities [5].

A truly contemporary account by a civilian surgeon, Dr J. E. Strode, who was attached to the 900-bed Tripler Army Hospital during the emergency, headed "Observations on the treatment of war wounds", was published in the local *Hawaii Medical Journal* in January 1942, a few weeks after the attack. He does not mention barbiturates but reports that: "Ether anesthesia, by the drop method, lends itself well to this type of surgery. It has the advantage of safety and, in addition, it may, when necessity demands, be given by those with little training" [6]. Note the mention of "safety". Is it implied that other techniques proved unsafe in untrained hands when dealing with mass casualties?

The Naval Hospital, at the centre of the attack, received a high proportion of the injured from targets in the harbour. By midnight on the day of the raids, 960 patients were accommodated in a hospital built to contain 506; the following day another 100 patients who had been temporarily cared for elsewhere were admitted. In his report on the treatment of acute surgical cases among the wounded, published in May 1943, a full year after the appearance of the previous account, Captain R. Hayden, Officer Commanding Pearl Harbor Naval Hospital, did not mention any problem or excessive mortality rate arising from anaesthesia in general or from the use of thiopentone. He commented "Most of our anesthesia was by drop ether. This lends itself well to this type of surgery and in addition, when necessity demands, it may be given by those with little training" [7]. This wording, from a different service and published in another journal, is surprisingly similar to that of Dr Strode's earlier paper. While the naval author could have read the previous account, why should he plagiarize? Could this be wartime censorship in action, suppressing damaging information?

The main "scare-story"

Censorship seems possible and may also have prevented actual numbers of cases being revealed in what is undoubtedly the main source of the "problems with Pentothal" legend, the short paper "A critique of intravenous anesthesia in war surgery" published in *Anesthesiology* in January 1943 by Dr F. J. Halford, a civilian surgeon assigned, under emergency conditions, by the Oahu Office of Civilian Defense to the Tripler Army Hospital in Honolulu. The hospital had three full-time nurse anaesthetists and Dr Halford must have been working near to Dr Strode, who has been quoted above.

Summarizing the experience gained in dealing with the seriously wounded from the Japanese attack, Dr Halford and his civilian colleagues considered that open drop ether was the best anaesthetic for war surgery. I.v. agents were dangerous for shocked patients suffering from heavy blood loss. Of the Pearl Harbor casualties, he said: "A number of patients were given Evipal by competent anesthetists only to have respiratory failures, some of which ended in death. After several such fatalities, Pentothal sodium was used, and again respiratory failures occurred, and, as in the case of Evipal, death ensued in enough cases to cause us to abandon it as too dangerous. In several cases when as small an amount as 0.5 g of Pentothal sodium had been administered, there suddenly appeared a 'cyanosis decolletage' which was the inevitable and irremediable predecessor of death. There was a definite lack of oxygen and equipment for administering continuous oxygen therapy" [8].

In the official account, released long after the war, it is clear that the Tripler Army Hospital received 482 battle casualties during the morning of December 7, of whom 138 were dead on arrival. Of the 344 wounded, 13 *did not survive* [9]. So, while it cannot be doubted that several additional deaths did occur in the hospital to which Dr Halford was attached, and perhaps elsewhere, as the direct result of barbiturate overdosage (thiopentone was not the only agent used), in the context of more than 2000 servicemen and civilians killed by enemy action on that day, and a greater number of wounded, many of whom underwent successful anaesthesia, comments of workers on the spot and the figures for inhospital deaths released after the war strongly suggest that

the Pearl Harbor barbiturate mortality horror story has, in common with so many wartime rumours, been grossly exaggerated.

Wartime censorship, which aimed at preventing rumours of medical mishandling at Pearl Harbor that might have been of comfort to the enemy, and discomfort to the allied troops, ended in 1945. Ten years later, Beecher, in his chapter on "Anesthesia for men wounded in battle" which formed part of the official account "*Surgery in World War II*", wrote that during the early stages of the war, inexperienced anaesthetists and unwise choice of agent were responsible for an excess mortality rate, of the order of 1 in 450 administrations from barbiturate anaesthesia. The barbiturate mortality rate from the early North African campaigns "paralleled", Beecher said "the experience reported from Pearl Harbor" [10], independently confirming the view that the excess mortality resulting from the use of this type of anaesthesia for the casualties of the Japanese attack was of the order of 4 or 5 cases—unfortunate, but hardly a major disaster in itself.

Calming troubled waters

From the medical descriptions it is clear that facilities for fluid and blood replacement for overwhelming numbers of severely injured patients were understandably inadequate. Hurriedly enrolled and unskilled anaesthetists were pressed into giving i.v. anaesthetics, sometimes in relative overdosage, and often without adequate means of administering oxygen or providing ventilatory support.

While British service and civilian anaesthetists were learning these lessons the hard way, disquiet spread in the United States during 1942, fanned no doubt, by tales of further difficulties in other theatres of war. In January 1943, more than 1 yr after Pearl Harbor, *Anesthesiology* published not only Halford's alarmist paper but also an account by Charles Adams of the Mayo Clinic of how to use the agent safely in trauma cases. He described the injection of 1–2-ml doses of 2.5 % thiopentone (5 %, and sometimes even 10 % were the customary concentrations at the time) to induce a patient who was in shock caused by severe gunshot injuries. Oxygen and nitrous oxide were given via a tracheal tube, with additional small doses of i.v. thiopentone totalling 400 mg over 1.5 h. When another operation was required by this patient 3 weeks later, thiopentone 400 mg was needed during the first 10 min alone [11].

The practical lessons were clear, and were hammered home by an editorial [12] in the same edition. Henry S. Ruth was then editor and the associate editors were Ralph Tovell (Lundy's former collaborator at the Mayo Clinic but seconded to the Army at the time) and E. A. Rovenstine. I suspect that Tovell was responsible for the editorial which did much to rehabilitate thiopentone in the USA. It described how the agent should be used in shocked and ill patients (small doses at long intervals, the necessity of oxygen availability, etc) and helped to form the basis of thiopentone's half century of service as the world's most popular induction agent.

Acknowledgement

The assistance of United States Military and Civilian authorities who advised and provided data for this paper is much appreciated.

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